PART TWO:
Building your own assessments

Introduction

Completing assessments is a significant challenge for community projects throughout the world. The exercises described in this manual help project participants structure assessments by identifying and building links between the sources, stressors and impacts that define environmental issues.

The structuring of assessments is an important first step in both preparing guidance for technical experts that you recruit to develop your reports and for readying the project participants to receive the reports in a way that allows them to make a bridge between a community vision and the solutions necessary to accomplish it.

The second step in completing assessments is equally challenging. Getting access to data describing each of the impacts for each of the stressors can lead to frustration and project paralysis. This second part of the manual includes basic sample assessments that can serve as a template for your own efforts and baseline information that you may find useful. The samples focus on two basic elements for your assessments: the impacts that result from certain exposures and an estimate of exposure for typical communities.

In many local environmental assessments, projects describe the range of possible impacts from environmental threats but lack the data necessary to specify which impacts are likely in their own community. The sample assessments include a discussion of possible impacts but also provide direction so that your project can identify actual impacts that may exist in your community. In general, there are two ways to determine if your community is subject to a specific environmental harm. The best way is if you have data specific to your community. Such specific data, however, is often lacking, in which case you may be able to make some rough approximations based on visual observation or by collecting local knowledge that will show similarities to other communities with more complete documentation.

Environmental assessments would be much more common if we could prove the impacts from certain environmental contaminants in a community. Unfortunately, the complexity of multiple exposures and the diversity of human populations make such proof difficult to establish.

It is for this reason that this guide and a wealth of policy are centred on risk (and not proof). Knowing that these may showcase negative impacts allows policy makers the ability to make decisions that insure against possible harm to human health.

The concept of risk

We all have our own definition of risk and for the purpose of considering comparative "risk" analysis as envisioned for LEAP development, it is important to emphasise some aspects of the term. Risk is the possibility of a negative outcome. However, it is not often measured in certain terms and proving a potential outcome from a particular threat may not be practical. For example, we may never be able to identify the specific individuals that are suffering from contaminated air, but because we believe it is possible that some people are suffering because of contaminated air, we act to reduce air pollution.

Many environmental threats confront us with possible harm. Contaminated drinking water may make us ill. Toxins in our food may result in cancer. Aesthetic degradation may deter investors from developing the potential within a community. While direct cause and effect makes for a powerful message, it is not necessary to prove direct causation to motivate a community to act; the "risk" of harm can be reason enough.

There are degrees of risk. Environmental problems which are more certain to result in harm or which may result in a large impact carry greater risk than problems leading to less certain probabilities of lower impacts. Therefore, the concept of risk captures both the probability of a negative outcome and the magnitude of its impact. When we carry out environmental risk assessments, we need to remember that both factors contribute to risk, and we need to communicate them clearly.

A caution on developing assessments

The sample assessments supplied in this guide suffer from a weakness common to the reduction of environmental issues to isolated factors of sources, stressors and impacts. There are additional impacts that result from the accumulated effects of many environmental stressors. Analysing a single chemical pollutant may suggest a very low incidence of health impact, but exposure to that pollutant, in combination with several other pollutants, may be the cause of significant impacts.

To address these cumulative risks, a community project may benefit from an overall assessment of public health and the health of the environment prior to carrying out individual assessments. The prevalence of cancer, asthma, liver problems, headaches or nausea may signify multiple environmental problems. In addition, trees that lose leaves prematurely or a decrease in certain species' populations provide ecological evidence of environmental damage. A community that is concerned about these general impacts may be more interested in investigating some of the causes, but applying the individual assessments to prove the impacts will be hard.

With these cautions, we invite you to review the sample assessments and seek applications for your own community. Good luck!

Using the sample assessments

The appendix includes some sample assessments. These assessments serve two purposes. They may help you better understand the impacts of environmental problems in your community, and the presentations may be useful to consider as templates for reporting your assessments.

Most of the assessments include three parts:

1) a visual map of sources, stressors, exposures-pathways and impacts;

2) a list of potential impacts described; and

3) dose-response graphic to identify the kinds of impacts you might expect at certain levels of exposure.

Drinking water

Microbiological contaminants in drinking water

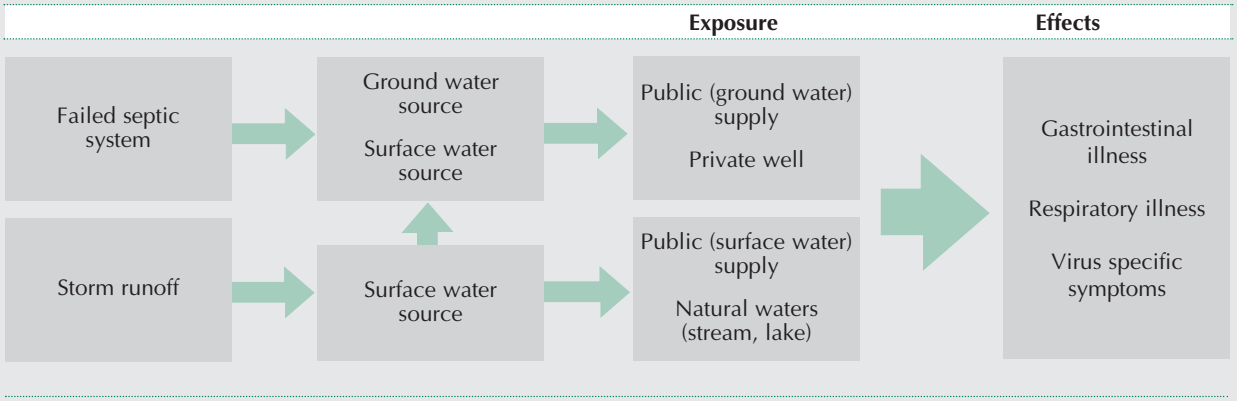
Possibly the first environmental hazard managed in human history was microbial contamination of drinking water. Microbial contaminants, or pathogens, include bacteria, viruses and parasites. Pathogens are transmitted in the excrement of infected humans and/or animals (carriers) that may not show any signs of disease. A small percentage of disease victims become permanent carriers of the causal organism (for example, less than 4 percent of typhoid fever victims) , in addition to the temporary carriers — those currently sick or recovering — who may excrete large quantities of pathogens.

Sources

Raw sewage and storm runoff will contain pathogens and most surface water is contaminated to some extent. Drinking water supplies are at risk where raw water is obtained from a surface source or where there is a surface water connection to a ground water source.

DIAGRAM 4

Tracing the effects of microbial contaminants



Exposure

The presence of fecal coliform bacteria in water (indicated by E.coli) is used to evaluate the potential for contamination by additional, disease-causing microorganisms. Public water supplies treated with filtration and disinfection may significantly reduce the health risks from microbiological contamination, but not all organisms are effectively controlled by standard treatment.

Health effects

Ingestion of water contaminated with bacterial pathogens (e.g., salmonella, shigella) may cause disease symptoms including fever, diarrhoea, cramps, nausea, jaundice, headaches, fatigue and even death. Unlike most environmental threats, the concentration of microbial contaminants is not critical to the assessment of health outcomes. Other factors related to personal health, immune status, nutrition and access to health care are important in understanding the potential impacts.

Any virus excreted in the faeces could theoretically be transmitted through drinking water. Waterborne outbreaks of infectious hepatitis have been well documented and water contamination has been identified as a factor in incidences of additional viruses such as echovirus, Coxsackie virus and adenovirus. Resulting health effects can include respiratory disease, meningitis, fever, vomiting, diarrhoea and eye infection.

Protozoan pathogens including Giardia lamblia and cryptosporidium cause symptoms ranging from mild diarrhoea to weakness, fatigue, dehydration and abdominal pain to extreme debilitating diarrhoea, fever and weight loss in people with compromised immune systems.

Calculating your assessment

As with all environmental assessments, there are two aspects to characterising the risks associated with contaminated drinking water: the population exposed and the magnitude of risk for each exposure. Filling in Table 3 provides the basis for your assessment.

TABLE 3

Classifying the risks to drinking water

Population served	Water system type	Degree of risk
	Untreated surface water supply	Very high probability of disease exposure; chronic gastrointestinal diseases prevalent
	Treated/untested surface water	Risk of operator error or insufficient treatment leading to desease outbreaks (sporadic)
	Untested groundwater with protected wellhead area	Small chance of contimination via surface water
	Untested ground water with no protection near well site	Significant chance for contamination from septic systems, agricultural waste or other sources, particularly during high precipitaion or runoff events
	Tested ground water	Minimal risk
	Filtered, disinfected and tested surface water	Low risks (see chlorinated drinking water assessment)
	Multiple sources	Risks equal to the least secure source

TABLE 4

Levels of drinking water contamination risk

Lower risk		Higher risk
Public (monitered, treated) systems		Private (unmonitered, untreated) wells
Ground water sources, drilled wells		Surface water sources, shallow wells
Fewer people exposed		More people exposed
Frequent testing		Infrequent testing
Fewer violations		More violations
Multiple treatment technologies		Single treatments such as filtration, activated carbon, reverse osmosis, ion exchange)

Chlorinated drinking water

Unlike most chemical contaminants considered in assessments, chlorine is intentionally added to drinking water to kill harmful microorganisms. The benefits from chlorination are considerable (see microbial contaminants of drinking water), but there are hazards that result as well. These hazards can be the consequence of chemical reactivity between chemically active chlorine species and other components in water.

Sources

Chlorine is intentionally added to drinking water. In water, chlorine is highly reactive and some of its by-products, such as chloride, are harmless. This assessment, however, focuses on its harmful by-products. Trihalomethanes (THM) include chloroform, bromodichloromethane, dibromochloromethane and bromoform. These result from the reaction of chlorine (and bromine contaminants) with natural chemicals associated with turbid source water.

Exposure

Chlorine exposure is the result of drinking treated water.

Effects

Some of the THM by-products from chlorination are carcinogenic. At typical levels found in drinking water (1-100 parts per billion), the increased risk for cancer is approximately one to one hundred excess lifetime cancer deaths per million population (or 0.014 to 1.4 excess cancer deaths per year per million population).

DIAGRAM 5

Tracing the effects of chlorine

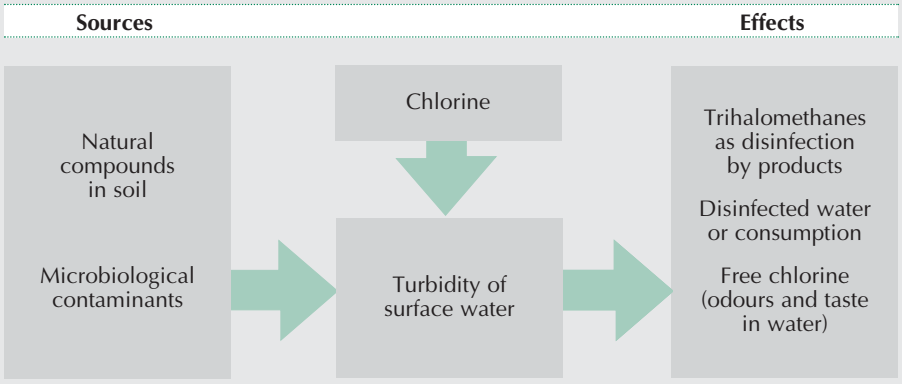
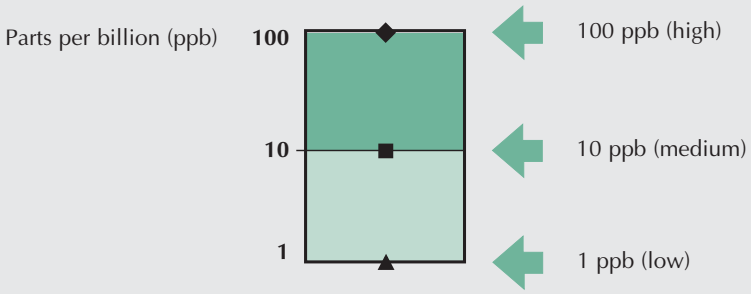


DIAGRAM 6

Trihalomethane levels in drinking water



Calculating your assessments

If you have data about the presence of THM in your drinking water, use that data to calculate excess cancer rates. In the absence of that data, you will get some sense of the possible magnitude of the problem by answering the following questions.

Is your source water very turbid?
Turbidity is the cloudiness or murkiness of water. Turbidity is an indicator of the presence of suspended solids, which not only provide the reactant material for THM formation, but also harbour microbiological organisms requiring disinfection.

Is your source water from a surface water or deep ground water supply?

High risk for THM
Treated water from visibly turbid sources can contain about 100 ppb THM. At this level, you can expect an additional 1.5 additional cancers per million population per year.

Medium risk for THM
Treated water from unfiltered surface water will still have significant suspended material even if it is not as visibly apparent. In these cases, the THM level may be in the 5-50 ppb range resulting in cancer risks ranging from 0.07- 0.7 excess cancer deaths per million population per year.

Low risk for THM
Filtered surface water or deep ground well sources require only minimal chlorination (if at all) and the generation of THM is small. In these cases the risk for cancer may be less than 0.01 excess cancer deaths per million population per year.

Air pollutants

Ozone

Ozone is a form of molecular oxygen comprised of three oxygen atoms linked together. At ground level, ozone forms as a noxious gas through chemical reactions between oxides of nitrogen (NO_x) and volatile organic compounds (VOCs) in the presence of sunlight. Ozone is the major component of smog and is measured in ambient air as a concentration in either micrograms per cubic meter ($\mu g/m^3$) or parts per billion (200 $\mu g/m^3$ = 100 ppb) .

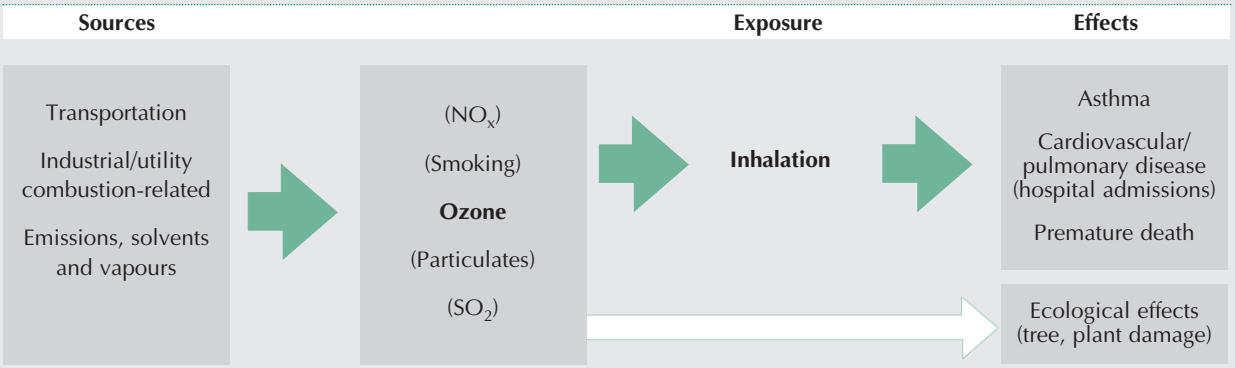
Sources

Ozone is formed in the atmosphere through a complex series of chemical reactions between NO_x, hydrocarbons and sunlight. The ability of these reactions to occur in the atmosphere is related to temperature and the intensity of sunlight. High ozone levels are most common on hot summer days.

Sources of hydrocarbons and NO_x include various industrial and combustion sources, gas stations (vapours) , and auto paint shops (solvents) . In urban areas, the majority of ozone-forming pollutants come from cars, buses, trucks and off-highway vehicles (construction vehicles, boats, etc.) .

DIAGRAM 7

Tracing the effects of ozone



Effects

Asthma

Ozone is an irritant to the lungs and mucous membranes. High ozone concentrations have been shown to increase the rate of emergency room visits for asthma within a period of two to three days following a moderate ozone event (exceeding 75 ppb) .

Premature death

At high levels, or for extremely sensitive populations, ozone may cause premature death from decreased lung function, lung damage or triggered asthma cases.

Long-term effects

Repeated exposure to ozone pollution may cause permanent damage to the lungs.

TABLE 5

Ozone effects

Ozone concentration (ppb, 8-hour average)	Heath effects
0-64	No health effects expected.
65-84	Unusually sensitive individuals may experience respiratory effects from prolonged exposure to ozone during outdoor recreation.
>75 (1-hour average)	33% increase in asthma-related hospital emergency room visits (Stieb, et al. 1996)
85-104	Sensitive individuals may experience respiratory symptoms (such as coughing or pain when taking a deep breath) and reduced lung function, which can cause breathing discomfort.
105-124	Sensitive individuals have a higher chance of experiencing respiratory symptoms (such as coughing or pain when taking a deep breath) and reduced lung function, which can cause breathing discomfort. **All populations may experience respiratory effects at this level.**
125 (8 hour)-404 (1 hour)	Sensitive individuals will likely experience increasingly severe respiratory symptoms and impaired breathing. Many healthy individuals in the general population engaged in moderate exertion will experience some kind of effect. According to EPA estimates, approximately half will experience moderately reduced lung function; and one-fifth will experience severely reduced lung function.

Calculating your assessment

The following table represents some examples of ozone levels monitored in Central Europe. (Additional data is available at <www.etcaq.rvm.nl/databases/airview.html>)

Your own community may not have ozone monitoring results, in which case you can get a rough estimate by comparing your community with these examples. Combining the data from monitoring with the table on page 40, which describes effects, will allow your project to gain some qualitative understanding of ozone impacts.

TABLE 6

Ozone concentration in selected areas

City	Maximum hourly concentrations	95th percentile (5% of the hours with readings greater than)	98th percentile (2% of the hours with readings greater than)
Sofia, Bulgaria	80-90 µg/m³ 40-45 ppb		
Budapest, Hungary	150 µg/m³ 75 ppb	 52ppb	
Farkasfa, Hungary	200 µg/m³ 100 ppb	 62ppb	
Several Czech sites	130-180 µg/m³ 65-90 ppb		 50-70 ppb
Several Poland sites	72-104		46-70ppb
Several Slovakia sites	53-103 ppb		45-70 ppb

Sulphur dioxide

Sulphur dioxide (SO_2) is the primary product from the combustion of fuels contaminated with sulphur. All coal has some sulphur, but the range of sulphur content is significant. Also, modern boilers can be equipped with scrubbers to reduce sulphur emissions.

Sources

The primary source of SO_2 is coal-fired boilers and coke manufacturing. Boilers are used to generate electricity, steam or heat.

Exposure

The primary exposure route for SO_2 is through inhalation. There are also extensive ecological effects from the acidification of poorly buffered lakes and the leaching of valuable micronutrients from the soil.

Effects

- Epidemiological studies during the London smog events of the 1950s show an increase in deaths of those with respiratory disease when SO_2 levels exceeded 0.2 parts per million (ppm). Most of these deaths occurred among the elderly. The statistics are complicated by the coexistence of particulates with the SO_2 contamination.

DIAGRAM 8

Tracing the effects of SO_2

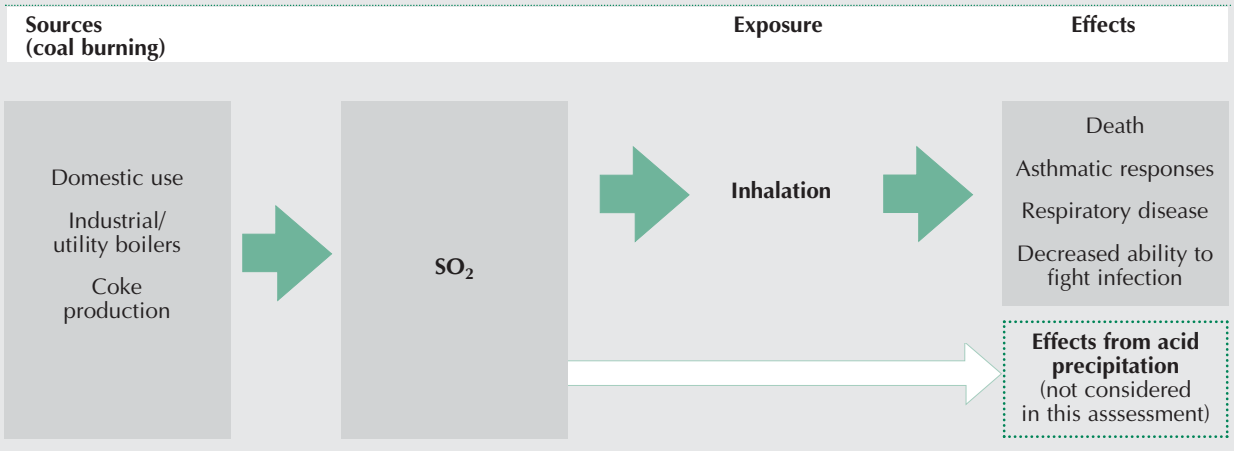


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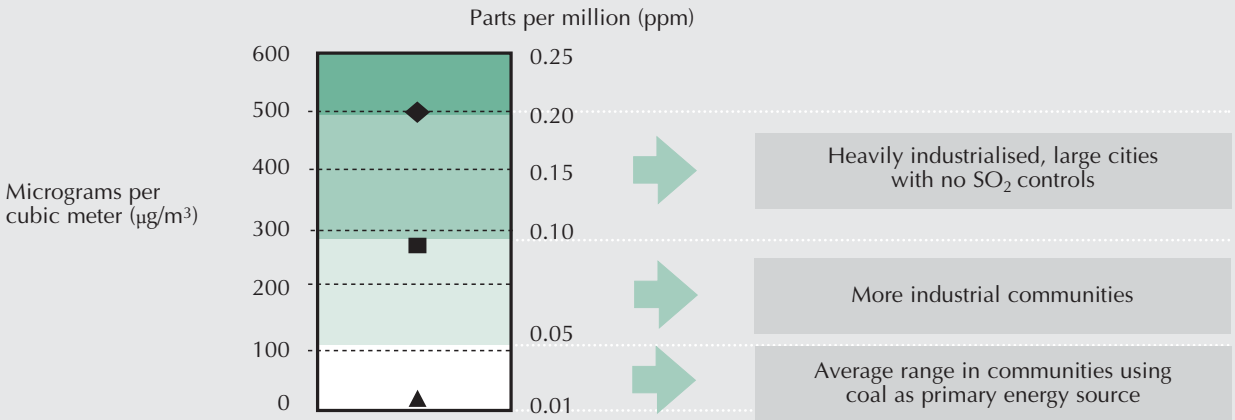
DIAGRAM 9

Industrialisation and SO_2



- Asthmatics react to SO_2 greater than 0.2 ppm with increased incidence of breathing difficulties.
- Children may suffer increased incidence of respiratory diseases at concentrations of 0.01-.05 ppm.
- At the levels of 0.01 ppm, some individuals may suffer from a reduced capacity to combat viral or bacterial infections.

Calculating your assessments

If you have a small number of large sources, a map of your city will help.

- For short stacks (<20 metres) the effects of SO_2 can be concentrated within two kilometres of the facility.
- For medium stacks (20-40 m) the range of effects will increase but the concentrations will be more dilute.
- For tall stacks (>40 m) there will be little local concentration effect.

Determining the population within the range of greatest influence will help identify the possible case numbers for your assessment.

If you don't know your SO_2 levels, look for these signs:

- Severe SO_2 pollution (greater than 0.2 ppm and especially in the presence of elevated ozone) affects plant growth. If you know that your community or an adjacent community burns significant coal, and there is evidence of plant growth damage, then your community falls in the range of high effects.
- SO_2 above 0.3 ppm has a distinct taste.
- Chronic SO_2 leads to acidification in unbuffered lakes.
- Depending upon humidity levels, SO_2 decreases visibility.

Oxides of nitrogen

Oxides of nitrogen (NO_x) are formed in all combustion processes.

Sources

- Large combustion sources — utility boilers and industrial combustion;
- Domestic and commercial boilers;
- Transportation — vehicles.
  (NO_x is also a significant indoor air pollutant, especially when gas appliances [natural gas or bottled gas] are in use.)

Exposure

The primary route of exposure is by inhalation. However, NO_x is also a precursor to acidic precipitation leading to ecological impacts such as the acidification of aquatic resources and the leaching of micronutrients from the soil.

Effects

NO_2 affects pulmonary function, with the greatest impacts seen in those with asthma or chronic obstructive pulmonary disease. There is also evidence that NO_x increases the incidence of respiratory disease in children.

DIAGRAM 10

Tracing the effects of NO_x

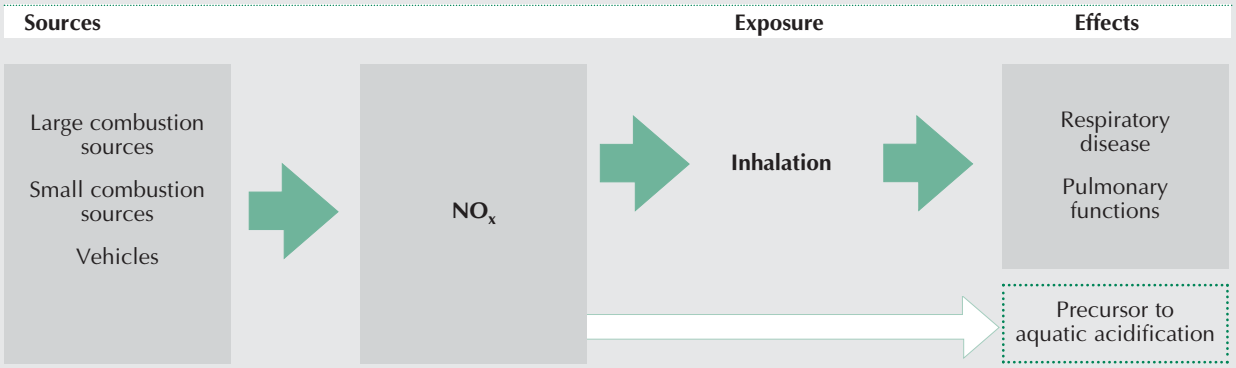


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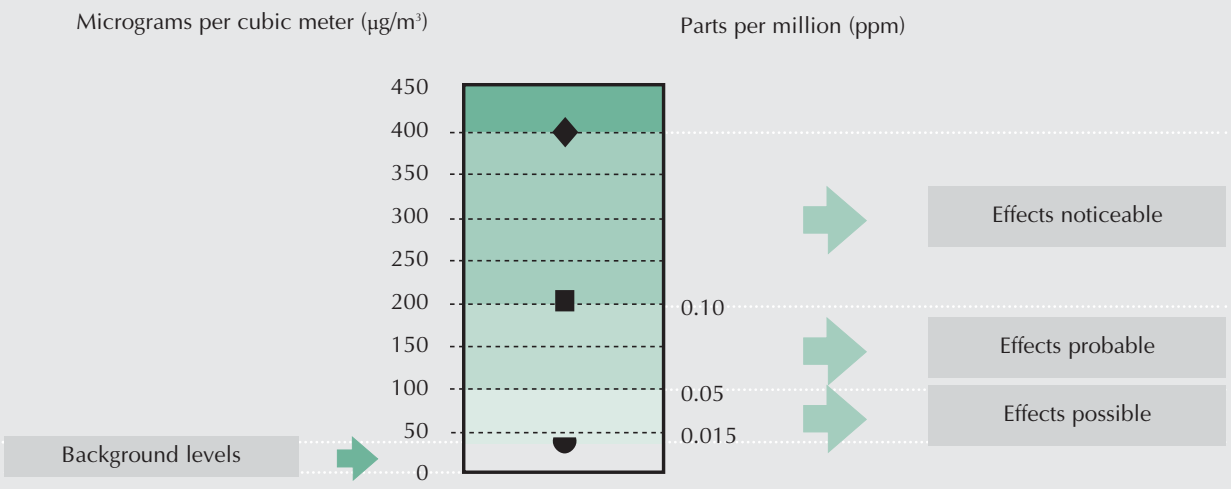
DIAGRAM 11

Likelihood of effects of NO_x according to concentration levels



Calculating your assessments

An initial assumption is possible for the portion of the population that is susceptible to the effects of NO_x. About 3 percent of the childhood and 1 percent of the adult population may be asthmatic. Similarly, 0.1 percent of the population may be suffering from more extreme pulmonary function problems. This information may be useful if no locally relevant information is available.

Using either local data or the assumptions, you can calculate the number of cases of increased asthmatic attacks keeping in mind that populations exposed to levels greater than 200 µg/m³ will lead to noticeable cases, populations exposed to levels higher than 100 µg/m³ will offer a probable estimate of impact and populations exposed to levels higher than 35 µg/m³ will provide a measure of possible impact.

If you don't know your community exposure, look for this sign:
A brown or yellow colour in the air (easily noted when flying at altitudes of 1,000-2,000 meters) is a sign of significant NO_2 (>0.2 ppm) .

Carbon monoxide

Carbon monoxide (CO) is the result of combustion in the absence of sufficient oxygen. Older, less efficient facilities, including automobiles, small stoves and industrial boilers, may be primary sources of CO generation.

Sources

Any combustion source is a potential source of carbon monoxide. Older, less efficient devices produce more CO than more modern devices. Older vehicles and boilers are a primary cause. A significant non-environmental source of CO is cigarette smoking.

Exposure

Carbon monoxide is inhaled through the air.

Effects

Carbon monoxide exerts its effect by displacing oxygen from haemoglobin in the blood. Therefore, the effects of carbon monoxide poisoning are related to asphyxiation. At very high concentrations death results. At lower concentrations, psychomotor skills are affected, impairing the ability to perform delicate tasks. For those with heart disease, the reduced availability of oxygen can cause angina (chest pain) .

If you do not know the CO concentration, look for these signs:

- significant automobile traffic;
- low wind conditions; and
- increased hospital admissions for chest pain (especially noted during cold days) .

DIAGRAM 12

Tracing the effects of carbon monoxide

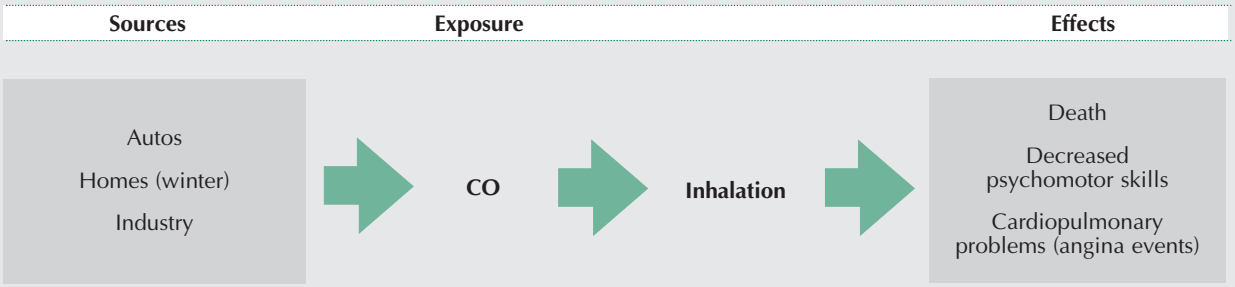
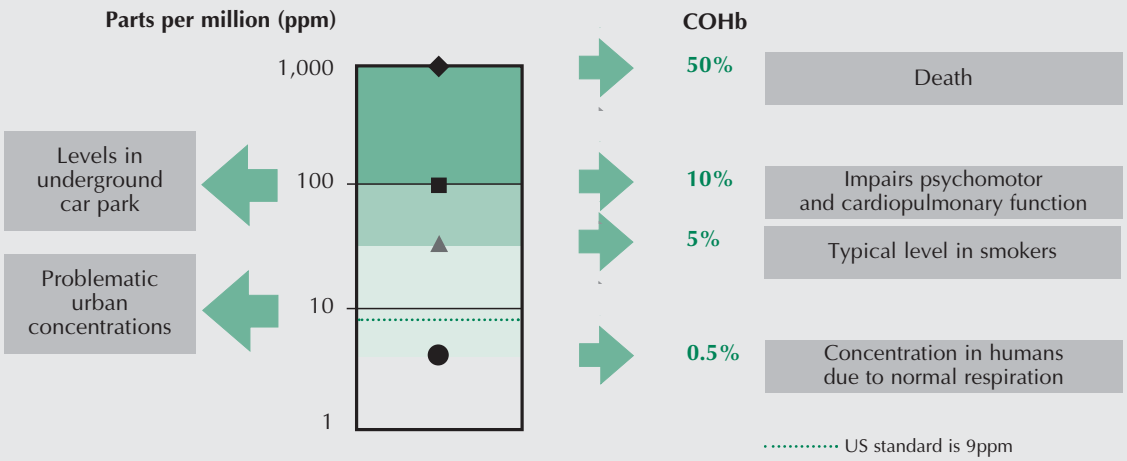


DIAGRAM 13

Dangers of high carboxyhaemoglobin (COHb) levels



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Particulates

Particulates are air pollutants of small size (less than 10 microns) that may lodge in the respiratory system, causing or aggravating health problems. Particulates have been associated with asthma, chronic bronchitis, pneumonia and premature death. Particulates in the air are measured by weight. For example, 100 µg/m^3 PM-10 describes 100 micrograms of particles smaller than 10 microns in diameter, within a cubic meter of sampled air. Smaller sized particles do more damage because these smaller particles enter more deeply into the lungs. Standards for smaller particles (PM-2.5) are set lower than for particles between 2.5 and 10 microns in size.

Sources

Particulate matter less than 2.5 microns in diameter are referred to as fine particles. Sources of fine particles include industrial and residential combustion sources as well as vehicle exhaust, so their composition varies widely. Fine particles can also be formed when combustion gases are chemically transformed into particles. Particulate matter larger than 2.5 microns in diameter are considered to be coarse particles. Coarse particles have many sources, including wind-blown dust, vehicles travelling on unpaved roads, the handling of materials, and crushing and grinding operations.

Exposure

Natural sources of particulates yield concentrations of 5-10 µg/m^3 for PM-10 and 1-5 µ/m^3 for PM-2.5. The health-based standard in the United States for annual average concentrations is 50 µg/m^3 for PM-10 and 15 µg/m^3 for PM-2.5. Twenty-four hour maximums are 150 µg/m^3 and 65 µg/m^3, respectively.

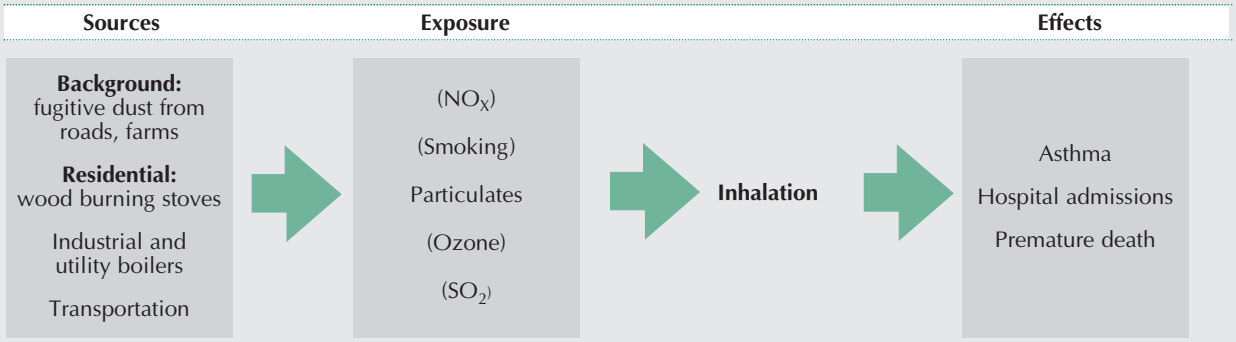
Effects

Asthma

Asthmatics represent about 2 percent of the community population in Europe, and children are

DIAGRAM 14

Tracing the effects of particulates



especially susceptible to its effects. There is consistent evidence that moderate exposure causes decreased lung capacity, which results in shortness of breath, requiring the use of inhalers or other treatment. At higher exposures, studies have documented decreases in lung capacity of up to 30 percent.

Hospital admissions

In many studies, the number of people admitted to the hospital for respiratory problems has been correlated with events of high particulate matter in the air. In cases of moderate exposure, there is some evidence of increased hospital admissions, but these exposures tend to be longer in duration, and thus it is more difficult to observe changes in admission rates. The default assumption is that there is a linear effect. At higher exposures, there are noticeable increases in hospital admissions — about 10-20 percent. This rate can be used to generate an estimate for additional hospital admissions for a specific population.

Premature death

Studies also show that death rates increase during events of high particulate matter concentrations. These deaths are among those with respiratory problems, and in many cases the deaths are accelerated from a natural disease progression. (The patients would probably die in the near term even without particulates pollution, but the elevated particulate levels hasten the death.) Significant increases in death rates have been documented in studies focusing on high exposures to particulates. These increases, which range from 2-10 percent, can be used to estimate the rate of premature death due to particulates for a specific population. Moderate exposures may increase the death rate, but statistical validity is more difficult to determine.

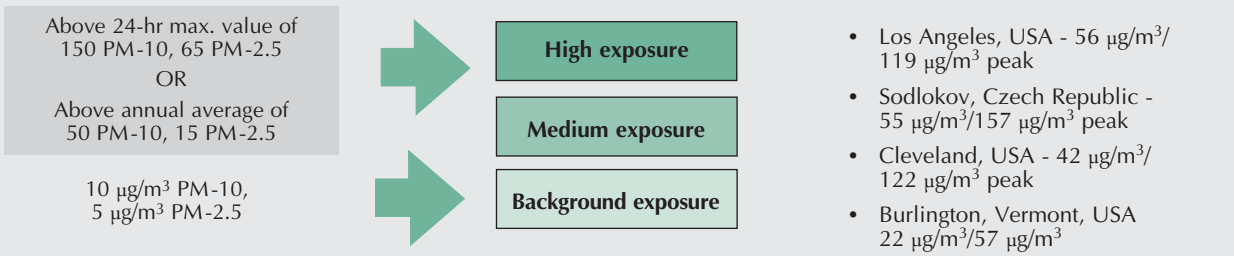
Calculating your assessments

If you don't know your community exposure, look for these signs:

- Soot deposited on parked cars on a daily basis is a possible indicator of high exposure.
- Deposits that are only apparent over longer time frames suggest moderate exposure.

DIAGRAM 15

Particulate levels in selected areas



Please check this table carefully!!!!!
What do hyphens and slashes on the right mean?

Volatile organic compounds

Volatile organic compounds (VOCs) are derived from many sources and have many properties. As a result, it is difficult to generalise about the risks associated with the entire class of compounds. However, there are certain common characteristics and clusters of compounds that share attributes that make describing them in a single assessment useful.

The following is a brief list that includes many of the VOCs with the greatest exposures in the environment:

Petroleum related
- Benzene
- Toluene
- Xylene
- Methyl t-butyl ether

Solvents
- Chloroform
- Carbon tetrachloride
- Formaldehyde
- Methanol16:

Feedstocks
- Vinyl chloride
- 1,2-butadiene
- Styrene

Sources

Many VOCs are petroleum products released during the handling or incomplete combustion of petroleum fuels. Therefore, each vehicle fueling location and each vehicle is a source, as well as the large sources associated with wholesale transfer and refining.

Several other VOCs are used as solvents in industrial or commercial processes. In these latter cases, the number of sources is large and their characteristics are varied.

A final class is chemical feedstocks. These chemicals are used in the production of chemical products. These sources tend to be localised.

Exposures

VOCs are present in the air, but also can be found in drinking water.

Effects

Each organic compound has different toxicological properties, but we will focus on cancer risks for this general assessment. Several VOCs are carcinogenic, and many of them cause liver damage, but liver damage is rarely evident from exposures encountered in outdoor air.

In the absence of detailed information about specific VOC concentration it is better to consider whole classes of VOCs.

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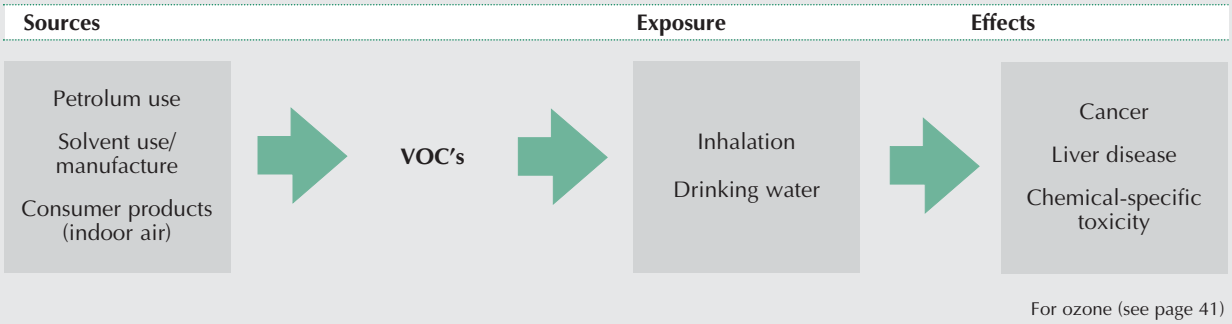
DIAGRAM 16

Tracing the effects of VOCs



For ozone (see page 41)

Calculating your assessments

VOC generation is dependent on three factors: the industrialisation of a community, the extent of vehicle use (particularly older vehicles) and the population. Diagram 15 gives some estimates for typical concentrations found in industrialised areas. If you have large numbers of VOC-generating companies, then you might expect to see these kinds of effects. If your VOC-generating facilities are limited, there may be localised effects.

Look for these signs:

- Ozone formation;
- Odours apparent (may best be recognised by visitors);
- Existence of fuel transfer or chemical manufacture;
- Chronic symptoms of VOC exposure (headache, nausea) in some members of your community.

TABLE 7

Cancer-causing VOCs

Chemical	**Typical exposure scenario**	**Concentration**	**Excess cancer –** number per million population/ lifetime (assuming continual exposure)
Benzene	Near fuelling station	4µg/m3	30
Carbon tetrachloride	Near manufacturing facility	1µg/m3	15

TABLE 8

Non cancer-causing VOCs

Chemical	**Typical exposure scenario**	**Concentration**	**Effects**
Toluene	Solvent use (ambient concentration outside of occupational exposures are slight)	1ppm	Possibility of liver damage is minimal, but toluene-specific narcotic effects are possible with long-term exposure
Formaldehyde	General exposure	1µg/m3	No known effects except the possibility of problems in sensitive populations through combination with other pollutants

Shouldn't this be expressed in the same unit?

Radiation

Atomic nuclei (especially heavy atoms) degrade, releasing high-energy particles and waves known as radiation. Ionising radiation has enough energy to alter biological molecules, including DNA. Ionising radiation includes X-rays, gamma rays, beta particles and alpha particles. Non-ionising radiation does not change the structure of molecules but can still cause damage by localised heating, such as when ultraviolet radiation causes sunburn.

Sources

Natural

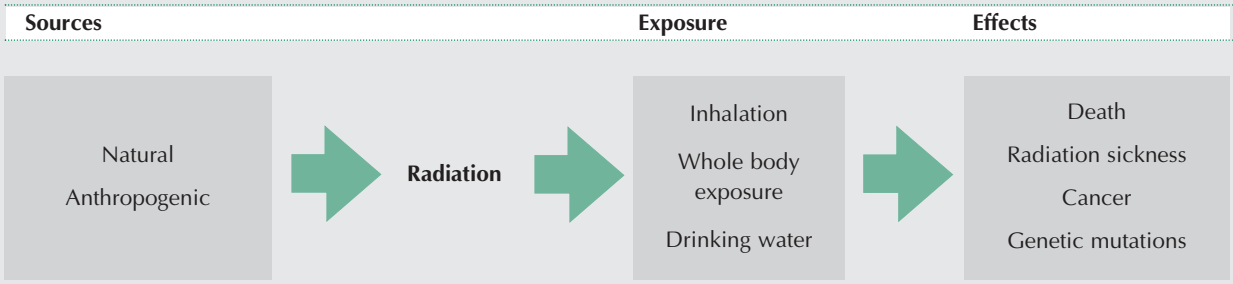
There are significant sources of natural ionising and non-ionising radiation. For most communities, these natural sources provide 90 percent of the total exposure. The natural sources include radon from soil and groundwater, other radiation from local geology and cosmic radiation. Variations in natural levels can be significant depending on local geology and altitude.

Anthropogenic — medical sources

Radiation used in medicine accounts for a large percentage of human-induced radiation. Most exposure is from diagnostic x-rays. Physicians use x-rays in more than half of all medical diagnoses to determine the extent of disease or physical injury. Routine medical exposures account for about 15 percent (53 millirems) of background sources. Radiation is also used in cancer treatments to destroy diseased cells. Radiopharmaceuticals are used to locate tumours in a patient's body and to treat cancer.

DIAGRAM 17

Tracing the effects of radiation



is this alright as is?

Anthropogenic — other

- Nuclear power generation — Nuclear power facilities release low levels of radiation during their normal operations (typically less than 1 percent of background levels). Workers receive higher doses than the general public, but the most significant exposures occur during accidents, which can inflict lethal doses on workers and significant long-term doses on the general public;
- Mining activity;
- Nuclear weapons manufacture;
- Research.

Exposure

Any release of radioactive material is a potential source of radiation exposure to the population. In addition to exposure from external sources, radiation exposure can occur internally by ingesting, inhaling, injecting or absorbing radioactive materials. Both external and internal sources may irradiate the whole body or a portion of the body. In the United States, the average person receives a dose of approximately 360 millirems (whole-body exposure) per year.

Specific case — Chernobyl

Depending upon the age at the time of exposure, an acute accidental dose of 25,000 millirems of radiation increases the risk of premature death by 20-21 percent. The Chernobyl accident resulted in 237 cases of acute radiation syndrome (ARS) , of which a total of 38 have died, 28 in the acute period in 1986, and 10 in later years due to various causes. There have been no documented cases of cancer in patients with confirmed ARS, though studies of exposed children have shown incidences of thyroid cancer. Based on Chernobyl, scientists estimate that a dose of 10,000 millirems received over 70 years in a population of 10,000 would result in almost double the cases of thyroid cancers, about a 40 percent increase in leukaemia and about a 3 percent increase in all cancer deaths over those 70 years.

Human health effects

Radiation affects people by causing cell damage or cell death. Large doses alter cellular systems and can result in death. With smaller doses, the person or particular irradiated organ(s) may survive, but the cells are damaged, increasing the chance of cancer. The extent of the damage depends on the total amount of energy absorbed, the time period and dose rate of exposure, and the particular organ(s) exposed.

All people are chronically exposed to background levels of radiation present in the environment. Many people also receive additional chronic exposures and/or relatively small acute exposures. For populations receiving such exposures, the primary concern is that radiation could increase the risk of cancers or harmful genetic effects.

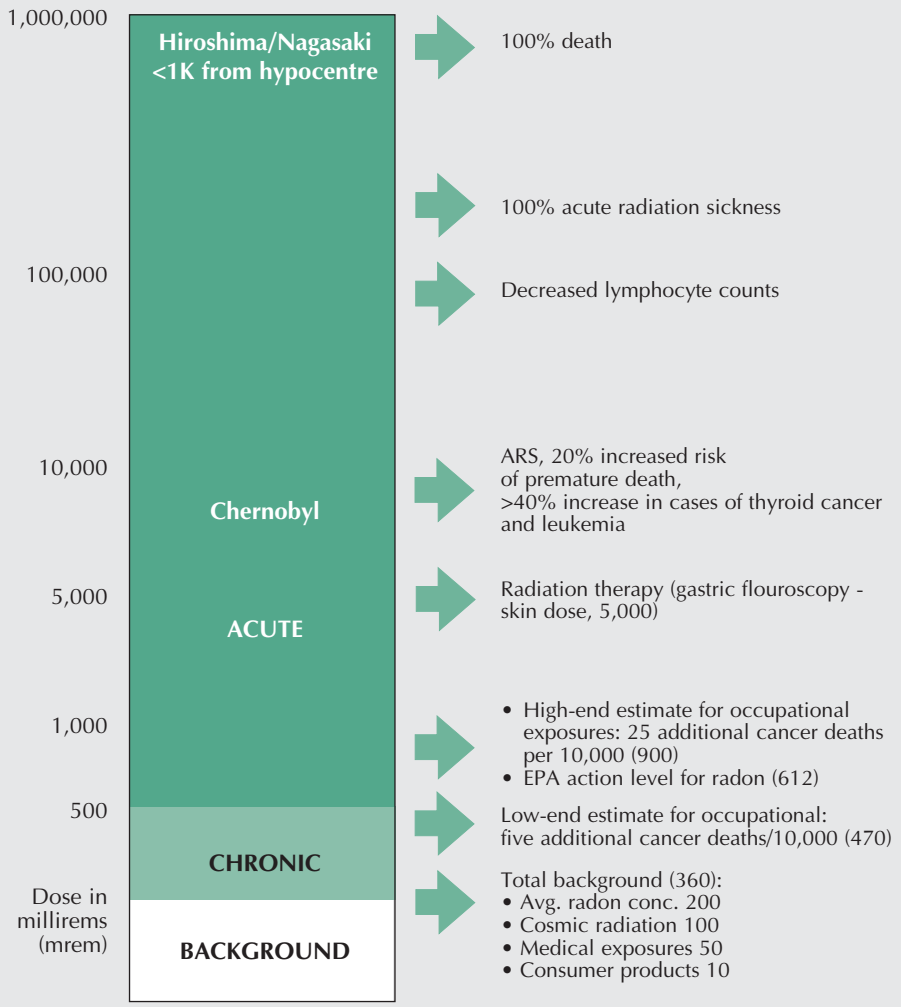
Chronic exposure is continuous or intermittent exposure to low levels of radiation over a long period of time. Chronic exposure is considered to produce only effects that can be observed some time following initial exposure. These include genetic effects and other effects such as cancer, precancerous lesions, benign tumours, cataracts, skin changes and congenital defects.

Acute exposure is exposure to a large, single dose of radiation, or a series of doses, for a short period of time. Large acute doses can result from accidental or emergency exposures or from special medical procedures (radiation therapy). In most cases, a large acute exposure to radiation can cause both immediate and delayed effects. For humans and other mammals, acute exposure, if large enough, can cause rapid development of radiation sickness, evidenced by gastrointestinal disorders, bacterial infections, haemorrhaging, anaemia, loss of bodily fluids and electrolyte imbalance. Delayed biological effects can include cataracts, temporary sterility, cancer and genetic effects. Extremely high levels of acute radiation exposure can result in death within a few hours, days or weeks.

Calculating your assessment

DIAGRAM 18

Effects from various levels of radiation



Other toxic chemicals

Lead

Lead has many uses in consumer products and industry, and as a fuel additive. Lead is also toxic and enters the human body readily through breathing polluted air or through accidental ingestion of lead-contaminated dust.

A primary effect of lead poisoning is changes in neurological development. This is particularly evident in children because of their rapid development and enhanced susceptibility to its effects.

Sources

There are three main sources of concern for lead contamination:

- burning leaded gasoline;
- lead smelters (and other lead handling facilities, such as battery factories and leaded gasoline refineries); and
- lead in paint.

Several secondary sources may be important in special cases such as the continuing use of lead pipes to deliver drinking water.

DIAGRAM 19

Tracing the effects of lead

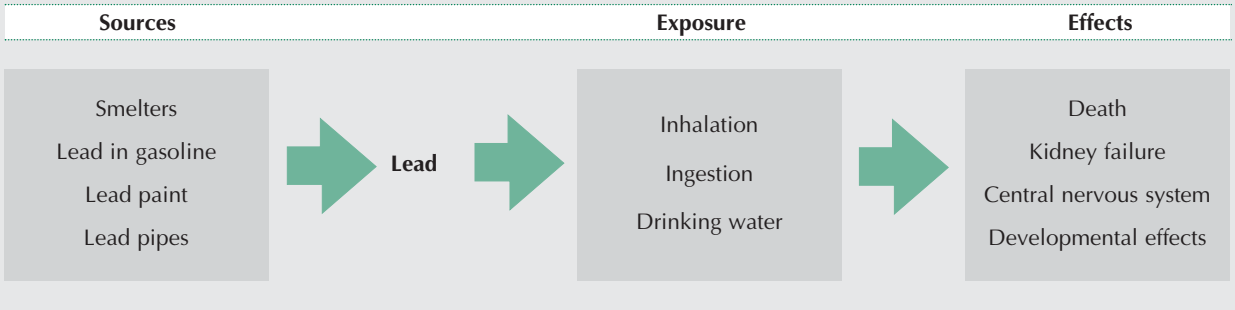
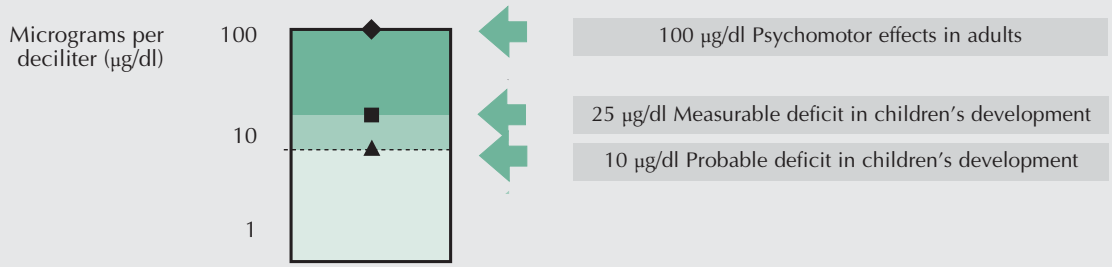


DIAGRAM 20

Effects of lead poisoning at different levels



Exposure

The standard method for determining lead exposure is by measuring the lead in the blood of potentially affected individuals (particularly children) . The standard measure of lead is micrograms per decilitre of blood. In the absence of information about blood-lead levels, there are other observations suggesting the possibility of lead poisoning.

Lead smelters

The existence of lead smelters, particularly those that are older and have limited pollution controls often leads to significant contamination of the air and soil in a community. These effects are most pronounced within five kilometres of a facility, and the most severe impacts occur down from prevailing winds.

Lead from gasoline

In areas where there is extensive automobile traffic, particularly idling vehicles, there can be significant contamination of soil and air with lead.

Inhalation

When the primary source of exposure to lead is inhalation, 1 µg/m^3 leads to an elevation of blood lead of about 7 µg/dl.

The diagram below gives some indication of the extent of lead poisoning in different communities.

Lead in paint

A different source of lead is evident inside houses that may have been painted with lead containing paint. In those houses, either peeling paint ingested by children or renovations that take place with significant sanding can cause extensive blood lead poisoning.

Calculating the assessments

In most cases, your assessment can focus on children. If the problem is from a smelter or vehicle use of leaded gasoline, a review of a local map can identify potential hot spots. Determining the number of children that live within the area of greatest concern is one part of the assessment. However, airborne contaminants distribute readily so that there will be community wide population concerns as well. The lead exposure to the childhood population for your entire city may be 50-60 percent of the levels of those nearest your most contaminated site. The "hot spots" will have a higher frequency of highly poisoned individuals.

In the case of leaded paint, a review of local housing stock can provide a good start for determining the extent of the problem. If a predominant portion of the housing has been treated with lead paint, then the risks are significant for most households, with a particular risk in houses that are in the process of renovation.

To get a general sense of impacts, consider the following possibilities

Greater than 25 µg/dl

- High — 10 percent of your children, if you have an active smelter (or other sources generating ambient concentrations of >2 µg/m^3);
- Medium — 2-3 percent combined ingestion of lead paint and high vehicle traffic (sources generating concentrations of 1-2 µg/m^3);
- Low — Less than 1 percent if you have few industrial sources and low housing problems (sources generating concentrations of <1 µg/m^3) .

Special Cases — Any homes undergoing renovation can expose children to lead causing elevated levels of the element in their blood.

Greater than 10 µg/dl

- High — Extensive leaded gas use can lead to more than 50 percent of children in urban areas with levels greater than 10 µg/dl
- Low — If there is no extensive use of leaded gas or other significant industrial source, fewer than 10% of your children should have blood lead higher than 10 µg/dl.

If you don't know your lead levels, look for these signs:

- If leaded gasoline is the primary fuel for automobiles and you have more than 50,000 vehicles in a 100 square kilometres area, the experience in the United States suggests a significant portion of your children (30-40%) will have blood lead greater than 10 µg/dl;
- If smelters are present;
- If housing stock has deteriorating dried paint — acute cases (more than 25 µg/dl) are possible.

Pesticides

Pesticides have the intended effect of toxicity towards unwanted pests. Unfortunately, that toxicity makes pesticides a significant environmental contaminant as well.

Sources

There are three categories of sources with very different potentials for exposure:

- The manufacture of pesticides provides the potential for catastrophic exposure and chances for worker exposure.
- Agricultural application can be a source for large concentrations of pesticides.
- The domestic use of pesticides is more dispersed, but it also provides many opportunities for environmental contamination.

Exposure

Pesticide assessments are challenging because of the many routes of exposure that are possible. During application and manufacturing accidents, airborne contamination is possible. Contaminated food is an important route for exposure because many pesticides are used in the production of food. Drinking water contamination is also a route of exposure, especially important when drinking water sources are near agricultural areas.

Effects

Different classes of pesticides have different effects. For the purpose of this analysis, we will focus on immune system responses from chronic exposure. Many other effects such as neurological damage are possible from acute exposure.

Acute effects result when large quantities of pesticides are inhaled or ingested. This usually occurs during accidental exposure from manufacturing or application error. We will not consider these effects here as the results are usually case specific.

Calculating your assessment

The intensity of pesticide use is one of the most important factors in determining the potential exposures through drinking water and the consumption of local produce.

There is a range of pesticide use represented by different agricultural economies. Comparing your local agricultural practices with some of those in Western Europe may provide a gauge for the intensity of pesticide use. Table 9 on page 66 identifies four nations with differing intensities of agriculture and pesticide use.

If they are available, figures for nationwide pesticide sales and agricultural acreage can give a picture of your country's overall pesticide use. Then you should consider the relative use of pesticides on farms near you.

Specific cases

- The application of 23 kilograms per hectare per year of pentachlorophenol in Moldova results in measurable helper T-cell reductions and leads to increased infection rates of two to five times the non-exposed population.
- Uzbekistan cotton farmers have increased susceptibility to respiratory, gastrointestinal and kidney infections due to the application of metal containing pesticide residue.

Other notes

- There are many pesticides, each with its own toxicity and effects.
- Acute poisonings occur, affecting chemical plant workers and applications.
- Chronic poisoning is much more difficult to identify.

If you do not know your community exposure, look for these signs:

- Is your water supply near farmland or industrial facilities?
- Do your farmers use pesticides?
- If so, are there unexplained fish kills?

DIAGRAM 21

Tracing the effects of pesticides

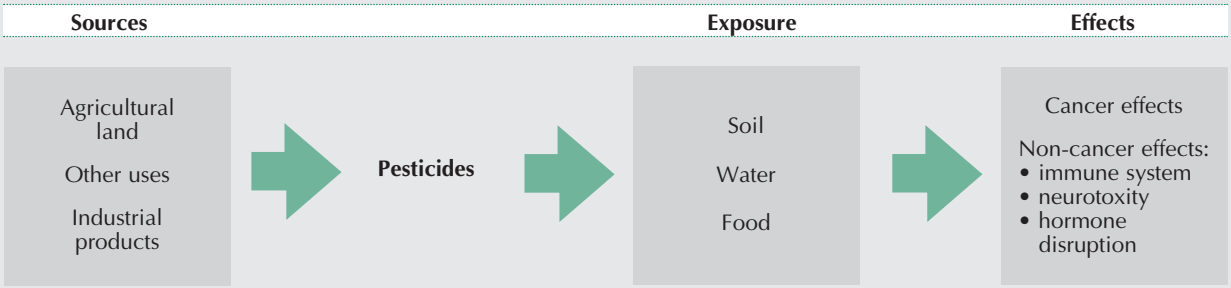


TABLE 9

Pesticide use in selected Western European countries

	FRANCE	AUSTRIA	ITALY	GERMANY
Cultivated land (thousands of hectares)	1,300	90	3,300	210
Fungicides (tonnes)	45,000	1,500	50,000	9,000
Herbicides (tonnes)	28,000	1,700	10,000	16,000
Insecticides (tonnes)	8,000	200	10,000	4,000
Total	75,000	3,300	70,000	30,000
Tonnes per 1,000 hectare	57.69	36.67	21.21	142.86

Data from the United Nations Environmental Programme

Please add a year to this!

Solid waste

The impacts from solid waste are some of the most noticeable in community projects. Everyone generates solid waste, and most members of the community are aware of the impacts.

Effects

The range of impacts from solid waste makes the issue difficult to assess, but it is important to understand the possible risks to the community.

Land use

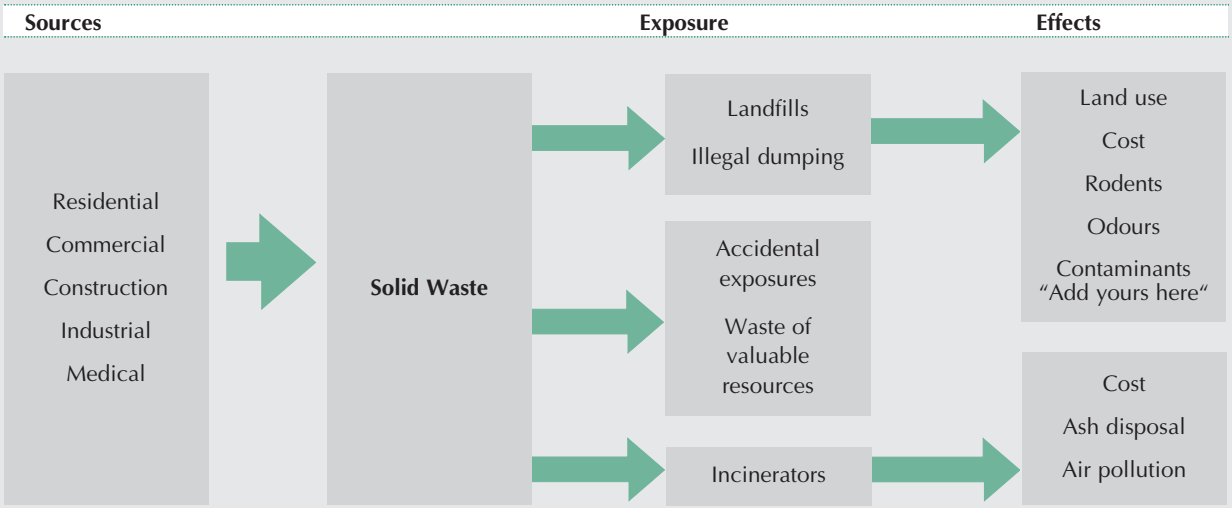
In urban areas, the need for transfer stations, landfills and incinerators can be a significant strain on the available land.

Vectors of disease

Poorly managed waste is a home for disease carriers such as flies, rats, birds and microorganisms.

DIAGRAM 22

Tracing the impacts from solid waste



Cost
The construction of new facilities, the transportation of waste to a facility and the management costs for a facility are all factors contributing to the normal costs of operation. There are also costs associated with the clean-up of contaminated water or soil that may result from poorly managed facilities.

Odours
Odours are a common complaint of solid waste management.

Air Pollution
The burning of municipal or special wastes often results in the release of toxic air pollutants. Unfortunately, the range of pollutants from burning waste is so large that assessment is difficult. In many cases, focusing on a few pollutants provides a starting point for considering the assessments.

Community
The presence of waste, waste management facilities, odours and costs can cause tension in a community. Proper management of waste includes the consideration of these community factors.

Calculating your assessments
Unlike most assessments, most of the impacts from solid waste management can be considered qualitatively by answering a few questions:

Land use
- If your waste is managed locally is the capacity of the landfill enough for future waste generation?
- If the capacity will not accommodate future solid waste generation, is there additional capacity available locally?

High risk: Insufficient capacity, limited land available

Low risk: Sufficient capacity or sites available

Carriers of disease
Are species associated with solid waste present (sea gulls, rats, flies)?

High risk: Vectors observed near residential communities (the potential for transmission of human disease is significant)

Medium risk: Vectors are observed but not near residential communities

Low risk: No vectors observed

Cost

- Are you aware of disposal costs in adjacent communities?
- Are they higher or lower than in your own municipality?
- Are there future considerations that will result in higher costs?

Odours

Are odours apparent from solid waste management facilities?

High risk: Odours apparent in residential or commercial communities (may effect property values and potential economic development)

Low risk: Odours not apparent to residential or commercial communities

Air pollution

- Does your community burn waste?
- If so, when was the facility designed?

High risk: Burning waste in an old (more than 20 years old) facility or open burning

Medium risk: Burning waste in a new facility

Low risk: No burning

Community

Has the subject of solid waste management been controversial in your community?

High risk: Yes

Low risk: No