ProSPeReS

INTRODUCTION TO CBRN THREATS

a guideline with basic information on CBRN

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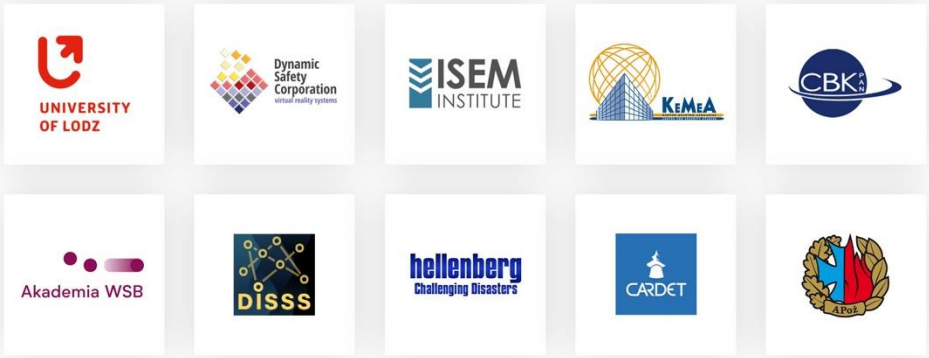
The ProSPeReS Consortium

Security experts, security research and academic institutions, providers of technical solutions and services



Law enforcement agencies (LEAs)

Faith-based organizations

1. Executive Summary

The **PRoSPeReS** project aims to support the implementation of the EU Action Plan to improve the protection of public spaces, in particular places of worship. The project is also part of the "*Action Plan on the Protection of Places of Worship: united and in solidarity for safe and peaceful worship*" published in September 2019.

The **PRoSPeReS** project serves to raise the level of protection of religious places by synergising the scientific knowledge of academia and the empirical knowledge and experience of security specialists (practitioners), public service officers and representatives of religious institutions (representing the Catholic Church, the Orthodox Church and the Jewish community) in preparing a comprehensive protection system. This system includes measures to improve prevention, protection, minimisation and response to various types of terrorist threats and incidents that may occur at places of religious worship, including attacks using CBRN (chemical, biological, radiation and nuclear) agents. “Measures" should be understood here as sets of tools, procedures, equipment, guidelines for infrastructure improvement and protocols for cooperation with public services adapted to a specific type of threat (scenario)

The main objective of the **PRoSPeReS** project is to create an integrated security system that will improve the security of places of worship in EU member states. The project itself focuses on both prevention and response to terrorist threats that may occur at such sites

The achievement of the main objective of the **PRoSPeReS** project is possible through the implementation of specific objectives, including the development of a handbook to raise the awareness of persons and representatives of institutions responsible for the security of places of worship, including potential scenarios for terrorist attacks, such as 'lone wolf' attacks, organised attacks, as well as attacks using various types of weapons and explosives.

This report “*D4.1 Introduction to CBRN threats - a guideline with basic information on CBRN*” fulfils the prerequisites of just such a handbook.

2. Introduction

According to the European Commission Glossary, the term "**CBRN**" is an abbreviation from "**chemical, biological, radiological, nuclear**". It represents four main threats that could harm the society through their deliberate release, dissemination or impact. CBRN term replaced terms used in the cold war era: NBC (nuclear, biological, chemical) and ABC (atomic, biological, chemical). This change comes from distinction in threats represented by fissile material and nuclear bombs on the one hand, and dispersion of radioactive material on the other. Currently, there is "e " added to CBRN abbreviation to emphasise that explosive substances and devices are an important part of CBRN threats as a way of spreading particular types of agents.

First responders distinguish incidents with dangerous materials between two types: hazmat (abbreviation from hazardous materials) incidents and CBRN incidents. Main difference in these two types of incidents is their cause. **In hazmat accidents, release of dangerous chemicals, bio agents or radiation is caused by accidents in using, stocking or transporting hazardous materials**. For example it could be the release of chlorine from a rail tank after derailment or explosion of wrongly stockpiled ammonium nitrate. There could also be human factors contributing, but in an unintentional manner. **CBRN threats are characteristic due to the fact that release of dangerous substances is intentional and designed to cause harm and induce fear**.

CBRN incidents could be conducted by state actors, non-state actors, organised crime groups or even by so-called "lone-wolfs". No matter what kind of organization or individuals stand behind CBRN incident, it is always terror act in nature. In general, a CBRN terrorist attack in European region is considered as low probability but potentially high impact on society when it happens. Law enforcement from European region consider the greatest threat come from jihadist and far-right elements, especially from small cells of these organizations or lone actors working autonomously. On-line discussion about use of CBRN agents has been observed, first focus was on chemical weapons but after 2019 main focus is on biological agents.

Ongoing COVID-19 pandemic clearly shows that European society is not fully prepared for an all-hazards scenario with the need to strengthen its resilience. Naturally occurring pandemic also shows us how hard it would be to respond and contain deliberated release of biological agents.

Figure 1 - Symbols of CBRN threats: radiological, biological, chemical

Source: https://nbn.business/cbrn-management/

2.1 Examples of CBRN attacks

History knows a lot of different situations where dangerous chemicals or pathogens were used in order to kill, hurt or threaten people. This kind of usage of dangerous materials fits well in frames of CBRN attack. In the below table you can find incidents back from the Second World War era till present times.

Table 1 - Examples of CBRN incidents

Date	Location	Agent	Casualties	Description
1946	Germany	Cyanide	2,283 injured	poisoning of bread
1984	USA	salmonella	751 injured	poisoning of food in several restaurants
1987	Philippines	pesticides	19 killed, 140 injured	water poisoning
1994	Japan	sarin	7 killed, 270 injured	release of CWA in city
1995	Japan	sarin	12 killed, 5511 injured	release of CWA in subway
2001	USA	anthrax	5 killed, 17 injured	letters with anthrax to officials and media
2003	Iraq	sulphur	41 injured soldiers, unknown number of injured civilians	arson of sulphur stockpiles
2004	Iraq	sarin	2 injured	improvised explosive device with CWA
2006	Iraq	mustard gas	2 injured	improvised explosive device with CWA
2007	Iraq	chlorine	115 killed, 854 injured	car and truck bombs with chlorine tanks, most fatalities were from explosions, most injuries from chemicals

		mustard gas	2 injured	improvised explosive device with CWA
2010	Iraq	pesticides	672 injured	gas attack on schools
2012	Afghanistan	rat poison (arsenic)	53 killed, 40 injured	poisoning of food at police stations
2013	Afghanistan	pesticides	1952 injured	attacks on school, water poisoning
2014	Iraq	chlorine	40 injured	improvised explosive device with CWA
2015	Iraq/Syria	chlorine	30 injured	truck bomb with chlorine canister
2016	Iraq	blistering agent	1 killed, 600 injured	attack on town
		sulphur	2 killed, 1500 injured	arson in sulphur stockpiles
2017	Malaysia	VX	1 killed	assassination of Kim Jong Nam
2018	United Kingdom	Novichok	1 killed, 7 injured	assassination attempt of Sergei Skripal
	Germany	ricin	none	foiled attack with ricin bomb
2020	USA	ricin	none	letters with ricin addressed to White House

Source: CBRN security manager handbook, Łódź 2018; EU preparedness and responses to chemical, biological, radiological and nuclear (CBRN) threats study, Brussel 2021.

Most examples of CBRN attacks took place in the Middle East region but it should be clearly distinguished - use of dangerous chemicals in terrorist acts and use of dangerous chemicals by regular armed forces in warfare. According to that distinction, not every use of chemicals in combat is considered a CBRN attack.

2.2 Classification of dangerous chemicals according to CLP and ADR

In general, it is considered that during the CBRN attack all labels and markings might be removed from containers with dangerous material, but it is not excluded that original marking will be present. In that way it is useful to know current systems of labelling dangerous materials, to be able to

decode the pictograms and possibly relate them to threats they present. Reading labels on containers is also the first thing that every first responder does when dangerous materials are involved.

In Europe there are two systems of classifying and labelling dangerous materials. Transportation of dangerous goods by cars and trucks is regulated by Agreement for Transportation of Dangerous Goods by Road (so called ADR agreement). Similar in used symbols to ADR is RID, which regulates railway transport.

According to ADR, dangerous materials are divided into nine classes and then up to six subclasses which can be found in Appendix C.

Class numbers describe every substance that is transported by road or railway. Vehicles carrying dangerous materials are equipped with orange plates divided in half. In the upper field there is so-called “Kemler code” - two or three digits describing main dangers. In the lower half there is a UN number of substances. If vehicle carries orange plate without numbers, it means there is more than one kind of material transported.

Figure 3 - Orange plates used in ADR transport for few different materials

Figure 2 - Orange plates used in ADR transport for single material, in these case - petrol

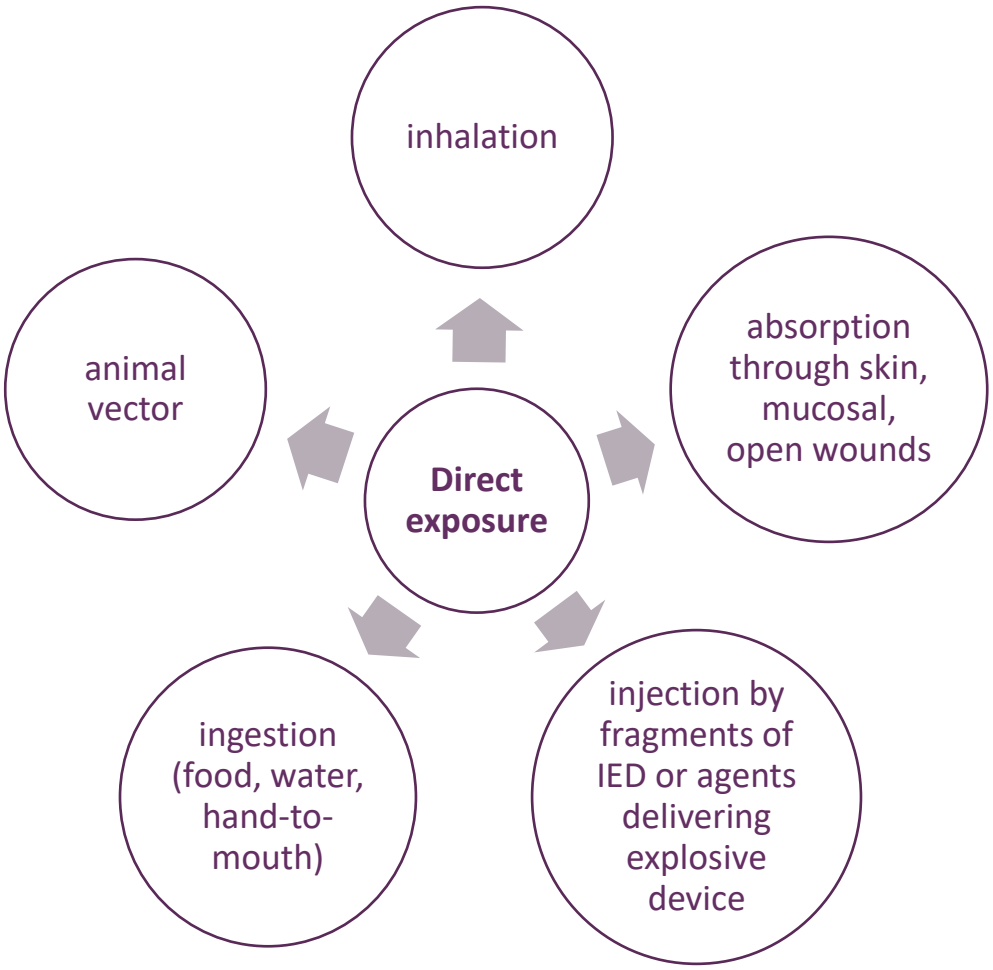
Another system of classifying and labelling dangerous substances is known as the Globally Harmonised System of Classification and labelling of Chemicals. It is an international agreement managed by the United Nations and acts as a complement to the UN numbered system used in transportation of dangerous materials. The core of the GHS system are standardised testing criteria, warning symbols and safety data sheets. The European Union implemented this system in 2008 as Classification, Labelling and Packaging Regulation. In 2017 there was a new format of pictograms implemented (white background with red edge), but old formats (orange background with black edge) could be found on older packages. Comparison of old and new CLP symbols with description of threat could be found in Appendix C.

2.3 Ways of exposure to dangerous substances

Adverse health effects from exposure to dangerous materials and agents are the main concern during and after CBRN events. Effects on affected people depend on many factors like: toxicity of agent, way of delivery, time of exposure, actual weather conditions and many more. One of these factors, which strongly influence health effects is the route of exposure.

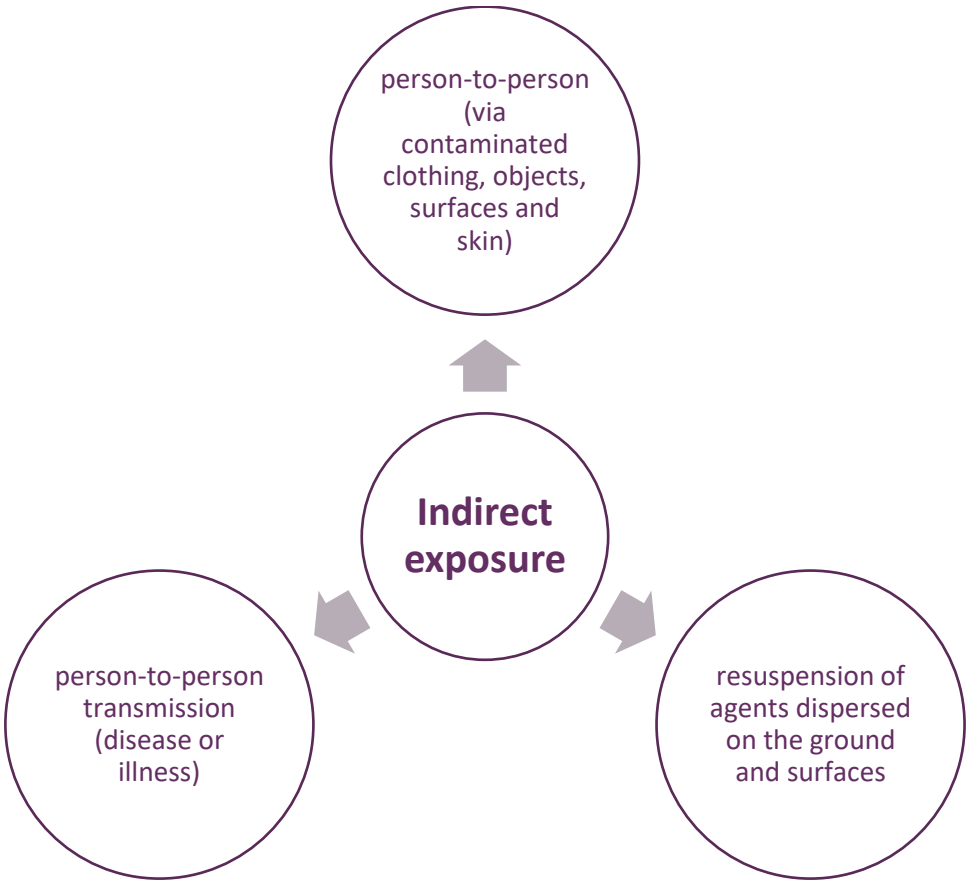
Ways of exposure could be divided into two parts: direct exposure and indirect exposure. In fact, indirect exposure is derivative of direct exposure in which the vector of contamination is different, focusing on cross contamination. Direct and indirect exposure are:

Figure 4 - Direct exposure



Source: Chemical, biological, radiological and nuclear response. Introductory guidance. ICRC, Geneva 2014r.

Figure 5 - Indirect exposure



Source: Chemical, biological, radiological and nuclear response. Introductory guidance. ICRC, Geneva 2014r.

In this guidebook we will discuss the most important routes of direct exposure as they are most relevant to unexpected releases during a CBRN attack.

Routs of Entry:

Inhalation exposure takes place when agents in the state of gas, vapour, aerosol or suspension enters the body via respiratory tract. Main threat in this way of exposure is absorption in the upper and/or lower respiratory tract. Region of absorption and its efficiency depends on solubility and particle size. Water-soluble agents are mainly absorbed in upper regions of the respiratory system whereas water-insoluble agents absorb in lower regions of the respiratory system. For particles the site of deposition depends on their size and density. Particles with a diameter of 10 micrometre and larger are mainly deposited in the upper tract. Smaller particles, 5 micrometre and below, pass beyond upper tracts and will reach alveoli which is part of the lower respiratory tract. There are also two main types of reaction after hazardous agents enter the respiratory tracts. Agents could harmfully react with tissues of upper or lower respiratory tracts or enter the bloodstream through lungs and attack specific organs or systems.

Dermal route of exposure, including skin, mucosal and open wounds, takes place when CBRN agent comes into direct contact with these elements. Skin could be exposed to dust, liquid,

vapour or aerosol. Effectiveness of absorption through unprotected skin depends on many factors. These are:

- **Anatomical differences of skin** - thickness of skin is different on various part of body
- **Age** - skin of children and elderly people is easier to penetrate
- **Temperature** - rise of temperature causes skin to produce moisture which protects against water insoluble substances but allows more water-soluble substances to come into contact with skin. Also rise of temperature causes rise of solubility of liquids and solids. For gases, the rise of temperature decreases solubility.
- **Humidity** - rise of humidity causes better contact of water-soluble substances with skin.

Specific way of exposure is via **mucosal and ocular route**. These parts of the human body are naturally prone to irritation by CBRN agents. Mucosal due to its natural moisture provide fine contact with water soluble substances. Eyes due to its mechanism of tearing are not an effective way of absorption of hazardous agents but could be the first signal of CBRN incident.

Corrosive substances like acids, bases or oxidisers could damage skin surfaces causing chemical burns. These lead to opening way for other dangerous materials via **intradermal route**. Open wounds allow hazardous agents to enter directly into the bloodstream, putting aside the protective function of skin. This is the most effective route of exposure - entering directly into the bloodstream requires lowest doses of hazardous agents to cause adverse health effects. Corresponding to that route - by broken skin - is exposure via contaminated splinters and pieces. They could be dispersed by improvised explosive devices or other means of explosive dispersion. As an example, an intradermal route of exposure was used to assassinate Georgi Markov, which was punctured with a needle containing ricin hidden in an umbrella.

Ingestion or oral route happens when CBRN agents enter an organism via mouth. This route of exposure includes following situations:

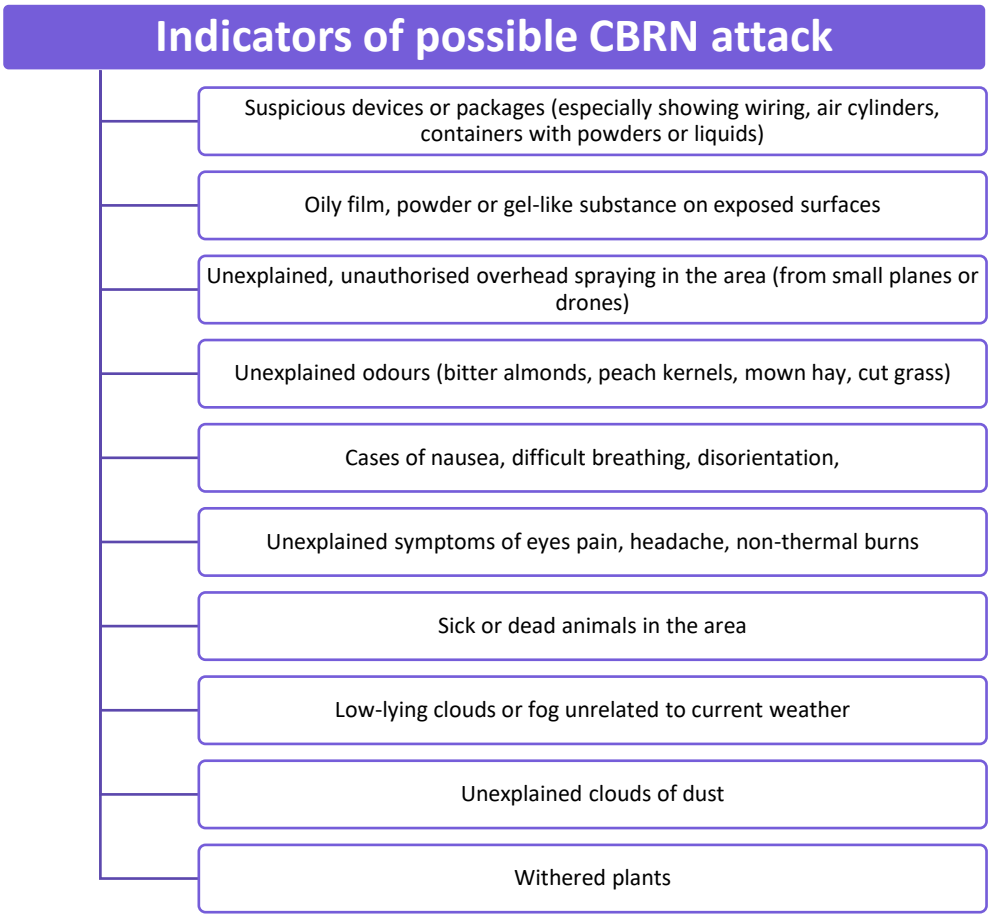
- Person touches the mouth with contaminated hands or objects (mug, utensils, cigarette, etc.)
- Aerosol of CBRN agents settles on person’s face
- Person eats or drink contaminated food and water
- Person could swallow respiratory mucus after CBRN agents' particles accumulate in the nose, throat and upper airways.

This particular way of exposure will be **most dangerous when radioactive dust (alpha emitter)** will be a CBRN agent of contamination.

2.4 Indicators of CBRN attack

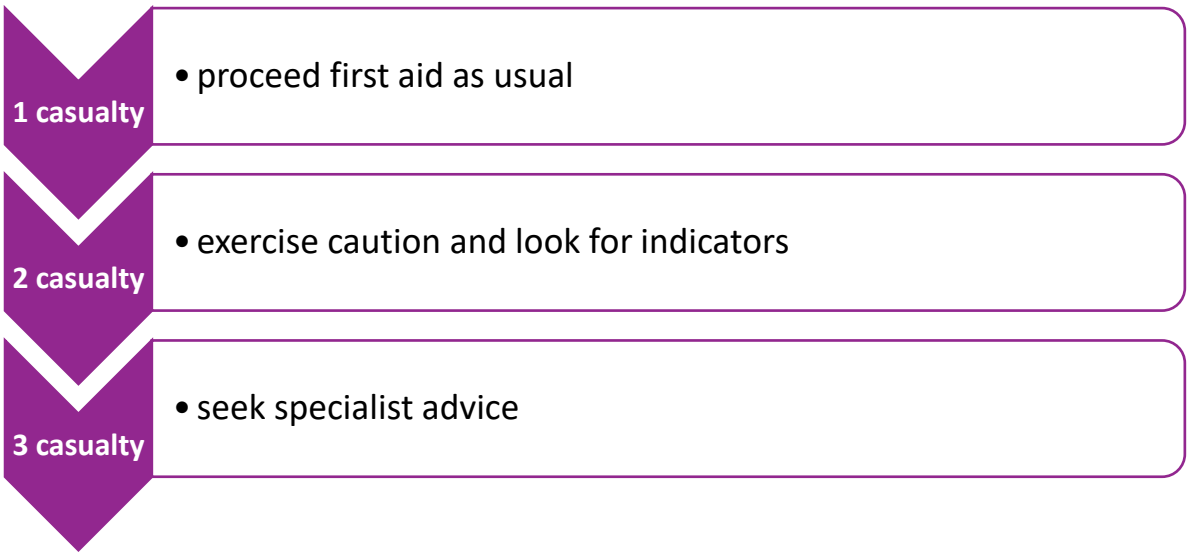
It is characteristic for CBRN incidents that it is difficult to detect or recognize released CBRN materials in initial stages. Used in attack hazardous materials could be odourless, colourless and tasteless, exposure may not be evident immediately. **There are some indicators which could point to a possible CBRN incident**.

Figure 6 - Indicators of possible CBRN attack



In recognizing CBRN incidents could be also helpful the so-called 1-2-3 casualty protocol. When there are injured people from unknown source:

Figure 7 - 1-2-3- Protocol



CBRN INCIDENT IMMEDIATE ACTIONS (Six Cs):

CONFIRM

- Put on personal protective equipment (PPE) (where available)
- Warn others nearby
- Identify possible routes of exposure (e g. food, airborne, skin)

CLEAR

- Move upwind, if gas, vapour or airborne particles
- Move to a safe distance (outside any exclusion zone)

CORDON

- Establish hot and warm (decontamination) zone
- Establish a formal clean I dirty line (CDL)

CONTROL

- Stop any eating, drinking or smoking in contaminated area
- Control and monitor re-entry and exit to I from zones
- Limit movement downwind of hazard
- Protect the area for further assessment including forensics

COMMUNICATE

- Inform Command using METHANE report and other incident report
- Warn local medical facilities and notify appropriate health authority

CONTAIN

- Prevent secondary contamination, if persistent hazard
- Prevent secondary infections, if contagious biological agent

PRINCIPLES OF CBRN CASUALTY MANAGEMENT:

- Recognition
- Safety (Six ‘C’s Confirm - Clear - Cordon - Control - Communicate - Contain)
- Self / Buddy first aid
- Triage
- Casualty assessment (Quick Look)
- Lifesaving interventions (T1 casualties only)
- Casualty hazard management (Decontaminate and/or Isolate/Quarantine)
- Supportive management (includes critical care)
- Definitive management (includes specific antidotes & antibiotics, and surgery)
- Rehabilitation

2.5 What to do after CBRN incident

After using information about CBRN incidents symptoms from previous chapter, person who is in charge of safety and security can state if there is a possibility of CBRN incident. After making that statement it is necessary to provide that information to local emergency services. During reporting the CBRN incident there should be given information described by ETHANE acronym increased by some specific intelligence. Below the list of information that should be reported is shown.

Basic information:

- E – exact location
- T – type of incident
- H – hazards that have been identified
- A – access routes
- N – number of casualties
- E – emergency service required

Complementary information:

- Why situation is suspicious?
- Is the place/building high or low profile?
- Are there any messages or intelligence about that suggest the incident?
- Who found it and when?
- Where is the threat?
- Who has had contact with suspicious material, where is that person now?
- What are local weather conditions?
- Is the threat inside the building or in the open?

If the CBRN incident is in open air people should:

- Move away from source of dangerous material, upwind and uphill,
- Assist others who are injured or less able to walk to move away from source of threat,
- Do not eat, drink, smoke or touch mouth and eyes – protect your mouth and nose
- If contaminated – remove the outer clothing
- Use personal decontamination kit if you have one (more to find about decontamination in chapter 6),
- Avoid seeking medical help on your own,
- Wait for emergency service and follow their instructions.

If the CBRN incident take place inside the building:

- Initial actions as in CBRN incident in open air,
- Evacuate affected area,
- Shout down the air conditioning,
- Gather evacuated persons in one place, do not let them spread.

3. Chemical threats

Hazardous chemicals have a long history of being used to poison individuals or groups. Through the Middle Ages and Renaissance plant extracts were mostly used as chemical agents, but it was the expansion of the chemical industry and breaking World War I to develop current understanding of dangerous chemical agents. It is said that chemical agents are the most brutal among the Weapons of Mass Destruction. Among thousands of different substances only few could be defined as a chemical weapon. Thus, substances should have high toxicity, be imperceptible to senses, rapid to action and persistent after dissemination. Chemicals that have such characteristics are listed as scheduled chemicals in the Chemical Weapons Convention. In present time, globalisation, easy access to raw materials and widely available technical information in the Internet causes that chemical terrorism is serious threat to security of societies.

3.1 Chemical warfare agents

Chemical warfare agents are a wide group of chemical substances from various classes of compounds, possessing different characteristics and causing different effects on the human body. However, they all have a common denominator - they were specially made to cause injury or kill. Chemical warfare agents (in brief CWA) are classified according to effects on the human body. Descriptions of chemical warfare agents with symptoms of exposure are listed in below table.

Table 2 - Chemical warfare agents

Agent	Appearance	Time of action	Main way of exposure	Symptoms
Nerve agents	Colourless to yellow liquids. Fruity or amine scent	Depending on dispersion – from seconds/minutes (aerosols) to minutes/hours (liquids)	Respiratory tract, skin	Miosis, headache, eye pain, dimness of vision, vomiting, tightness in chest, cough, diarrhoea, convulsions
Blistering agents	Colourless to yellow/amber or blue/black liquids. Garlic, mustard, onion or fruity scent	Minutes to hours	Skin, eyes, inhalation, ingestion tract,	Skin burns, blisters
Blood agents	Highly volatile liquids. Characteristic smell of bitter almonds	Seconds to minutes	Respiratory tract	Hyperventilation, weakness, sense of warmth, pale-red skin colour
Chocking agents	Colourless or yellowish gases. From odourless to irritating, chlorine or moulding hay scent.	Seconds to minutes	Respiratory tract	Coughing, vomiting, irritation, shortness of breath
Riot agents	Colourless, white, yellow crystalline solids	Seconds to minutes	Respiratory tract, eyes	Burning sensation of eyes, nose, throat, tears, salivation, coughing
Psychomimetic agents	Colourless, odourless, tasteless solids	Minutes to hours	Respiratory or gastrointestinal tract	Dizziness, weakness, blurred vision, changed behaviour

More detailed information about chemical warfare agents could be found in Appendix C.

3.2 3.2 Toxic industrial chemicals

Toxic industrial chemicals (TIC), sometimes named toxic industrial materials (TIM), are chemicals that are widely manufactured, stored, transported and used. These chemicals can be in gaseous, liquid or solid state. They also represent different hazards, according to previously mentioned classifications of dangerous chemicals in transport (ADR) and in storage (CLP).

Due to the large quantity of different chemicals and their different qualities, it was hard to tell which one is most or least dangerous to health. To keep toxic industrial chemicals in order, International Task Force-25 introduced in 1996 a rank called Hazard Index (HI). It consists of four factors:

- Toxicity
- State of matter
- Distribution
- Producers

Each factor is assigned with a number between 1 and 5. The maximum value of the hazard index is 625. Hazard Index divide toxic industrial chemicals into three classes:

- **High hazard** - hazard index 81 and greater - widely produced, stored or transported chemicals which have high toxicity and are widely produced
- **Medium hazard** - hazard index between 36 and 80 - indicates toxic industrial chemicals which may rank high in some categories but lower in others in example number or producers, physical state or toxicity
- **Low hazard** - hazard index below 36 - indicates that this chemical is not likely to be a hazard unless specific operational factors indicate otherwise.

Examples of toxic industrial chemicals divided into classes are shown in the below table.

Table 3 - List of toxic industrial chemicals by hazard index

HIGH	MEDIUM	LOW
Ammonia	Acetone cyanohydrin	Allyl isothiocyanate
Arsine	Acrolein	Arsenic trichloride
Boron trichloride	Acrylonitrile	Bromine
Boron trifluoride	Allyl alcohol	Bromine chloride
Carbon disulphide	Allyl amine	Bromine pentafluoride
Chlorine	Allyl chlorocarbonate	Bromine trifluoride
Diborane	Boron tribromide	Carbonyl fluoride
Ethylene oxide	Carbon monoxide	Chlorine pentafluoride
Fluorine	Carbonyl sulphide	Chlorine trifluoride
Formaldehyde	Chloroacetone	Chloroacetaldehyde

Hydrogen bromide	Chloroacetonitrile	Chloroacetyl chloride
Hydrogen chloride	Chlorosulfonic acid	Cyanogen
Hydrogen cyanide	Crotonaldehyde	Diphenylmethane-4'-diisocyanate
Hydrogen fluoride	Diketene	
Hydrogen sulphide	1,2-dimethyl hydrazine	Ethyl chloroformate
Fuming nitric acid	Dimethyl sulphate	Ethyl chlorothioformate
Phosgene	Ethylene dibromide	Ethylene imine
Phosphorus trichloride	Hydrogen selenide	Ethyl phosphonothio dichloride
Sulphur dioxide	Iron pentacarbonyl	Ethyl phosphonous dichloride
Sulphuric acid	Methanesulfonyl chloride	
Tungsten hexafluoride	Methyl bromide	

Toxic industrial chemicals could be used in the CBRN incident. They are much easier to obtain and to deliver onto place of attack. Toxic industrial chemicals, with a little knowledge obtained via internet, could be weaponised to kill or injure people. Due to the great number of different chemicals, with different qualities it is hard to describe them all. The best way to find information about what to do after the release of chosen toxic industrial chemical are Material Safety Data Sheets and Emergency Response Guidebook. Both of these information sources could be found on-line for free. To show what kind of threat they represent, there are short description of chosen chemicals. Description on chosen toxic industrial chemicals could be found on Appendix C.

4. Biological threats

Biological materials as a class of CBRN constitute materials including pathogenic bacteria, fungi, viruses, and biological toxins. These materials may be the cause of a fatal or chronic illness, epidemics as well as collective panic and a social order breakdown. From its' definition a bioterrorism is "the deliberate release of viruses, bacteria or other biological agents used to cause illness or death in people, and also in animals or plants". Potential sites for attack with biological agents are places of mass human migration such as churches and temples where people gather to pray together. A terrorist attack using biological agents, besides direct human losses, can also cause enormous economic losses. Dangerous biological agents are very effective and may cause mass casualties because of:

- high morbidity and highly lethal potential,
- very low LD_{50} parameters, which make them highly toxic in small amounts,
- the ease of mass production and storage until delivery, without loss of pathogenic/toxic potential,
- relatively cheap cost and ease of setting up a laboratory in which bio-agents can be produced,
- availability of the equipment and reagents for producing bio-agents on the civilian market (even from Internet shops),
- the lack of supervision of relevant security services,
- compact size of a homemade laboratory, which can have the dimensions of a cargo container, or caravan,
- possibility of incorporation into the daily activity of medical or scientific laboratories, as well as within the infrastructure of the cosmetic and pharmaceutical industries,
- exceptionally low costs of production in comparison with other weapons,
- ease of hide,
- the lack of colour or/and odour,
- difficulties in finding the source of the attack,
- high time of action,
- relatively high stability after dissemination for periods long enough to infect humans.
- self-propagating mechanism since it can be transmitted over long distances by wind, contaminated people, animals or objects,
- difficulties in identifying contamination zones.

4.1 Biological agents as a weapon

Biological agents can be deployed in several forms, with the choice primarily determined by the technological capabilities of terrorists. These forms include:

- An aerosol causing air contamination, which is the most dangerous and most realistic outcome for bioterrorist attacks. Inhalation is associated with the greatest risk of widespread dispersion of biological material and imitation of infection.
- The contamination of food and water supplies. This type of attack is easier to carry out than aerosol dispersion since it doesn't require heavy equipment. Mass poisoning can be carried out with pathogenic microorganisms that naturally spread through the digestive tract, or pure biological toxins.
- Contaminated fleas, ticks, and mosquitoes to spread infectious pathogens. Recent reports from European intelligent services suggest that terrorists may be deliberately infecting themselves with bio-agents to spread the disease. This may also include suicide bombing, which not only kills people through a mechanical impact, but also serves to transmit infectious disease through the blast.
- The possibility of food crops being targeted, causing epidemics of serious infectious diseases in farmland.

The most popular form of bioterrorism in modern history is the use of envelopes containing powdered pathogens, addressed, and sent through the mail to the intended victims. For example, in the USA in the autumn of 2001, a series of letters containing anthrax spores were sent by mail to various senators, journalists and media offices. As a result, 22 people were seriously harmed, five of whom later died.

The range of biological agents that can be used in bioterrorism is large and includes pathogens causing serious and fatal infections in a few weeks or even days, and biological toxins, that typically attack both humans and animals. The nervous system is mainly affected but depending on the type of toxin, different side effects can occur. Some types of toxins cause death within a few hours or 72 hours while others may result only in poisoning symptoms.

The Centre for Disease Control and Prevention (CDC) maintains a list of potential critical biological agents, including those usable in bio-weapons. These are classified into three categories (A, B and C), based on their ease of transmission, morbidity, mortality rates, as well as a likelihood of actually being used.

4.2 Category A

Category A includes the highest-priority pathogens, which pose a risk to national security. These have the following features:

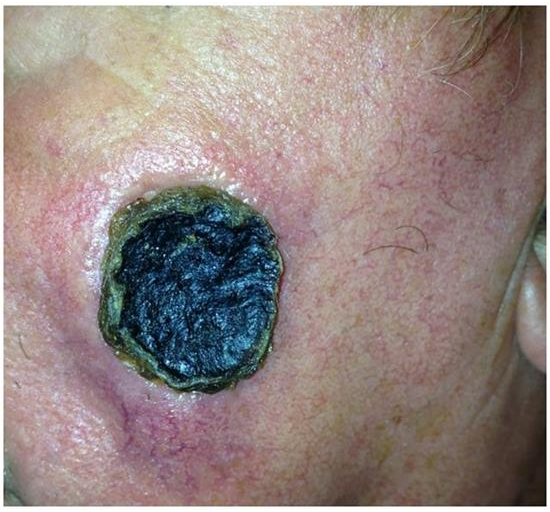
- They can be easily disseminated or transmitted person to-person, causing secondary and tertiary cases;
- They cause high mortality and a potential major public health impact, including an impact on healthcare facilities;
- Their use could cause public panic and social disruption;
- They require special action to counteract.

The CDC has classified 6 biological agents as Category A. They are listed below.

4.2.1 Bacillus anthracis

Bacillus anthracis is a gram-positive, rod-shaped bacterium, and one of the most likely agents to be used in a bioterrorist attack. This pathogen is responsible for a serious infection called anthrax. Anthrax most commonly occurs in wild and domestic herbivores, but it can also occur in humans when they are exposed to infected animals, tissue from infected animals, or high concentrations of anthrax spores. Dormant spores are highly resistant to adverse environmental conditions including heat, ultraviolet and ionizing radiation, high pressure, and chemical agents. This means that the spores can survive for long periods of time in contaminated soils. It has been established that spores are able to survive in soil for up to 40 years. Anthrax takes three different forms: cutaneous anthrax, which penetrate through damaged skin; gastrointestinal anthrax, and pulmonary (inhalation) anthrax. The cutaneous form of anthrax is the most common naturally-occurring form (95% of all cases), and the most pronounced. After infection via abrasions, cuts or insect bites, a small pimple will develop within two to three days. Initially, a 1–3mm bubble appears at the entry site, and after 2 days this becomes a black scab, known as “the black pimple”. The surrounding tissues form a hard, painless ulcer. In the next 2 weeks, the pimple dries and falls off, often without leaving a permanent scar. Although treatment with antibiotics does not make the scab fall off any faster, it is important in reducing the likelihood of fever, chills, lymph node damage and sepsis. Without antibiotics, mortality is around 20%, but with treatment death is rare. General symptoms after inhalation include fever, coughing and chest pains about 12 hours after infection. Later symptoms include a shortness of breath, swelling of the neck area and first signs of sepsis. Next comes necrosis of the lymph nodes, pulmonary edema. In the absence of treatment, mortality is 97% and death usually occurs 3 days after the onset of symptoms. Gastrointestinal anthrax is an extremely rare form of the disease and has a very high mortality rate (about 50%). This form of anthrax often presents after an incubation period of 1–7 days following ingestion of B. anthracis in contaminated food or drink. The gastrointestinal form is manifested by severe abdominal pain, fever, nausea, vomiting, and bloody diarrhea. If treatment isn’t introduced in time, toxemia and shock develop, followed by death. The Anthrax Vaccine is highly effective at preventing spread of Anthrax. Antibiotics can effectively treat Anthrax, if administered in time, that is 12h from exposure.

Figure 8 - Anthrax-like cutaneous lesion on left check of 70-year-old male Florida resident.

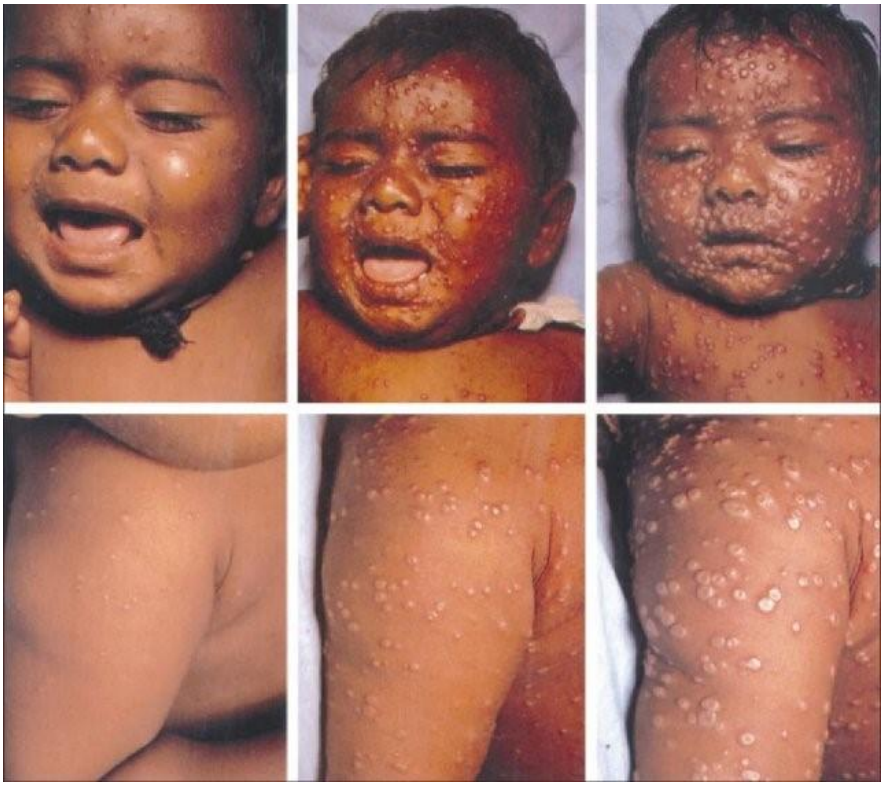


Source: Marston CK, Ibrahim H, Lee P, Churchwell G, Gumke M, Stanek D, Gee JE, Boyer AE, Gallegos-Candela M, Barr JR, Li H, Boulay D, Cronin L, Quinn CP, Hoffmaster AR. Anthrax Toxin-Expressing Bacillus cereus Isolated from an Anthrax-Like Eschar. PLoS One. 2016 Jun 3;1: e0156987.

4.2.2 Poxvirus variolae, smallpox Virus

Smallpox is a serious, contagious, and sometimes fatal infectious disease. A characteristic feature of this virus is its strict specificity to humans. The name smallpox is derived from the Latin word for "spotted" and refers to the raised bumps that appear on the face and body of an infected person. The virus can be transmitted in several ways, including a direct contact from person to person. The virus can also be transmitted through the air by droplet route when an infected person coughs, sneezes or speaks. In rare instances the virus can be spread through the ventilation systems. The first symptoms of smallpox usually appear 10 to 14 days after infection. Smallpox generally begins with fever, headaches, body aches, and weakness on day 1. Then in days 2–3 small, round pox (blisters) appear and spread on the face, arms, legs, and inside the mouth. On day 12 the blisters crust over; stomach pain and confusion can also occur. By week 3–4 the blisters turn into scabs and fall off, leaving pitted scars on the skin. There is no specific treatment for smallpox disease, and the only prevention is vaccination.

Figure 9 - Typical case of smallpox infection in a child (Day 3, Day 5, Day 7)



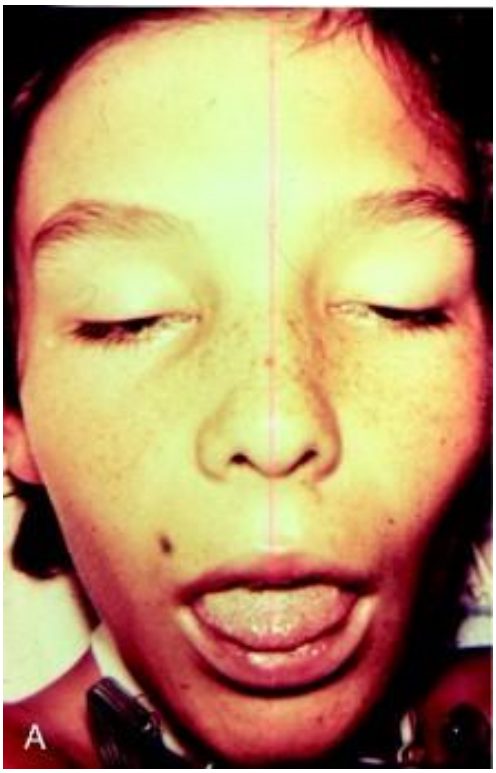
Source: Henderson DA, Inglesby TV, Bartlett JG, Ascher MS, Eitzen E, Jahrling PB, Hauer J, Layton M, McDade J, Osterholm MT, O'Toole T, Parker G, Perl T, Russell PK, Tonat K. Smallpox as a biological weapon: medical and public health management. Working Group on Civilian Biodefense. JAMA. 1999 Jun 9;281: 2127-2137.

4.2.3 Clostridium botulinum toxin

Botulinum toxin, also called “miracle poison”, is one of the most poisonous biological substances in the world. It is a neurotoxin produced by the Clostridium botulinum bacterium, which is an anaerobic organism commonly found in plants, soil, and water. Spores are extremely resistant to environmental factors, such as heat, radiation, chemical substances, and UV light. For these reasons, Clostridium spores can remain dormant for 30 years or more. The action of botulinum toxin is to block the

neurotransmission between nerves and muscles which results in respiratory and musculoskeletal paralysis. The most frequent source is home-canned foods, prepared in an unsafe manner. Wound botulism occurs when C. botulinum spores germinate within wounds. Infant botulism occurs when C. botulinum spores germinate and produce toxin in the gastrointestinal tract of infants. Symptoms of botulism might include: difficulty swallowing, muscle weakness, double vision, drooping eyelids, blurry vision, slurred speech, difficulty breathing, difficulty moving the eyes. Possible signs and symptoms in foodborne botulism might also include: vomiting, nausea, stomach pain, diarrhea. Signs and symptoms in an infant might include: constipation, poor feeding, drooping eyelids, pupils that are slow to react to light, face showing less expression than usual, weak cry that sounds different than usual, difficulty breathing. People with botulism might not have all of these symptoms at the same time. If untreated, the disease may progress and symptoms may worsen to cause full paralysis of some muscles, including those used in breathing and those in the arms, legs, and trunk (part of the body from the neck to the pelvis area). In foodborne botulism, symptoms generally begin 18 to 36 hours after eating a contaminated food.

Figure 10 - Portrayal of typical symptoms of mild botulism



Source: Johnson, E. A., & Montecucco, C. (2008). Chapter 11 Botulism. Neuromuscular Junction Disorders, 333–368.

4.2.4 Yersinia pestis

Yersinia pestis is a Gram-negative bacterium which is responsible for the bubonic plague (or “the plague”). This bacterium does not produce spores but has very high vitality. It can survive up to 6 months within the bodies of dead animals, in water less than a month, and for a few days in dry conditions. Y. pestis is maintained in nature as an infection in rodents and their fleas in large areas of Asia, Africa and the Americas. Transmission to humans occurs through contact with fleas and respiratory droplets from animals or infected humans. In naturally occurring plagues, the bite of an infected flea leads to the introduction of bacteria into a patient’s skin. The

Bubonic Plague has killed an estimated 200 million humans throughout history and is endemic in many areas of the world. There are three clinical forms of this disease: classical bubonic plague, primary septicemic plague, and pneumonic plague, determined largely by the way the pathogen enters the body. When infection occur through the subcutaneous route the main focus of the disease are the lymph nodes, the armpit and the neck. Patients typically develop the first symptoms of bubonic plague 2 to 8 days after receiving an infected flea bite. These include fever, chills, weakness and headache, followed by the development of an acutely swollen lymph node, or “bubo”. Bubos are painful, non-fluctuant, and warm, with surrounding oedema. The necrotic process causes occasional extrapulmonary outflow from the lymph nodes, which is often associated with an improvement in the patient’s condition. In addition, thromboembolic symptoms occur very often in the skin, leading to necrosis of the fingers and feet. In the severe form of the disease, infection of the lymph nodes can then occur, with the development of secondary sepsis and worsening of the general symptoms. Untreated bubonic plague is fatal after 2–4 days of illness and has a mortality rate more than 50%. When antibiotic treatment is applied, mortality drops. Septicemic plague is an infection of the blood, most commonly spread by bites from infected fleas. Pathogens are introduced to and proliferate in the body, without producing a bubo. Patients become feverish and actually die from bacteria in the blood, but without detectable lymphadenitis. The third form, pneumonic plague, occurs when Y. pestis infects the lungs. There are two paths of infection: the primary, which is inhalation of aerosolised plague bacteria, and secondary, in which septicemic plague spreads into the lung tissue from the blood. Pneumonic plague can spread from person to person through the air. Transmission occurs when someone breathes in aerosolized bacteria, which could happen during a bioterrorism incident. The most obvious symptom of pneumonic plague is coughing, often accompanied by haemoptysis (coughing with blood). With pneumonic plague, the first signs of illness are fever, headache, weakness and rapidly-developing pneumonia, characterized by shortness of breath, chest pains. The pneumonia plague progresses for two to four days and can cause respiratory failure and shock. This is a very aggressive infection requiring early antibiotic treatment (within 24 hours from presentation of first symptoms, in order to reduce the risk of death). Without therapy, the mortality rate reaches almost 100%.

Figure 11 - This patient presented with symptoms of plague that included gangrene of the hand causing necrosis of the fingers

Source: Stefan Riedel (2005) Plague: From Natural Disease to Bioterrorism, Baylor University Medical Center Proceedings, 18:2, 116-124.

4.2.5 Francisella tularensis

Tularemia also known as "rabbit fever" is an infectious disease caused by the bacterium Francisella tularensis contracted through contact with fur, inhalation, contaminated water uptake or insect bites.

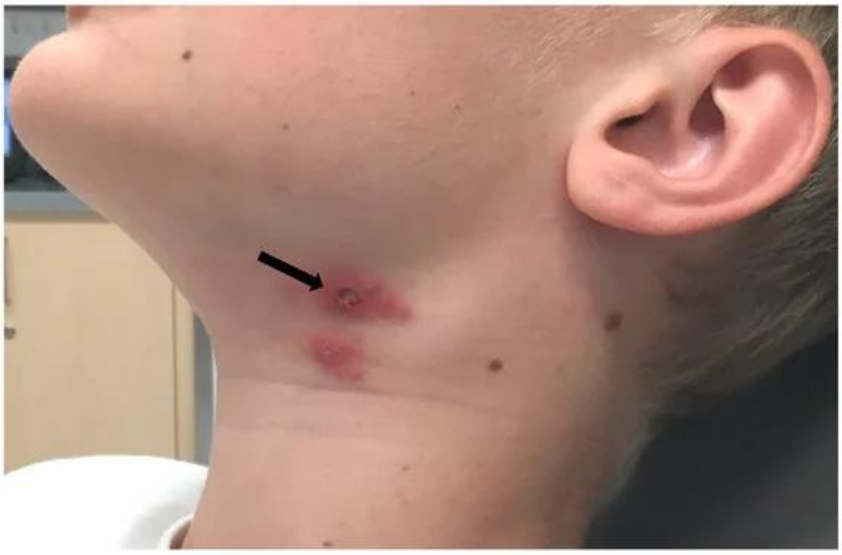
Tularemia is not spread directly from person to person. This pathogen is a gram-negative, rod-shaped coccobacillus. It is highly infectious pathogen, however, in comparison to other potential bio-agents it is very sensitive to chemicals and high temperatures. Tularemia has a very low fatality rate if treated, but it can severely incapacitate those infected.

The bacteria that cause tularemia occur widely in nature and can be isolated and grown in quantity in a laboratory, although manufacturing an effective aerosol weapon to deploy them would require considerable sophistication. There are several types of tularemia. The most common is transmitted via skin contact or bites by infected ticks, and this is ulceroglandular tularemia. However, if F. tularensis is used as a weapon, the bacteria would likely be made airborne for exposure by inhalation. People who inhale it as an infectious aerosol would generally experience severe respiratory illness, including life-threatening pneumonia and systemic infection. WHO simulations have shown that bioterrorism using F. tularensis would be very effective.

The signs and symptoms of tularemia vary depending on how the bacteria enter the body. Illness ranges from mild to life-threatening. All forms are accompanied by high fever. Main forms of this disease are listed below:

- **Ulceroglandular** This is the most common form of tularemia and usually occurs following a tick or deer fly bite or after handing of an infected animal. A skin ulcer appears at the site where the bacteria entered the body. The ulcer is accompanied by swelling of regional lymph glands, usually in the armpit or groin.
- **Glandular** Similar to ulceroglandular tularemia but without an ulcer. Also generally acquired through the bite of an infected tick or deer fly or from handling sick or dead animals.
- **Oculoglandular** This form occurs when the bacteria enter through the eye. This can occur when a person is butchering an infected animal and touches his or her eyes. Symptoms include irritation and inflammation of the eye and swelling of lymph glands in front of the ear.
- **Pneumonic** This is the most serious form of tularemia. This form results from breathing dusts or aerosols containing the organism. It can also occur when other forms of tularemia (e.g. ulceroglandular) are left untreated and the bacteria spread through the bloodstream to the lungs. The pneumonic form is the most potentially lethal form of tularemia. Besides the general symptoms, patients also experience chest pain and can have trouble breathing – and even sometimes stop breathing. The mortality rate of this form is about 30%.
- **Typhoidal** This form is characterized by any combination of the general symptoms (without the localizing symptoms of other syndromes)

Figure 12 - Clinical presentation of ulceroglandular tularemia after tick bite. The ulceration on the site of the tick bite is indicated by an arrow. Lymphadenopathy visible right from the inoculation site.



Source: Markowicz M, Schötta AM, Penatzer F, Matscheko C, Stanek G, Stockinger H, Riedler J. Isolation of Francisella tularensis from Skin Ulcer after a Tick Bite, Austria, 2020. Microorganisms. 2021 Jun 29;9(7):1407.

For detailed microscopic images of specific pathogens please see Appendix B.

4.2.6 Viral Hemorrhagic Fevers

Viral hemorrhagic fevers (VHFs) are a group of illnesses that are caused by several distinct families of viruses: Arenavirus, Filoviridae, Bunyaviridae and Flavivirus. Some of these cause relatively mild illnesses, while others can cause severe, life-threatening disease. Examples include Lassa fever, Marburg virus, Ebola virus, Bolivian haemorrhagic fever, Korean hemorrhagic fever, and Dengue hemorrhagic fever.

4.3 Category B

Category B agents are moderately easy to disseminate and have low mortality rates. They include:

- Brucellosis (Brucellaspecies);
- Food safety threats (for example, Salmonella species, E. coli O157:H7,
- Shigella, Staphylococcus aureus);
- Glanders (Burkholderia mallei);
- Melioidosis (Burkholderia pseudomallei);
- Psittacosis (Chlamydia psittaci);
- Q fever (Coxiella burnetii);
- Typhus (Rickettsia prowazekii);

- Viral encephalitis (alphaviruses, for example, Venezuelan equine encephalitis, eastern equine encephalitis, western equine encephalitis);
- Water supply threats (for example, Vibrio cholerae, Cryptosporidium parvum).

4.4 Category C

Category C of the highest-priority bio-agents includes emerging pathogens that could be engineered for mass dissemination. These agents are readily available, easy to produce and disseminate, and have potentially high morbidity and mortality rates, as well as a major impact on public health. The main bioagents in this category include:

- Nipahvirus;
- Hantavirus;
- SARS;
- H1N1 (a strain of influenza);
- HIV/AIDS;
- Multi-drug-resistant tuberculosis;
- Tick-borne haemorrhagic fever viruses;
- Tick-borne encephalitis virus;
- Yellow fever virus.

4.5 Other biological agents

There are also potent biological toxins that are derived from plant sources and fungi as described below.

Ricin is one of the most potent poisons in the plant kingdom derived from castor beans. Because of the wide availability of its source plants, ease of production, stability and lethal potency, ricin toxin is considered to be a bioterrorism threat. The estimated LD50 parameter of pure ricin is around 10–20 μg/kg of body weight. Oral exposure to ricin is far less toxic as some of the poison is inactivated in the stomach. Symptoms of ricin poisoning, which can take several hours to present, vary depending on how the toxin is absorbed by the organism.

- Inhalation – within a few hours of inhaling significant amounts of ricin, the likely symptoms would be respiratory distress (difficulty breathing), fever, coughing, nausea, and tightness in the chest. Heavy sweating can follow as well as build-up of fluid in the lungs (pulmonary oedema). Finally, low blood pressure and respiratory failure can occur, leading to death.
- Ingestion – the first symptoms are vomiting and diarrhea that can become bloody. Severe dehydration may occur, followed by low blood pressure. Other signs or symptoms can include seizures, and blood in the urine. Within several days, the patient's liver, spleen, and kidneys might stop working leading to death. Initial symptoms of poisoning by inhalation can occur as early as 4–8 hours and as late as 24 hours from exposure. Following ingestion of ricin, initial symptoms typically occur in less than 10 hours. Death from ricin poisoning can take place within 36 to 72 hours from exposure.

Abrin is a plant-based toxalbumin (like ricin) obtained from the seeds of Abrus precatorius (the jequirity bean, or 'rosary pea'). Abrin is similar in its molecular structure and mechanism of action to ricin, however, abrin is more toxic than ricin, with an estimated human fatal dose of just 0.1–1 μg/kg. The symptoms of abrin poisoning are the same as in ricin poisoning.

T-2 mycotoxin is a toxic secondary metabolite produced by the Fusarium species fungi. Potentially hazardous concentrations of this toxin occur naturally in mouldy cereal grains and other agricultural products that are inadequately stored. It can also be produced commercially and used as a bio-terror weapon. It is an extremely stable compound that has a strong resistance to heat and UV light inactivation. The substance is quite insoluble in water but highly soluble in ethanol, methanol, and propylene glycol. T-2 mycotoxin is a potent active dermal irritant and is the only potential bio-agent that can be absorbed through intact skin, causing systemic toxicity. The symptoms can present within seconds of exposure. T-2 can be also delivered via food or water sources, as well as droplets, aerosols, or various other dispersal systems. T-2 mycotoxin has multiple mechanisms of action via inhibition of protein synthesis and cell proliferation, which causes acute or chronic intoxication of humans and animals. Toxic effects include myelotoxicity, haematotoxicity, and necrotic lesions at contact sites. Exposure causes skin pain, pruritus (itchiness), redness, vesicles (fluid-filled blisters), necrosis (tissue death) and sloughing of the epidermis. Severe intoxication results in weakness, ataxia (lack of muscle control), collapse, shock, and death. These properties make T-2 mycotoxin a potentially viable biological agent for use in terrorist attacks.

5. Radiological and Nuclear threats

The development of civilisation, the search for new resources and markets has changed the image of the world through two wars which have claimed the lives of tens of millions of people. Incessant technological progress has for centuries contributed to the development of tools for destruction and killing - despite the efforts of the international community to ensure peace and stability, conflicts constantly break out. During the Second World War, developments in physics proved that mankind was able to harness the power of the atomic fission reaction, wielding a lethal weapon of previously unimaginable destructive force, as well as a practically inexhaustible source of energy. However, the dangers of ionising radiation are not new and have not arisen solely as a result of human activity.

[If you would like to read more about this topic, you can find more information in the → ***Appendix RN****]*

5.1 Types of ionising radiation

There are natural radioactive elements in nature, present in soil, rocks, air and water, contained in minerals, assimilated by various plant and animal species, found as components of commonly used construction materials, or lighter ones - synthesised in the atmosphere as a result of the reaction of atmospheric constituents with cosmic radiation. These elements have existed since the creation of our planet and the radioactivity originating from them has accompanied organisms since the origin of life on Earth. Cosmic radiation, which is important, is also included in the set of natural radiation sources. The discovery of natural radioactivity by Henri Bequerel in 1896 and the discovery of polonium and radium by Maria Skłodowska-Curie and Peter Curie in 1898 brought mankind closer to understanding the nature of the surrounding world made up of atoms. In the world around us there is a whole range of elements. The fact that an atom belongs to a given element is determined by the composition of positively charged protons and electrically neutral neutrons which make up the atomic nucleus of this atom. At present 264 stable atomic nuclei are known, i.e. such nuclei which do not disintegrate (the heaviest such nucleus is bismuth with the atomic number, i.e. the number of protons in the atomic nucleus, Z = 83 and the mass number, i.e. the number of protons and neutrons in the atomic nucleus, A = 209) and nearly 2450 unstable nuclei, which do not disintegrate (called radioactive). Radioactivity is the ability of atomic nuclei to undergo radioactive decay, which is most often associated with the emission of particles and electromagnetic radiation (so-called gamma radiation).

The radiation emitted during the decay of an atomic nucleus is ionising radiation. Ionising radiation means all types of radiation which cause ionisation of a material medium, i.e. removal of at least one electron from an atom or molecule or breaking it out of a crystal structure. Electromagnetic ionising radiation is defined as radiation whose photons have an energy greater than that of photons of visible light. Nuclear ionising radiation mainly includes: alpha radiation (α), beta radiation (β-, β+), gamma radiation (γ) and indirectly neutron radiation. Substances that emit ionising radiation are called radioactive.

5.2 Health effects of ionising radiation

When ionizing radiation traverses through matter, it loses energy progressively through the various interactions along the length of its path. For a particular absorber, the rate of the energy loss depends on the energy, the type of radiation and the density of the material. When a high-energy particle hits a human cell, it produces a narrow ‘track' as it proceeds through the material. Radiation absorbed by cells has the potential to affect a variety of critical cellular targets (nucleic acids, proteins, lipids, carbohydrates), causing ionisation and excitation. The chemical and biological effects of ionizing radiation on living matter are the result of the deposition of radiation energy directly into the target

macromolecule, which is a component of the cell, and of the indirect action resulting from the excitation and decay of water molecules - radiolysis of water. Together, the direct and indirect effects of radiation initiate a series of biochemical and molecular signalling events that can repair the damage or culminate in permanent physiological changes or cell death.

The molecular mechanisms of ionizing radiation-induced cellular injury depend on many factors that primarily include radiation dosage as well as the cell type, cycle phase, and its transformed status. Taking into account the chemical processes occurring during irradiation, the extent of the cellular damage that results from the specific type of ionizing radiation is similar in terms of the dose and the amount of DNA. However, the final ionizing radiation effect is determined by the post-radiation processes such as DNA damage repair and the proliferative activity of the cell.

The biological effect of radiation depends on the type of tissue (organ) which has been irradiated. To quantify the total damage from the exposure of several tissues (organs), the concept of the effective dose (E) was introduced.

Throughout their lifetime, humans are exposed to low doses of ionizing radiation from natural sources. The man-made sources which also contribute to ionizing radiation exposure include industrial (nuclear power plant workers), and medical (radiotherapy, diagnostic X-rays) sources. However, radiation accidents and incidents (nuclear weapon explosions, terrorist acts with radioactive materials) have led to much higher exposure doses. Despite the many practical applications of ionizing radiation, exposure to high radiation doses has fatal consequences. Radiation harm can occur from external irradiation (outside the body), external or internal contamination with radioactive materials (the latter include inhalation, digestion or absorption through the skin), as well as from combinations of all of these exposure types. Injury from a nuclear detonation depends on the location of the victim relative to the hypocentre and the resulting exposure to heat, bomb blast, and radiation. Heat and light cause thermal injury, such as skin and retinal burns and blindness. The blast wave can result in fractures, wounds, rupture of internal organs and pulmonary haemorrhage and oedema. Acute overexposure to ionizing radiation results in Acute Radiation Syndrome (ARS). The effects of ionizing radiation on the human population are commonly divided into somatic and genetic. Somatic effects include harm that individuals exposed to ionizing radiation suffer during their lifetime, such as the increased risk of ionizing radiation-induced cancers (carcinogenesis), opacification of the eye lens, infertility, and shortened lifespan. Genetic effects (also called hereditary effects) include ionizing radiation-induced changes in genomic DNA of the exposed individual's (chromosome aberrations, mutations), which can contribute to the birth of defective descendants. Depending on the dose and the stage of development at the time of exposure, the main effects of radiation on a foetus include: foetal or neonatal death, malformations, growth retardation, congenital defects and cancer induction. The biological effects of ionizing radiation on the human body depend on the nature and energy of the radiation, the time and mode of interaction and radiosensitivity of the exposed cells.

Radiosensitivity is a broad term which can be applied to cells, tissues, organs and individuals. Cellular radiosensitivity defines the degree of response of the cells to ionizing radiation. The response to ionizing radiation can vary by cell type. The vulnerability of tissue to radiation injury depends on the degree of differentiation of the cells in the tissue and their proliferative activity. Additionally, the radiation's effects can be modified by different factors, such as the type of radiation, dose, dose rate, dose fractionation, the mass of the irradiated tissue, tissue oxygenation, the organ irradiated, and the addition of radical scavengers. The individual radiosensitivity of various representatives of the population is relatively diverse. For the description of radiosensitivity of the given population the concept of lethal dose (LD) has been introduced. All variants of this dose assume a single irradiation in a short time (up to several hours) of the whole body and no medical assistance after irradiation. The most useful for comparison is the mean lethal dose. It can be measured by assays such as LD 50/30 which defines the radiation dose required to kill 50% of a given population within 30 days of exposure. Minimal lethal dose (LD) refers to the smallest radiation dose at which deaths can occur due to irradiation of a given

population, while the maximal lethal dose (LD) defines the minimal radiation dose that causes death of all individuals of max the irradiated population.

5.2.1 Deterministic and stochastic effects

The timescale involved between the breakage of the chemical bonds in the vital macromolecules and the biological effect may be hours to years, depending on the type of damage. The acute effects of ionizing radiation are known as so called deterministic effects (non-stochastic effects). They have a specific threshold dose. A deterministic effect (tissue reaction) is defined as a one that increases in severity with increasing dose, usually above a threshold dose, in affected individuals. For example, the total body irradiation at the dose greater than 5 Gy results in the bone marrow suppression, but this suppression is not observed for the smaller dose. A stochastic effect is defined as a one in which the probability of occurrence increases with an increasing dose but the severity in affected individuals does not depend on the dose. These are statistically measurable effects. There is no threshold dose for the effects that are truly stochastic, because these effects arise in single cells and it is assumed that there is always some small probability of the event occurring even at very small doses. Stochastic effects (usually chronic effects) are for example ionizing radiation-induced genetic mutations, chromosome aberrations, carcinogenesis.

Table 4 - Deterministic effects of radiation

Threshold value of radiation (Sv)	Deterministic effect
< 0.1	Temporary sterility
0.5	falling vision
< 1	nausea, vomiting (2-4 hours after exposure)
1 - 2	nausea, vomiting (1-2 hours after exposure)
2 - 8	Vomiting (in minutes), headache, fever, diarrhea, skin burns
> 8	Vomiting (in minutes), severe headache, fever, diarrhea, skin burns, loss of consciousness, permanent sterility

5.2.2 Whole body irradiation

The response of humans to a single dose of whole body irradiation (WBI) can be characterized by four overlapping syndromes: prodromal, haematological, gastrointestinal, and neurovascular syndrome, which are manifested following different doses, at different post-radiation time.

- The neurovascular syndrome occurs following large ionizing radiation doses (more than 20 Gy) and usually causes a rapid death (within hours to days) due to dysfunction of cardiovascular and nervous system.
- The gastrointestinal syndrome occurs following exposure to doses above 8-12 Gy and generally result in death (within a week) which is mainly caused by a severe damage of the gastrointestinal tract mucosa. A subsequent loss of the protective barrier results in infection, loss of electrolytes and the fluid volume imbalance. In human victims, intensive treatment with

antibiotics, replacement of fluids and electrolytes can prevent early death from this syndrome. However, these patients can die due to the injury of other organs.

- The haematopoietic syndrome in rodents occurs at doses in the range of 2-8 Gy, which is caused by a severe depletion of blood morphotic elements, such as red blood cells (RBCs), white blood cells (WBCs) and blood platelets (PL). Treatment of human victims may include bone marrow transplantation to prevent death, provided that the radiation dose was not too high. For human the mean lethal dose (LD_{50}) has been estimated at 4 Gy (4-7 Gy depending on the supportive care).

In relation to human, high instantaneous doses (more than 10 Gy) can occur accidentally (explosions of nuclear weapons, nuclear power plant accidents, handling unshielded radiation sources or radioactive waste). In opposite, doses of ionizing radiation below 0.15 Gy produce no noticeable symptoms or signs. This range includes, for example: lifetime radiation exposure from natural background radiation, the majority of nuclear diagnostic tests or nuclear power plant functioning. Increased radiation doses (0.15 to less than 0.5 Gy) result in subclinical responses, characterized by very few, if any, clinical or haematological symptoms. This level of ionizing radiation exposure produces no visible manifestations, with the probability of chromosomal breaks occurring. At radiation doses from 0.5 to 30 Gy or more, clinical responses do occur. Acute radiation syndrome (ARS) is seen in individuals following acute whole body irradiation with doses of 1 or more Gy.

Acute Radiation Syndrome (ARS), is defined as ‘an acute illness caused by irradiation of the entire body (or most of the body) by a high dose of penetrating ionizing radiation in a very short period of time (usually a matter of minutes)’. ARS is also known as radiation sickness, and can be seen after exposures to doses greater than 1 Gy. The degree of ARS may be classified by the absorbed dose and the time over which the energy from the radiation has been deposited in the tissues. The clinical phase of ARS can also be divided into four overlapping stages:

- a mild phase (0.5-1 Gy);
- the haematopoietic syndrome (1-8 Gy);
- the gastrointestinal syndrome (8-30 Gy);
- the central nervous system syndrome (>30 Gy).

Each syndrome can be divided into four stages: the initial, latent, and manifesting illness stages. The last of these is recovery or death. Depending on the dose absorbed, symptoms can appear at different times.

5.3 Radiation exposure

The purpose of radiological protection is to carry out activities aimed at reducing exposure of people and the environment to ionising radiation. Radiological protection is mainly based on two systems:

- licensing and surveillance of activities involving exposure to ionising radiation,
- limitation of exposure from ionising radiation.

In order to reduce the exposure of the population to ionising radiation, the following principles should be observed:

- avoid unnecessary and unneeded radiation sources,
- control the practice of exposure in such a way that the radiation doses received are as low as reasonably achievable taking into account health, but also economic and social aspects,
- plan activities involving radiation sources in such a way that the benefits obtained justify the necessary dose from ionising radiation received,
- comply with the provisions for so-called dose limits (threshold doses to be discussed later in this material).

In order to assess human exposure to ionising radiation, the concept of dose was introduced. However, there are different types of doses associated with exposure to ionising radiation. The reason for the existence of so many different types of dose is that, up to now, it has not been possible to precisely describe in quantitative terms all the effects accompanying the irradiation of tissues and organs of the human body. The most important physical quantities used for measuring the effects of ionising radiation which can be measured directly are:

- activity (A), measured in becquerel (Bq);
- exposure dose (X), measured in coulomb per kilogram (C/kg);
- absorbed dose (D), measured in Gray (Gy);
- dose equivalent dose (H_T) and
- effective dose (E), both measured in Siewert (Sv).

If irradiation of tissues and/or organs occurs with different types of ionising radiation or with radiation of different energies, then the total equivalent dose is determined from the sum of the respective radiation weighting factors multiplied by the corresponding tissue or organ absorbed dose averaged over the different types of radiation. The weighting factors are unmeasured quantities. The unit of equivalent dose is called sievert (Sv). The unit of equivalent dose formerly used was the rem. The equivalent dose takes into account only the weighting factors and the energy of the ionising radiation, but does not take into account the sensitivity of the tissues and organs being irradiated. For this reason, the effective dose, also called the effective dose, was introduced.

Exposure to ionising radiation when a radioactive isotope enters the body is calculated in a slightly different way. If one is dealing with an external source of radiation, there is only a risk while one is close enough to the source to be affected. The risk from the intake of a radioactive isotope into the body depends largely on its chemical, physical and biological properties, such as:

- the type and energy emitted by the isotope,
- its chemical form and solubility in water,
- the characteristics of the organ where the accumulation of the isotope takes place,
- the metabolic rate of the organism,
- the routes of introduction of the isotope into the body (inhalation, ingestion).

The equivalent committed dose $H_T(\tau)$ is the dose to an organ or tissue at time τ due to the entry into the body of a radioactive element. The unit of the equivalent committed dose is the sievert (Sv).

If the body is irradiated (external exposure) and internal contamination occurs, the effective dose is calculated by adding together the effective dose from the external ionising radiation source and the effective committed doses (in a given age group) due to the ingestion of a specific radionuclide via oral and/or inhalation, respectively, of a given activity.

In order to protect people, in particular exposed employees, but also the general public from the adverse effects of radiation, standards have been introduced on the basis of which threshold values of effective and equivalent doses have been defined, which, apart from certain exceptions defined by law (Atomic Law), must not be exceeded. These doses are called dose limits.

The dose limits include the sum of doses from external and internal exposure. However, they do not include exposure from natural radiation if this exposure is not increased by human activity, i.e. they do not include exposure from the following sources:

- radon ^{222}Rn present in residential buildings,
- radioactive isotopes contained in the human body,
- cosmic radiation at the earth's surface,
- radioactive elements in the earth's crust,
- individuals undergoing medical exposure,
- emergency exposure situations resulting from actions designed to: prevent a major health detriment; avoid significant irradiation of a large number of people; prevent a major disaster.

For persons in the general population the dose limit, expressed as an effective dose, shall be 1 mSv in a calendar year. For occupational workers with exposure to ionising radiation and schoolchildren, students and apprentices aged 18 and over, the dose limit shall be 20 mSv in a calendar year. In the case of workers, the dose may be exceeded up to 50 mSv in a year, if such exceeding is authorised by the certain authority responsible for radiation safety at the state level, or other authority competent to grant authorisation or accept a notification of the practice. The dose limit for school children, students and apprentices between the ages of 16 and 18 shall be 6 mSv. Pupils, students and apprentices under the age of 16 shall be subject to the dose limit for the general population.

According to the principles of the law, dose limits must not be exceeded. However, it is possible to deviate from this rule in an emergency exposure situation resulting from actions aimed at:

- prevention of a serious loss of health,
- avoidance of significant irradiation to a significant number of people,
- prevention of a major catastrophe.

In one of these cases, it is advisable to make every effort to ensure that the person taking part in such activities does not receive an effective dose exceeding 100 mSv. If, on the other hand, a volunteer takes part in saving human life, he or she may receive an effective dose exceeding 100 mSv, but care must be taken not to give an effective dose exceeding 500 mSv. Only individuals who are volunteers and who have been informed in advance of the health effects of exposure to ionising radiation may take part in such actions.

Based on data published in annual reports of relevant radiation safety authorities (e.g. for Poland - National Atomic Energy Agency), in 2020, the exposure to radiation from natural sources constitutes about 61.9% of the total exposure, with the remaining 38.1% from artificial sources. For example, a statistical Pole in 2020 received an average annual effective radiation dose estimated at 3.96 mSv. In the case of artificial radioactivity, practically the whole part of the effective dose corresponding to it comes from medical diagnostics. It must be remembered, however, that the doses from this radiation source indicated above are average and statistical. During a single examination the effective dose received is usually higher, e.g., during a single mammography examination - 0.02 mSv, X-ray examination - 1.2 mSv, chest X-ray - 0.11 mSv, spine X-ray and lung X-ray - from 3 to 4.3 mSv.

If we compare the above dose levels with the average effective dose of ionising radiation to the whole body received by people in Europe during the first year after the Chernobyl disaster, and if we take into account the dose received in a single whole-body computed tomography scan, one may conclude that the process of transporting and storing radioactive waste and improper handling of radioactive sources is more dangerous than an accident at nuclear facilities.

5.4 Ways to reduce exposure to radiation

In order to improve safety when working with ionising radiation sources, it is necessary to follow rules that are directly derived from the physical properties of ionising radiation. There are different rules for working with open radiation sources and those for sealed sources. In the case of devices emitting ionising radiation, or the use of sealed sources, only external radiation can be produced. In the case of working with open sources, you may additionally be subject to internal contamination.

To minimise exposure to ionising radiation in the case of external radiation sources, the following rules should be applied:

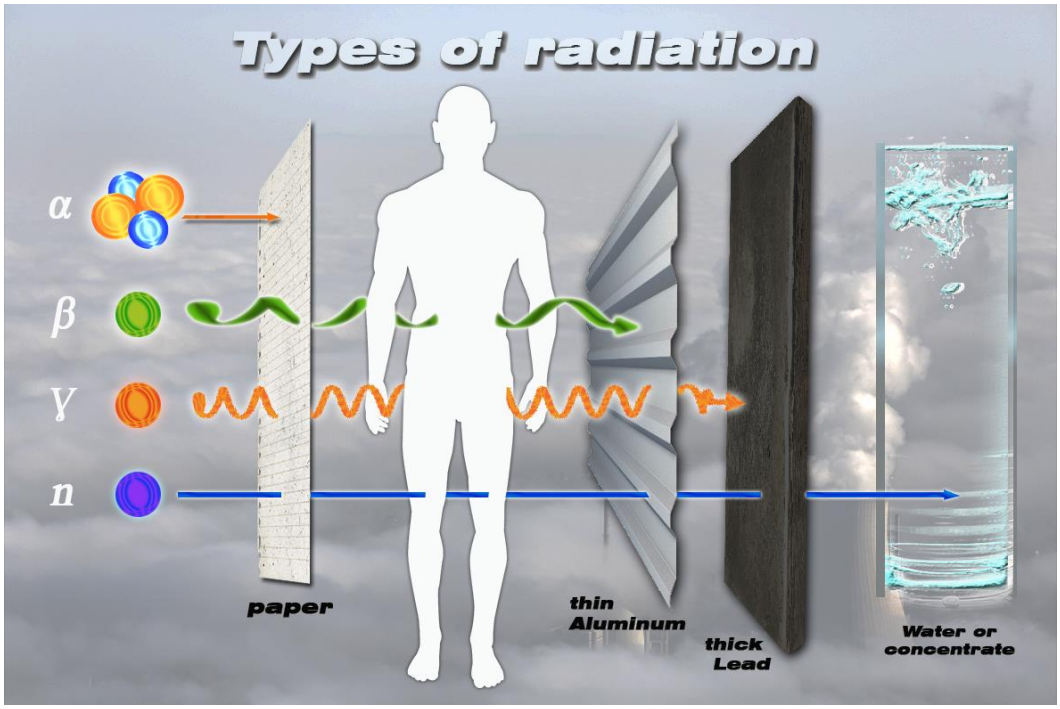
- Maintain an adequate distance from the radiation source, due to the fact that the value of the dose is inversely proportional to the square of the distance from the source, thus, for example, increasing the distance by four times, the dose that will be deposited in the body will decrease by sixteen times.
- Limit the time spent in the vicinity of the source, since the value of the dose depends directly proportional to time, and thus by reducing the time spent in the vicinity of the source, e.g. by a factor of three, the dose deposited in the body will also decrease by a factor of three.
- Use appropriate radiation shielding to limit exposure. In this case, the thickness and type of shielding material used depends on what type of ionising radiation you are dealing with and the energy of the radiation. Protection against alpha and beta radiation is easiest, due to the fact that alpha radiation is already absorbed by a sheet of paper or a few tens of centimetres of air. For complete absorption of beta particles with energies up to 10 MeV, a one centimetre layer of organic glass is sufficient. For higher energies of beta particles, suitably thicker shields should be used, which are made of materials containing elements of small atomic number. Gamma radiation is much more penetrating than alpha or beta radiation. Only thicker layers of heavy materials such as lead, concrete or depleted uranium protect against this type of radiation.
- In the event of contact with open radioactive sources, some of the substance may be unbound and leak into the environment, contaminating it. In addition, radioactive material may also enter the body by inhalation, ingestion or directly through the skin. The following rules must be observed to avoid internal and environmental contamination:

- All work with open sources of ionising radiation should be carried out in isotope laboratories or in another location that has been properly prepared and secured in advance under the supervision of a radiation protection officer.
- Work with sources should be carried out in accordance with work regulations and technological work instructions, and, in the case of isotopic apparatus, in accordance with the operating instructions.
- Work with sources may only be carried out by persons who have received prior training from the radiation protection officer and have been approved by the relevant doctor.
- Procedures for working with and handling radioactive sources must be strictly followed.
- All items used when working with radioactive sources should be properly labelled.
- Drinking, eating and smoking are prohibited in radiochemical laboratories.
- Dishes used for working with radionuclides and laboratory equipment must not be taken outside the radiochemistry laboratory and are not permitted in the regular laboratory.
- All radioactive substances should be stored in strictly segregated magazines meeting specific criteria.
- Any radioactive contamination should be disposed of as soon as possible in accordance with pre-established procedures.

The primary directive of the Council of the European Union in the context of radiation protection is the relevant regulations and standards recommended by the International Commission on Radiological Protection (ICRP). The ICRP principles are mainly concerned with the justification of the use of nuclear radiation, radiation protection, dose limitation and safety of radiation sources. The following are the three main principles proclaimed by the International Commission on Radiological Protection:

- Justification of activities involving exposure to ionising radiation - According to this principle, an exposure to ionising radiation is only acceptable if the scientific, economic or societal benefits will outweigh the possible damage to human health and the environment caused by the activity.
- ALARA (As Low As Reasonably Achievable) - According to this principle, work with ionising radiation sources should be so planned that the number of people exposed and their doses are as low as reasonably achievable, taking into account economic and social factors.
- Application of individual dose limits - Exposure of individuals to ionising radiation shall be limited through the application of dose limits, so-called dose constraints, except in the case of natural radiation and medical uses.

Figure 13 A&B - Ways to reduce exposure to radiation (infographics)



Source: own collaboration

5.5 Source of ionising radiation

The atomic nucleus in most cases remains in the so-called ground state, i.e. the state which has the lowest total energy. However, it can also exist in an excited state. Atomic nuclei of elements in the excited state (and also some atomic nuclei in the ground state) can undergo decay, which changes their composition, as a result of which, among others, an α particle (^{4}He nucleus), a β- (electron) or β+ (positron) particle, a proton or a neutron can be emitted from the nucleus, or it can undergo fission, leading to division into heavier fragments. Invariably, a prerequisite for any decay is that a certain amount of energy is released, which boils down to the condition that the mass of all decay products is less than that of the initial system. In order to determine the rate of spontaneous decay of atomic nuclei of a radioactive isotope (isotopes are different forms of atoms of a chemical element, differing in the number of neutrons in the nucleus), the concept of **half-life** has been introduced (denoted by $T_{1/2}$, is the time during which the number of unstable nuclei decreases by half.

High energy cosmic radiation reaching the Earth from the Sun and other parts of the Universe is one of the main sources of natural radiation. This radiation, called primary radiation, causes the creation of new particles and radiation called secondary radiation, as a result of collisions with the nuclei of the atoms of gases which make up the Earth's atmosphere. Primary radiation consists of high-energy protons (90%), alpha particles (7%), electrons (1%) and nuclei of heavy atoms (1%) with energies ranging from 108 - 1020 eV (electron-volts)

Natural ionising radiation is also due to the presence in the earth's crust of natural radioactive elements with very long half-lives of billions of years. These include such radioactive isotopes as e.g. ^{238}U, ^{235}U, ^{232}Th, ^{40}K. These isotopes are part of the so-called natural radioactive series:

- uranium-radium (^{238}U - ^{206}Pb),
- uranium-actinium (^{235}U - ^{207}Pb) and
- thorium (^{232}Th - ^{208}Pb).

In rocks, the most common radioactive isotopes include ^{40}K, ^{87}Rb and the decay products of the uranium-radium and thorium series. Of the so-called long-lived radioactive elements, ^{235}U, ^{238}U, ^{226}Ra, ^{228}Ra and ^{210}Pb make the greatest contribution to natural radioactivity.

In addition to the natural sources of radiation which have been present in our environment since the beginning of time, additional radioactive isotopes have found their way into the environment as a result of human activity. This is due to the use of radioisotopes and artificial sources of radiation e.g. in radio-medicine, science, industry, power generation (as a result of nuclear reactor accidents) and, above all, through military applications (nuclear test explosions). X-ray examinations (e.g. dental bone or chest radiographs) are primarily responsible for the radiation dose received by men from medical artificial radiation sources. The remainder is related to so-called nuclear medicine characterised by the use of radioactive isotopes for diagnostic purposes and therapy with X-ray emitting apparatus. Several tens of radioactive isotopes are currently used in nuclear medicine. These include: for imaging of pathogenic lesions: technetium ^{99m}Tc, chromium ^{51}Cr, phosphorus ^{33}P, indium ^{111}In, sodium ^{24}Na, carbon ^{11}C, or for treatment of specific pathogenic lesions, e.g. boron ^{10}B, cobalt ^{60}Co, strontium ^{89}Sr, gold ^{198}Au, palladium ^{103}Pd.

In science, radioactive isotopes are used, among others: in biological sciences (analysis of DNA sequences, determination of hormones and enzymes in blood samples, tracing biologically active substances in the body and their selective uptake by specific organs, analysis of uptake processes by plants of metals present in soil and their circulation in the food chain), geological sciences (e.g. application of methods: carbon, tritium, helium to determine the age of rocks and geological samples).

Radioactive isotopes are also used in industry, e.g. to measure the thickness of materials (cerium ^{144}Ce), to determine the mass of products (thallium ^{204}Tl), to detect defects in materials (iridium ^{192}Ir), to detect smoke (americium ^{241}Am), to power electric cells in satellites (plutonium ^{238}Pu), to sterilise medical products (cobalt ^{60}Co), to test the wear of friction parts in machines (iron ^{59}Fe), to determine the course of underground rivers (sodium ^{24}Na), to remove static electricity (polonium ^{210}Po).

Shortly after its discovery, the phenomenon of nuclear fission was used in nuclear energy and for military purposes. Albert Einstein formulated the special theory of relativity, one of whose most revolutionary conclusions was the equivalence of mass and energy. The phenomenon of forced fission of the atomic nucleus under the influence of neutrons observed in 1938 by Otto Hahn and Fritz Straßmann opened up the possibility of obtaining and using chain fission reactions on a macroscopic scale.

Nuclear energy uses so-called nuclear fuel which contains radioactive isotopes: most commonly enriched uranium (i.e. uranium with a higher than natural relative isotope content of ^{235}U, ranging from a few to 90%) and plutonium ^{239}Pu. These same radioactive isotopes are used in the manufacture of nuclear weapons, where the phenomenon of an uncontrolled chain reaction of fissile material is exploited. Before such tests were banned in all countries, more than 450 explosions were carried out, resulting in radioactive isotopes such as: ^{140}Be, ^{131}I, ^{141}Ce, ^{3}H, ^{103}Ru, ^{95}Zr, ^{91}Y, ^{89}Sr, thereby subjecting ourselves to additional ionising radiation from long-lived radioactive elements, i.e. ^{14}C, ^{90}Sr, ^{137}Cs, ^{239}Pu and ^{240}Pu.

5.6 Contemporary threats related to radioactivity

All useful activities in various fields of life which use radioactive sources or fissile materials, may pose a risk to people and the environment in the event of an uncontrolled release of isotopes or an uncontrolled fission reaction.

Nuclear power represented a breakthrough in the development of new energy sources. The first nuclear reactor was built in 1942 in Chicago by a team of physicists led by Enrico Fermi. Fermi's achievement was crucial and in a short time enabled the United States to develop the first atomic bombs, which were used in the attack on Hiroshima and Nagasaki on 6 and 9 August 1945. The effects of the first explosions of the new bombs surprised even their creators with their range of impact and power of destruction. However, the emergence of new, deadly weapons started an arms race between the world's major powers. In addition to military applications, scientists and politicians were quick to recognise the non-military benefits of harnessing nuclear energy. Concepts were developed to build nuclear power plants - industrial plants that generate electricity by using the energy from nuclear fission, the heat of which is used to produce steam. The first nuclear power plant was built in 1954 in Obninsk (USSR) based on the light-water reactor (RBMK) (such reactors were only designed and built in the former Soviet Union). In Great Britain, the first power reactor was built in 1956. A year later, the first prototype pressurised-water reactor (PWR) started operating in the USA.

The development of nuclear power in Western European countries and the USA was accompanied by the priority of safety over economic considerations. One exception to this rule was the construction of RBMK type nuclear reactors in power stations in the Soviet Union. Because of their additional use, which was to produce enriched plutonium for the construction of nuclear weapons, they were characterised by instability and the need to transfer responsibility for reactor safety to the operator.

Despite all radiological protection rules, procedures and safety regulations, there is always the possibility of an accident resulting in people and the environment being exposed to radiation which exceeds the radiation dose limits. Improper handling of radioactive sources, a fire in a radiology laboratory or a road accident during transport of radioactive materials are just some examples of such events. The main problem that arises in the early stages of such an incident is the identification of the hazard, its extent and level of danger. Unlike other hazards such as fires or floods, ionising radiation

cannot be seen by sight, smelled or tasted. It is necessary to use dedicated equipment - ionising radiation detectors. With their use, it is possible to determine what measures and methods should be applied to minimise the risk and remove the effects of such an event, as well as to assess how long it is safe to operate in the danger zone. Such information is particularly important for the services providing assistance to all those affected.

Fortunately, accidents and incidents involving the risk of ionising radiation are not frequent. However, there is still concern that radioactive materials, particularly nuclear weapons, may fall into the wrong hands and become a tool of terrorism. The most frequently mentioned methods and ways of using radioactive isotopes as weapons and tools of terrorism include:

- Detonation of a nuclear bomb or warhead. Terrorists can gain possession of nuclear weapons by stealing an already manufactured bomb or by constructing one themselves. There are indications that stealing a bomb from a poorly guarded or unsecured storage facility is quite likely. Particularly given the large number of nuclear warheads stored in the arsenals of the nuclear powers. It is also possible for terrorists to make their own bomb, especially given the availability of plans for such weapons on the internet. However, such production requires the possession of an adequate quantity of high-grade fissile material (5-25 kg of uranium ^{235}U or 1-8 kg of plutonium ^{239}Pu), the production of which is a very complicated and expensive process, but which can be obtained illegally.
- Use of radiological weapons. Unlike to the use of the atomic bomb, today's terrorists are much more likely to use so-called "radiological weapons", i.e. devices, means or methods to disperse radioactive material into the environment with the aim of causing health disorders and fear and panic among the population (radiological dispersal device RDD). The intended radioactive contamination of the environment may be achieved, for example, by direct spraying of a radioactive isotope mixture from an aircraft or by dispersal from a moving vehicle, but the most effective seems to be the mixing of the radioactive material with a conventional explosive charge to create a so-called "dirty bomb". A source of radioactive raw material for a "dirty bomb" could be either fissile material from nuclear warhead, spent nuclear fuel from power stations or much easier to acquire radioactive isotopes commonly used in industry and medicine (among other ^{99}Tc, ^{131}I, ^{60}Co, ^{137}Cs, ^{192}Ir, ^{226}Ra, ^{103}Pd, ^{14}C, ^{51}Cr, ^{65}Zn, ^{57}Fe, ^{85}Kr, ^{90}Sr, ^{169}Yb, ^{239}Pu, ^{241}Am). Unlike a nuclear explosion, a dirty bomb explosion usually does not cause contamination of an area with a radius greater than a few hundred metres (the radius of a conventional bomb explosion determines the area in which the radioactive material is scattered), and the level of contamination is rather low. The direct victims of the use of such a bomb are people in the vicinity of the detonation, who suffer thermal and mechanical injuries and become radioactively contaminated. However, an additional significant effect (apart from the health effect) of the use of such a bomb is disruption of the sense of security in the society, spreading panic and destabilisation of the state, local government structures or important critical infrastructures.
- Attack on a nuclear installation. Currently (data as of 2022), there are more than 415 nuclear power reactors in operation worldwide, another 26 are in long-term storage and 53 are under construction. To this number must be added nearly 600 reactors used for scientific purposes. It is therefore not surprising that the threat of a terrorist attack on nuclear installations in power plants or research centres should be regarded as unlikely, but possible. Although the operation of a reactor is immediately blocked in the event of an accident, the explosion could release significant amounts of radioactive material (principally iodine ^{131}I) into the environment, similar to what happens when a relatively small nuclear charge is detonated (as happened in the Chernobyl accident). It is estimated that the radius of the contamination zone for an explosion of a power reactor of the order of 3 000 MW could be between 25 and 100 km, and for an explosion of a research reactor of the order of 30 MW between 2 and 20 km.

When analysing the threats associated with the use of radioactive isotopes by mankind, one cannot also underestimate the possibility of their point (dedicated) use to eliminate (kill) specific individuals. In particular, alpha-emitting isotopes (emitting alpha radiation), which become lethal after entering the human body, can be used for this purpose. Alpha radiation is poorly penetrable, so that when emitted from radioisotopes introduced into the body, it deposits considerable energy in the tissues and organs, causing the human being to receive an equivalent lethal dose in a short time. The advantage, from the point of view of a terrorist or assassin, is that this radiation will hardly be emitted outside the contaminated person's body, so it will not be dangerous to other people or the environment. At this point it is worth mentioning that it is assumed that deterministic effects of irradiation can be observed from effective doses above 500 mSv, where at a dose of 1-2 Sv a person experiences vomiting, diarrhoea, decreased resistance to infections, at a dose of 2-3 Sv there is a strong radiation sickness (ARS), nausea, and probability of death (about 25%), at a dose of 3-4 Sv the chance of human death increases to 50%, while higher doses in most cases lead to death within a few days due to, i.e. destruction of bone marrow. Smaller single effective doses of 150-200 mSv can cause stochastic effects, including delayed tumours and chromosomal abnormalities.

6. Personal protection equipment and decontamination

6.1 Personal protection equipment

6.1.1 Respiratory protection

Respiratory tract, as was mentioned in earlier chapters, is the easiest way for dangerous material to enter the human body. According to the fact that humans need to breathe, protection of the respiratory tract is the most important in consideration of personal protective equipment. Also, using a mask simultaneously protects the gastrointestinal track of exposure. There are several levels of respiratory protection which are as follows, beginning with the safest:

Self-contained breathing apparatus (SCBA) - consists of a full-face mask and cylinder with compressed air. Allows to operate in a highly contaminated environment and in lack of oxygen. Main disadvantage is limited time of action, limited by volume of air in the cylinder. SCBA is the first choice for most first responders when operating in contaminated environment. Great advantage in using SCBA is that there is no need to know the contamination agent.

Figure 14 - USMC CBRN specialists wearing self-contained breathing apparatus

Source: Lance Cpl. Jered Stone, Public domain, via Wikimedia Commons

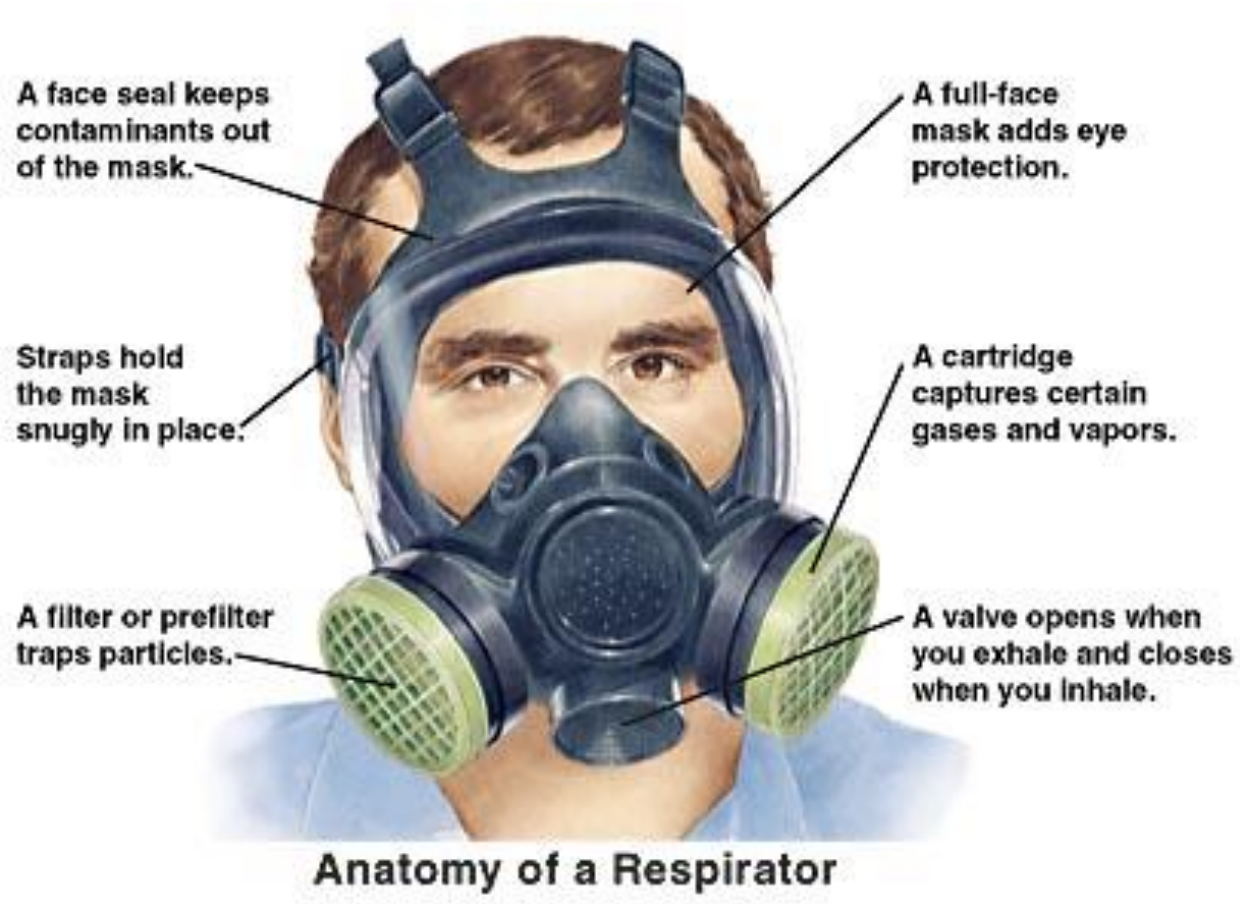
Full face mask with absorbing cartridge - offers protection of respiratory tract and eyes. Adsorbing cartridge allows to operate much longer than with SCBA but under several conditions: oxygen level is sufficient for breathing, concentration of chemicals is quite low, and the type of cartridge is matched to present substances. Time of usage of absorbing cartridge is limited due to its absorbing capacity. It depends on cartridge size, concentration of chemical agent and volume of filtered air. Absorbing cartridges can also have P3 filter which block dust and biological agents. To estimate if

absorbing cartridge is still operational after exposure to chemicals, producers of cartridges begin to equip them with end-of-service-life indicators, showing when cartridge should be changed or when operator should leave the contaminated zone.

Figure 15 - Full-face mask with absorbing cartridge

Source: Sgt. 1st Class Javier Orona, Public domain, via Wikimedia Commons

Figure 16



Types of cartridges, substances group which they protect against and sorbent capacity are coded by colours and by alphanumeric code covered by EN 14387 norm.

Table 5 - Colour and alphanumeric codes of cartridges according to EN 14387

Harmful substances group	Colour	Alphanumerical marking		
		Low sorbent capacity	Medium sorbent capacity	Large sorbent capacity
Organic gases and vapours with boiling point above 65 °C	Light brown	A1	A2	A3
Inorganic gases and vapours, with the exception of carbon monoxide	Grey	B1	B2	B3
Sulphur dioxide and other acid	Yellow	E1	E2	E3

gases and vapours				
Ammonia and its organic derivatives	Green	K1	K2	K3
Organic compounds with low boiling temperature (<65°C)	Dark brown	AX		
Particular gases specified by the manufacturer	Violet	SX		
Nitrogen oxides	Blue	NO		
Mercury vapours	Red	Hg		
Radioactive iodine including methane iodine	Orange	Reactor		
Carbon monoxide	Black	CO		

Filtering mask - offers different levels of protection depending on the construction and used materials. Most preferred are filter masks classified as FFP3, they offer best protection from dust or powdered substances. Filtering masks do not offer protection against gases and vapours. According EN 149 standard there are three levels of protection.

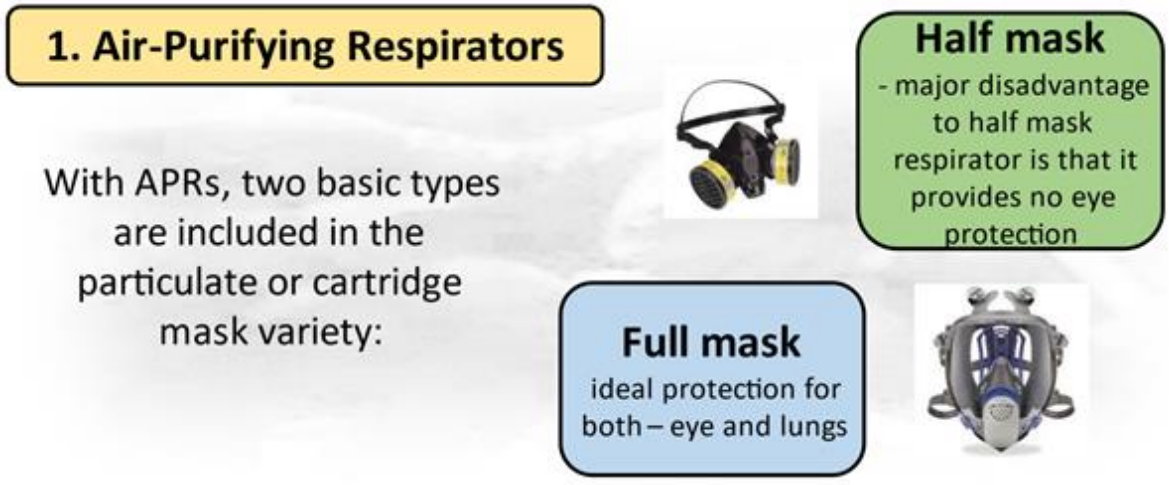
Table 6 - Class of protection of filters

Class of protection	Filter penetration limit (air flow - 95 l/min)	Inward leakage	Colour code
FFP1	at least 80% of airborne particles	< 22%	Yellow
FFP2	at least 94% of airborne particles	< 8%	Blue or white
FFP3	at least 99% of airborne particles	< 2%	Red

Figure 17 - FFP3 filtering mask

Source: Rudolf Goldhammer Ru-go, public domain, via Wikimedia Commons

Figure 18 - Air-purifying respirators



Source: own collaboration

6.1.2 Skin protection

Skin is the second most important way of exposure to dangerous materials. Protecting skin from contact with gases, vapours, droplets or solid particles is important to conduct operations in a contaminated environment because of protection of personnel and to minimise cross contamination. Similar to respiratory protection, there are several levels of protection which are as follows:

Fully encapsulating, positive pressure suit - this suit completely separates its wearer from the outside environment. It is made from highly resistant to chemicals material and also has positive pressure inside so even after some damage, risk of contamination of the inside of the suit is minimised. Despite offering the best chemical protection, it has some disadvantages. It requires using SCBA (positive pressure comes from exhaled air) and is very physically demanding, also operating in encapsulating suit is difficult due to narrowed vision and hard gloves. Fully encapsulating suit needs to meet requirements of EN 943 norm. According to this norm it is Type 1 protection, in United States level A offers equivalent protection.

Figure 19 - Firefighters in fully encapsulating suit

Source: Pressestelle BFK Urfahr-Umgebung, CC BY 2.0 <https://creativecommons.org/licenses/by/2.0>, via Wikimedia Commons

Hooded chemical protective suit - overall made from chemical resistant material. In combination with a full-face mask, chemical resistant gloves and boots offer protection against liquid chemicals. This type of protection suit is non gas tight but offers protection against liquid and gaseous chemicals (type 2), jet liquid (type 3), saturated liquid (type 4) or dry particulates (type 5) or light spray (type 6). Depending on used respiratory protection – SCBA, absorption cartridge or filter, it is equivalent of level B, C and D protection. Type 2 protection suit need to meet requirements of EN 943 norm, type 3 and 4 EN 14605 norm, type 5 EN ISO 13982 and type 6 EN 13034.

Figure 20 - Firefighters in type 3 suits with SCBA

Source: own collaboration (Ł. Faralisz)

Biological protective suit - in general looks similar to chemical protective suit, the main difference is material from which it was made. It is lighter than typical chemical protective suit. Biological protective suits can be also encapsulating or non-gas tight overalls. This kind of protection suits needs to meet requirements of EN 14126 norm.

Figure 21 - Rescuers in biological protection suite (white)

Source: Ministério da Defesa, CC BY 2.0 <https://creativecommons.org/licenses/by/2.0>, via Wikimedia Commons

Properly chosen and done personal protective equipment allows to safely conduct operations in contaminated environments and then conduct a successful decontamination process.

6.2 Decontamination

After the CBRN incident one of main concerns is released chemical, biological or radiological material. Area, where the materials spread and pose a threat is called the hot zone or red zone. Every person and the piece of equipment in these areas is contaminated or is considered as contaminated. Decontamination is the process of removing contaminants (chemicals, biological agents or radiological agents) from persons, objects, areas. Main goal of decontamination is to cut time of exposure to dangerous agents (only in the case of skin exposure) and keep the hot zone as small as possible. People, vehicles or equipment, if not properly decontaminated, can spread contamination from the place of initial spread to their homes, hospitals and many other places, where CBRN agent could be a threat to other people.

In general, there are few types of decontamination. It could be initial decontamination, final decontamination, single person/equipment decontamination or mass decontamination. Type of decontamination that needs to be perform depends on number of causalities, type of agent, place of release and first responders' capacity.

6.2.1 Mass decontamination

Mass decontamination (mass decon) is a process conducted when CBRN agent release take place in large gatherings of people. In these cases, where lots of casualty is present and a lot of people

are considered contaminated, it is technically difficult to provide a proper decontamination process to every single person. In this case, for first responders, it is important to remove contamination with as many people as possible as soon as possible.

Foundations of mass decontamination are decontamination corridors. They could be built in many different ways, depending on technical capabilities and tactics. Decontamination corridors could be established on containers, tents or made from fire hoses and fire engines. Most important in mass decontamination is to provide quick decontamination to as many affected persons as it is possible. People, who can walk by them self, go through decontamination corridor where are sprayed with clean water or water with mild soap solution. Things to consider in this kind of decontamination is where to place the corridors, so as much as possible people will walk through, and what to do with run-off water. In most situations, where chemical or biological agent are involved, water in excess will dilute and hydrolyse most agents to safe state. With radiological contamination, it is important to collect that water, to not spread this kind of contamination.

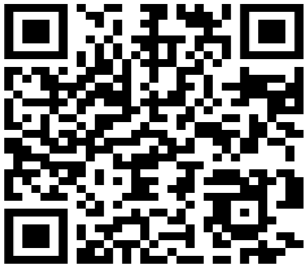
Mass decontamination with fire engines

6.2.2 Personal decontamination

After an CBRN incident not every person will be injured but every person should be treated as contaminated. In particularly in mass event taking place on open areas, it is possible that people will leave place of attack without going through decontamination corridor. After returning home they should conduct self-decontamination. Process of self-decontamination should be conducted even after going through decontamination corridor. As was shown before, it is method of rough cleaning, it is possible that some contamination will stay on body or clothes.

Self-decontamination could be conducted in two manners. First, after returning to home person should take off clothes, pack them in plastic bags and seal them. Then take a shower in lukewarm water with soap. This process is greatly described and shown on Centres for Disease Control and Prevention.

Would be great to have a layout picture as an example …



Self-decontamination after exposure to chemical agents

Self-decontamination after exposure to radiological agents

Personal decontamination could be conducted also with individual decontamination kits. Depending on the producer they can consist of decontamination powders or wet decontamination wipes. In some cases, self-decontamination kits with powder or wipes and substitute clothing will be provided by first responders. This kind of decontamination is possible when there are few people contaminated, due to limited number of decontamination kits and long-time which consist of instruction how to use it and then actual use of kit.

Figure 22 - Polish self-decontamination kit with wet wipes and substitute clothing

Source: own collaboration (Ł. Faralisz)

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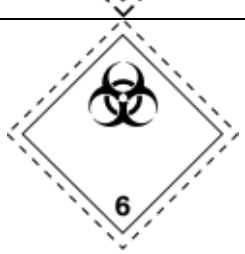
8. Appendix C

Table 7 - ADR classes

Pictogram	Hazard class description
	Class 1 .Explosives substances and articles
	Class 1.1 substances and articles which have a mass explosion hazard
	Class 1.2 substances and articles which have a projection hazard but not mass explosion hazard
	Class 1.3 substances and articles which have a fire hazard and either a minor blast hazard or a minor projection hazard or both, not a mass explosion hazard
	Class 1.4. substances and articles which present only a slightly risk of explosion in the event of ignition during carriage
	Class 1.5. very insensitive substances having a mass explosion hazard
	Class 1.6 extremely insensitive articles which do not have mass explosion hazard

Class 2 Gases	
	Class 2.1 Flammable gas - gases which are ignitable when in mixture of 13% or less by volume with air; have a flammable range with air of at least 12 percentage points regardless of the lower flammable limit
	Class 2.2 non-flammable - gases which dilute or replace the oxygen normally in the atmosphere or may by providing oxygen, cause or contribute to the combustion of other material more than air does.
	Class 2.3 toxic gas - gases which are known to be so toxic or corrosive to humans as to pose a hazard to health or have a LC50 value equal or less than 5000 ml/m3
	Class 3 Flammable liquids - liquids which have a flash-point of not more than 60°C
Class 4 Flammable solids	
	Class 4.1 Flammable solids, self-reactive substances and solid desensitised explosives
	Class 4.2 substances liable to spontaneous combustion - pyrophoric substances (ignite on contact with air), self-heating substances
	Class 4.3 Substances which in contact with water, emit flammable gases
Class 5 Oxidizers	

	Class 5.1 Oxidising substances - substances which, by yielding oxygen, cause the combustion of other materials
	Class 5.2 Organic peroxides - substances with weak oxygen bonding, which are liable to exothermic decomposition at normal and elevated temperatures
Class 6 Toxic substances	
	Class 6.1 Toxic substances - substances which are known that in small quantities can cause damage to human health
	Class 6.2 Infectious substances - substances which are known to contain pathogens (including bacteria, viruses, rickettsiae, parasite, fungi and prions)
Class 7 Radioactive materials	
	Class 7 Radioactive materials – extremely low level radiation
	Class 7 Radioactive materials – low level radiation
	Class 7 Radioactive materials – higher level radiation

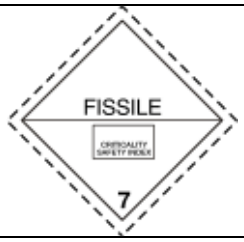
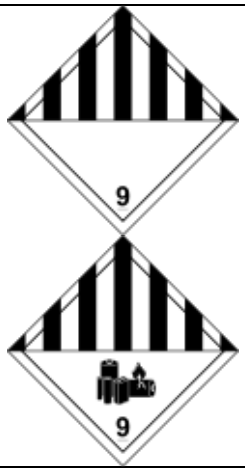
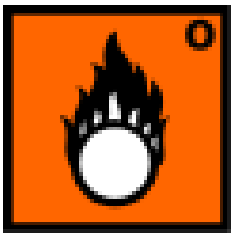
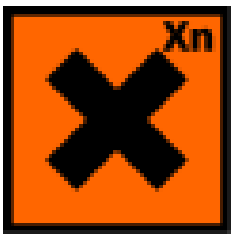
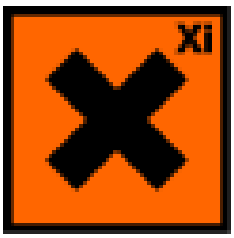
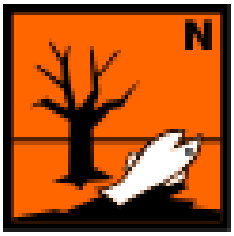
	Class 7 Radioactive materials – fissile materials
Class 8	
	Class 8 Corrosive substances - substances which, by chemical action, attack skin or mucous membranes or are capable of damaging or destroying other materials.
Class 9	
	Class 9 Miscellaneous dangerous substances - substances and articles which present danger not covered by other classes. This class contains also: - substances in form of fine dust, which may cause danger after inhalation; - substances which, in event of fire can form dioxins; - substances evolving flammable vapours; - lithium batteries; - life saving appliances; - environmentally hazardous substances; - elevated temperature substances; - other dangerous substances.

Table 8 - CLP pictograms

Old Pictogram (before 2017)	New pictogram (after 2017)	Description
		Unstable explosives, self-reactive substances, organic peroxides

 		Flammable gases, aerosols, liquids, solids, oxidising gases, organic peroxides, self-reactive substances, emitting flammable gases in contact with water, self-heating, pyrophoric
No pictogram		Compressed gases
		Skin corrosion, eye damage, corrosive to metals
		oxidising liquids and solids

		Acute toxicity
No pictogram		aspiration toxicity, cell mutagenicity, carcinogenicity, reproductive toxicity, damage to specific organs
 		Irritants, respiratory or skin sensitizers, lower class acute toxicity, narcotic, respiratory irritant, hazardous to ozone layer
		hazardous to the environment

8.1 Blistering agents

Blistering agents otherwise known as vesicants, are toxic substances causing skin injuries resembling burns. There are two main groups of blistering agents depending on chemical composition:

- **Mustards - including sulphur mustards (HD) and nitrogen mustards (HN)**
- **Arsenicals - lewisites (L).**

Sulphur mustard which is known also as mustard gas or yperite (also known as king of chemical warfare agents) in pure state is colourless and odourless liquid. It has low volatility and is barely soluble in water. Sulphur mustard has great chemical and physical stability which contributes to long shell life.

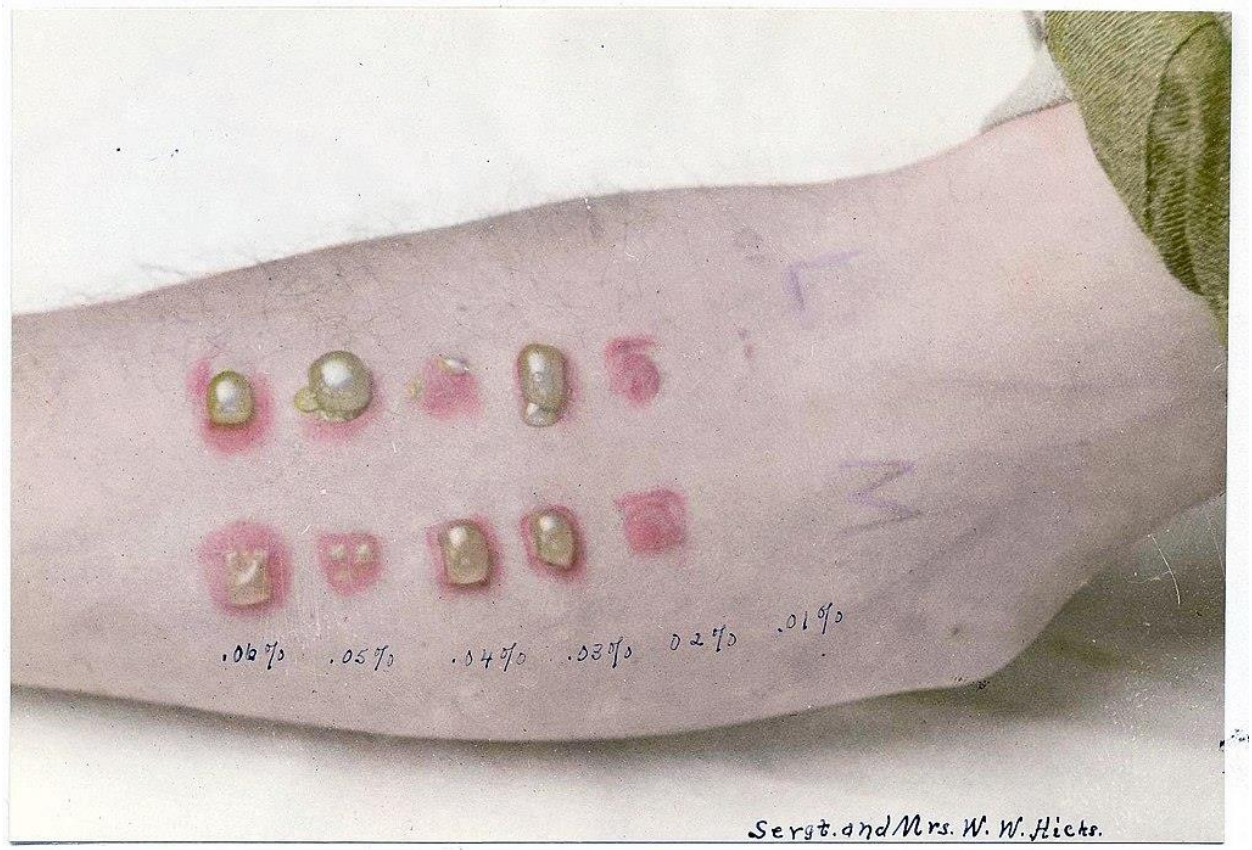
These properties make it a great chemical warfare agent. Impure **sulphur mustard could have yellow to amber colour and characteristic smell similar to garlic, mustard or rotten onions**. Characteristic smell can be felt only after initial exposure due to dullness of sense of smell after a few breaths. Also, after long years of storage sulphur mustard became denser up to solid state, resembling amber. Nitrogen mustards in pure state are also colourless liquid but are less soluble and less stable in storage then sulphur mustard.

Lewisite was synthesised for military purposes due to its non-flammable property with toxicity similar to sulphur mustard. Pure lewisite is odourless, colourless oily substance. It is insoluble in water but is less stable in storage than sulphur mustards. Ale it is more volatile than mustards. Technical impurities of **lewisite could give them blue, brown or black colour and fruity to geranium like smell**. Lewisit has no verified use as chemical warfare agents but it was used as a freezing-point depressant for sulphur mustard.

Exposure to blistering agents may occur via liquid and vapours, via inhalation, ingestion and by skin contact. This agent attacks skin, eyes, lungs, gastrointestinal tracts and, when absorbed into the body, blood-generating organs. **Most characteristic symptoms of exposure to blistering agents, especially sulphur mustard, are skin burns that begin to appear after 1 or 2 minutes of contact with the substance. Burns transform to blisters; feelings of pain appear after 4 up to 6 hours.** Sulphur mustards are lipophilic (easily dissolves in fats) and due to that they quickly penetrate skin, textiles and rubber, causing cells death and reaching blood vessels. Delayed effects of exposure are specific to sulphur mustard. Symptoms of eye contamination appear from 30 minutes to 3 hours after exposure, depending on time of exposure and concentration of agent. Symptoms of eye contamination are feeling of grittiness, soreness, bloodshot appearance, evolving pain, lacrimation and photophobia. Lewisite is absorbed by skin up to ten times faster than mustards due to that, exposure will cause immediate pain and irritation of affected organs. Lewisites also could attack skin, eyes, lungs and gastrointestinal tracts, depending on the way of exposure. They also cause symptoms of arsenic toxicity (because of arsenic in chemical structure) which leads to diarrhoea, neuropathy, haemolysis, shock and encephalopathy.

Sulphur mustard could still be found in old (WWII and WWI) aerial bombs and artillery shells buried in ground, dumped in the sea or in forgotten, hidden war magazine.

Figure 23 - Blisters from lewisite (upper row) and sulphur mustard (lower row)



Source: Otis Historical Archives nat'l Museum of Health & medicine (OTIS Archive 1), CC BY 2.0 <https://creativecommons.org/licenses/by/2.0>, via Wikimedia Commons

8.2 Blood agents

Blood agents are chemicals containing cyanide groups, main agents in this class are hydrogen **cyanide (HCN) and cyanogen chloride (CNCl)**. Name of this class, “the blood agents”, is a misnomer. Firstly, it was believed that blood agents interfere with oxygen uptake from the blood. In fact, their toxic effect relies on interrupting the electron transport chain in the inner membranes of mitochondria, which take place on cellular level, they also inhibit certain specific enzymes.

Hydrogen cyanide at temperature between -14°C and 26°C and in atmospheric pressure is colourless to yellowish-brown, highly volatile liquid. In gaseous state it is lighter than air so in open spaces it will dilute within atmospheric air. Hydrogen Cyanide could be used with high efficiency in closed spaces. **HCN has the characteristic scent of bitter almonds or marzipan.** In fact, not everyone is able to smell it. Due to recessive genetic trait, some people can’t smell hydrogen cyanide. Most likely route of exposure for hydrogen cyanide is via inhalation. One of the **first symptoms of HCN poisoning is hyperventilation** which, in a contaminated environment, leads to increasing inhaled dose. Hydrogen cyanide can penetrate skin only in liquid state (also as aerosol). As it was said one of the earliest symptoms of poisoning is **hyperventilation, weakness, dizziness, confusion, sense of warmth, metallic taste in mouth, pale-red colour of skin then poorer vision, painful respiration, lack of coordination, hypoxic convulsion, unconsciousness and in the end respiratory failure.**

Cyanogen chloride was introduced as a chemical warfare agent to overcome the disadvantages of hydrogen cyanide. Unlike hydrogen cyanide, it is **heavier than air gas and has a cumulative poisoning effect**. It is also a powerful lachrymator and choking agent. Cyanide compounds are widely used in the chemical industry, especially in production of plastics, and in gold and silver mining.

8.3 Nerve agents

Nerve agents, in some sources known as “nerve gases”, are organophosphorus or organophosphate compounds. Their name refers to the mode of action of these substances. The poisonous effect works on the basis of inhibiting the acetylcholinesterase enzyme in its reaction with acetylcholine. This process takes place between muscles and the nervous system. Without acetylcholinesterase, enzyme muscles don’t know when to relax and remain constantly stimulated. This effect can lead to death via different routes.

In general, nerve agents are far more toxic than other chemical warfare agents. They can cause toxic effects or even death in low concentrations and short time of exposure. There are two groups of nerve agents:

- G-agents - Tabun (GA), Sarin (GB), Soman (GD)
- V-agents - VX

As pure substances, nerve agents are colourless, odourless liquids. Impurities can give **them colours from slightly yellow to brown**. Also, impurities from synthesis could give nerve agents a certain smell, for g-agents, it will by faint **fruity scent**, for v-agents **amine odour**. G-agents are quite volatile and water-soluble, in water they hydrolyse to non-toxic products. V-agents are less volatile and less water-soluble than g-agents and therefore more persistent in the environment. They were developed out of a need for a more chemical stable version of nerve agents.

Despite different chemical structures, all nerve agents have the same toxicity mechanism generating the same symptoms of poisoning. First, and **most characteristic symptom is miosis** (pinpoint pupils), later with increasing concentration and duration of exposure the symptoms will be as follows: frontal headache, eye pain on focusing, dimness of vision, nausea, vomiting, tightness in chest, wheezing breathing, cough, diarrhoea, weakness, muscular twitching, tremors, convulsion.

Finally, death results due to respiratory paralysis, with enough high concentration of nerve agent, death can be immediate. Nerve agents can be absorbed through anybody surface, most rapid is via the respiratory tract.

Nerve agents were used several times, long after the development, in an example sarin attack on Kurds (Iraq 1988), sarin attack in the subway (Japan 1995) or assassination of Kim Jong Nam.

Footage of Kim Jong Nam assassination with VX

8.4 Choking agents

Choking agents are a group of chemicals consisting of the following substances:

- Chlorine
- Phosgene
- Diphosgene
- Perfluoroisobutene (PFIB)
- Chloropicrin

Chlorine, at room temperature, **in green-yellow gas with a characteristic smell**. It is widely used in many chemical processes and reactions, also it can be easily obtained from commonly available substances. Chlorine gas is heavier than air and after release will accumulate at bottom spaces. With moisture for example at the mucosa of lungs or eyes, the chlorine will form hydrochloric acid causing chemical burns of these organs. **Chlorine poisoning causes coughing, vomiting, lung damage, and finally death**.

Chlorine leak

Phosgene (carbonyl dichloride) is a colourless gas at room temperature, in temperatures below 8℃ phosgene is fuming liquid. Similar to chlorine, phosgene in the gaseous state is heavier than air and will accumulate in lower grounds. Phosgene does not occur naturally but is widely used in the chemical industry, also is produced during the thermal decomposition of PVC, it is described **as smelling like fresh-cut grass or moulding hay**. Phosgene mainly attacks the eyes and respiratory tract. Irritation of eyes, nose, and throat, chest tightness, shortness of breath, and cough are the first symptoms of phosgene poisoning. Second, the asymptomatic phase may occur from 1 hour after exposition up to 48 hours. The third phase of poisoning is pulmonary oedema which may end up being fatal.

Diphosgene is a derivative of phosgene after reaction with chloroform. Depending on impurities it can be colourless up to yellow-brownish liquid. Diphosgene has a much higher boiling point than phosgene, due to that it is easier to handle and more persistent in the environment. The poisoning effects of diphosgene are similar to phosgene.

Perfluoroisobutene (PFIB) is colourless, odourless gas. PFIB is a by-product of teflon (polytetrafluoroethylene) synthesis, also it could be obtained by heating this kind of polymer to temperatures of thermal decomposition (above 360℃). PFIB toxicity based on damaging the air-blood barrier of lungs causing oedema. Although lungs are the main attacked organ, high concentrations of PFIB also irritates eyes, nose and throat. Symptoms of exposure appear 1 up to 4 hours after exposure, initially they could be mistaken for influenza (increase in temperature, shivering and sweating, sense of discomfort when breathing, dry cough).

Chloropicrin at ambient temperature is colourless up to yellowish-green oily liquid. It produces highly irritating vapour. It works as a lacrimator (riot-control agent) and as lung irritant. Exposure to chloropicrin is via inhalation and direct contact. Effects of exposure strongly depend on concentration and time. Low concentration will cause eyes, nose and throat irritation, lacrimation and coughing. Higher concentrations can lead to respiratory tract injury, pulmonary oedema and even death. Skin irritation from direct contact may result in permanent scarring. Ingestion of chloropicrin causes pain, nausea, gastroenteritis and even death.

8.5 Riot-control agents

Riot-control agents are chemicals that cause temporary incapacitation causing sensory irritation (lacrimation, sternutation, vomiting and/or pain). After exposure, the sensation of irritation is so strong that people exposed cannot behave rationally or perform coordinated activities. Riot-control agents are popularly called “tear gas” but in fact they are solids which need to be aerosolized to obtain incapacitation effect. There are several substances qualified as riot-control agents:

- Adamsite,
- CN agent,
- CS agent,
- CR agent,
- OC agent.

Adamsite (diphenylaminechlorarsine or DM) is **yellow to brown crystalline solid**. Developed during the First World War as a sternutator. Adamsite is highly irritant to the nose, throat and respiratory tract. Symptoms appear after 2 - 3 minutes after exposure, they are **sneezing, flow of viscous mucus, coughing, choking, headache in forehead, feeling of pressure in the ears and pain in jaws**. There are also sensation of **pain in the chest, nausea, weakness in leg, all-over trembling, mental depression** may also occur. Recovery from symptoms is complete after 1 or 2 hours after the exposure.

CN agent (2-chloroacetophenone) is **white, crystalline solid**. Symptoms occur instantly after exposure, main symptoms are **burning sensation of eyes, nose and throat, excess tears and salivation, chest tightness, shortness of breath**. Skin contacts with CN causes itching, erythema and even necrosis. In closed spaces CN could damage lungs leading even to death. After exposure symptoms persist for about 15 to 30 minutes. Damaged skin may recover up to 5 weeks.

CS agent (2-chlorobenzalmalononitrile) is **white, crystalline solid**. It was developed to replace CN for police use. It is more rapid in action then CN (about ten times), **intensely irritating to the eyes, nose and upper respiratory tract and also skin in direct contact**. Irritation of eyes and respiratory tract occur after 1 minute of exposure. Main symptoms are **stinging and burning eyes, lacrimation, salivation, sneezing, coughing, tight chest.** On exposed skin begins sting, burn and erythema may follow. After use, CS powder may be active for 5 days.

Soldiers exposed to CS gas

CR agent (dibenz-(b,f)1:4-oxazepine) is a **pale-yellow solid**. It is irritating to the eyes, nose and upper respiratory tract. CR is up to six times more potent than CS. Symptoms are similar to other riot-control agents, **burning eyes, lacrimation, salivation, sneezing and coughing**.

OC agent - is oleoresin capsicum, **natural oil obtained from chilli pepper**. It is widely used in the form of "pepper spray" containing about 1 to 10% of OC oil with solvent and propellant. OC is **irritant to eyes, nose and respiratory tract and also skin**. Irritation is caused by direct contact and occurs almost immediately. After exposure, irritation lasts for about 45 minutes. Main symptoms of exposure are the same as with other riot-control agents.

More detailed information about warfare chemical agents can be found on-line in example via Centres of Disease Control and Prevention.

CDC chemicals fact sheets

Police OC training

8.6 Psychomimetic agents

Psychomimetic agents are a group of chemicals causing conditions similar to psychotic disorder, loss of feeling, paralysis, hallucinations and other symptoms of damage to the central nervous system. These agents, besides behavioural effect, could also cause physical incapacitation. Physical symptoms of psychomimetic agents include blurred vision, fainting and vomiting. There are two psychomimetic agents with potential to use in CBRN incident:

- **Lysergide (LSD)**

- **Agent BZ**

Lysergide, better known as LSD, **is colourless, odourless and tasteless, water-soluble solid**. LSD is active after inhalation, oral or intravenous exposition. **First symptoms include mydraisi, dizziness, weakness, drowsiness, nausea and paraesthesia.** Psychophysiological symptoms begin after 30 up to 60 minutes after exposure, peak of effects occur after 3 - 5 hours after exposure. Recovery time is about 12 hours. Behavioural effects of LSD are different for each individual and are influenced by the local environment. Generally, in a familiar environment, people exposed to LSD may be more talkative and excited. In unfamiliar surroundings the main symptoms will be anxiety and panic attacks.

Agent BZ is water-soluble solid. It is structurally and pharmacologically similar to atropine. Agent BZ affects both the peripheral and central nervous system. Most likely route of exposure is inhalation, agent BZ could be disseminated in solution as aerosol, pyrotechnically or as a powder. Symptoms of exposure appears in phases which are as follows:

- First phase (1-4 hours after exposure): **restlessness, involuntary movement, ataxia, dizziness, vomiting, dry mouth, blurred vision, flushed skin, dry skin, dilated pupils, confusion, sedation**
- Second phase (4 - 12 hours after exposure): **stupor, inability to respond to environmental stimuli, hallucinations**
- Third phase (12 - 96 hours after exposure): **random unpredictable behaviour, increasing activity as individuals return to normal, hallucinations (entertaining or terrifying**), real objects may be ignored.

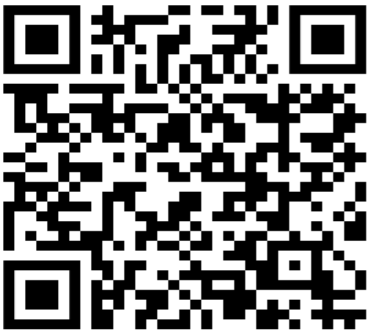
8.7 High hazard index toxic industrial chemicals

Representation of chemical compounds classified with high hazard index could be ammonia, formaldehyde and fuming nitric acid. **Ammonia (NH_3) is colourless gas with characteristic, pungent smell.** Gaseous ammonia is lighter than air, in specific concentration with air, between 15% and 25%, **ammonia could form flammable mixture**. Ammonia is water-soluble, forming basic solution. Exposure to gaseous ammonia causes **burning of the eyes, nose, throat and respiratory tract**. It could cause blindness, damage of lungs and even death. In lower concentrations it causes coughing, nose, throat and eyes irritation or burns. Threats from ammonia solution depends on its concentration. Solutions up to 10% are irritating in contact with skin, solutions with concentration higher than 10% are corrosive. Gaseous ammonia is also widely used in refrigeration, especially in industry, with boiling point at –33,34°C. Indirect contact with ammonium from refrigeration installation can cause cold burns. Global production of ammonia in 2021 was 236 million tons.

Ammonia release accident

Formaldehyde (CH_2O) as a pure substance is **colourless gas with pungent scent**. It is widely use as aqueous solution called “formalin”. Formaldehyde vapours are flammable and corrosive. In contact with skin, eyes and respiratory tract can cause **lacrimation, breathing difficulties, cough, burning of nose and mouth**. Contact with strong solutions can cause **skin irritation, contact with eyes can cause even loss of vision**. When ingested, formaldehyde cause irritation of mouth, throat, stomach and in most severe examples even death. Exposure to formaldehyde can cause permanent changes in the nervous system also, it is known as carcinogen. Formaldehyde is used as chemical intermediate, in production of resins and also as fumigant.

Fuming nitric acid is water solution of nitric acid with concentration of at least 86%. Depending on the amount of nitrogen dioxide fumes could be red or white. Nitric acid is colourless to red-brown liquid with suffocating odour. It is very corrosive, any contact with liquid or vapours may cause severe burns and even death. Characteristic to nitric acid burns are yellow stains caused by reaction with keratin. Nitric acid is widely used in many industrial applications also it is most common type of acid used in acid attacks.



Fuming nitric acid

8.8 Medium hazard index toxic industrial chemicals

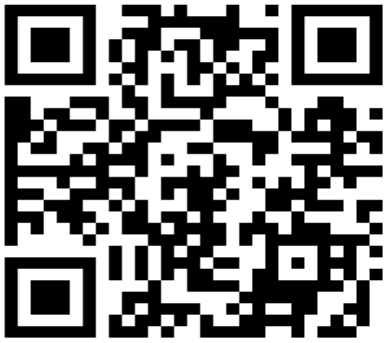
Chloroacetone is **colourless liquid with pungent scent**. When exposed to light it turns to **dark yellow colour**. Chloroacetone is use in organic synthesis, in manufacture of perfumes and drugs. Vapours of chloroacetone are heavier than air, it is also **highly flammable**. It is toxic by all routes of exposure. Effects of poisoning with chloroacetone contains **burns to the skin and eyes, nausea, caught**. Contact with liquid chloroacetone causes forming **blisters on skin**. High concentrations may lead even to death. Chloroacetone was used as a tear gas during World War I.

Dimethyl sulfate (DMS) is **colourless oily liquid with onion-like scent** (only in high concentrations). Due to its low cost and high reactivity, DMS is used in industrial scale methylation of phenols, amines and thiols. Dimethyl sulfate in **poisonous, carcinogenic, mutagenic and also corrosive**. It attacks all exposure tracts. Contact with skin causes **severe blistering**. Exposure via inhalation or oral can cause **inflammation, necrosis, injuries to internal organs and central nervous system**. First **symptoms of exposure may be delayed from 6 up to 24 hours**, due to that there could be no warning symptoms of exposure. Dimethyl sulfate is decomposed by water, in example form natural moisture of mucosal, forming sulfuric acid with evolution of heat.

Acrylonitrile is **colourless, very volatile liquid with onion or garlic like smell**. Impurities change its colour to yellow. Vapours of acrylonitrile are heavier than air. Acrylonitrile is widely used to produce plastics, rubber, resins and carbon fibers. It is highly toxic even in low concentrations and highly flammable. Main routes of exposure are via inhalation and skin. Symptoms of exposure to acrylonitrile are **headaches, nausea, difficult breathing, dizziness, convulsions even collapse**. It causes irritation when in contact with unprotected skin. Acrylonitrile is also anticipated to be carcinogen.

8.9 Low hazard index toxic industrial chemicals

Bromine at room temperature **is volatile red-brown liquid which evaporate quickly forming red-brown vapours.** It has **sharp, pungent smell**. Bromine has wide variety of applications, especially as organobromide compound in example as flame retardant. Liquide bromine causes chemical burns when in contact with skin, bromine vapours cause irritation, coughing, difficult breathing, chocking and finally death.



Elemental bromine

Cyanogen (CN_2) is **colourless gas with pungent, almond-like scent**. It is used in organic synthesis, welding and thermal cutting or as fumigant. When burning with oxygen, cyanogen flame can reach temperature up to 4525°C. Cyanogen is **highly flammable**, giving toxic fumes in fire. Also, cyanogen as gas is **toxic and irritating, causing giddiness, headache, nausea, loss of consciousness and even death**.

9. Appendix B

Microscopic images of selected microorganisms used as biological weapons

Figure 24 - Bacillus anthracis (figure used with permission under Creative Commons license)

Figure 25 - Yersinia pestis (figure used with permission under Creative Commons license)

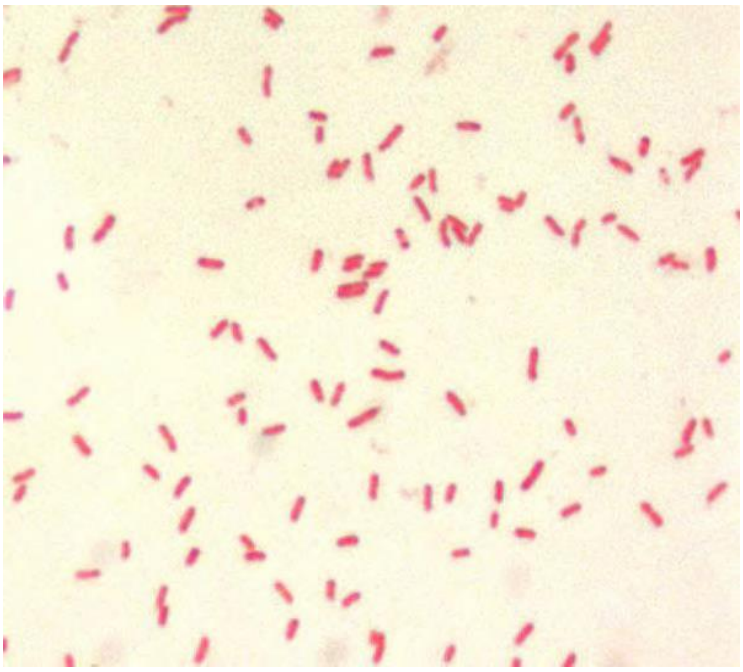
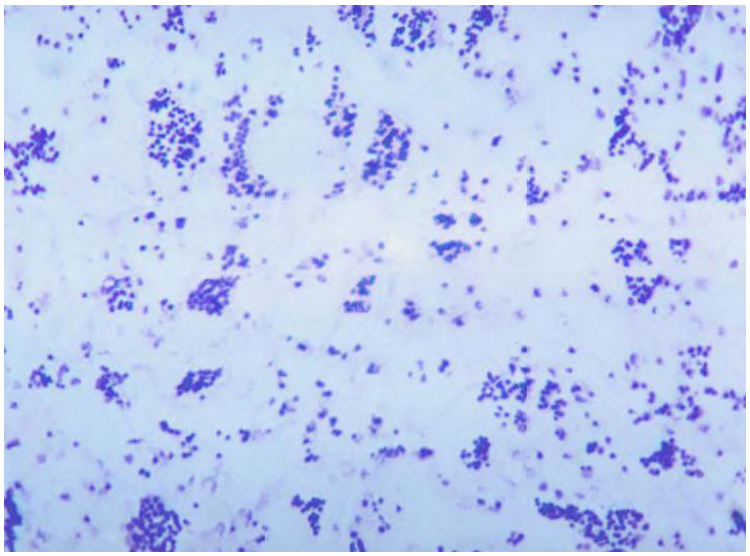


Figure 26 - Francisella tularensis
(figure used with permission under Creative Commons license)

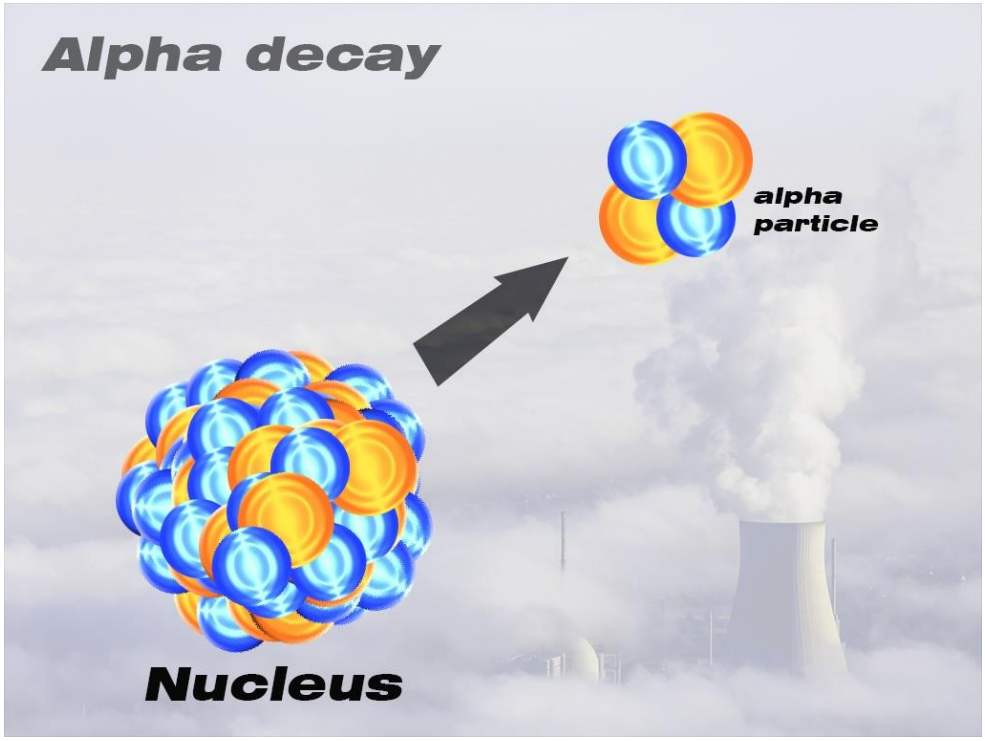


10. Appendix RN

10.1 Types of ionisation radiation

Alpha radiation - (denoted by the symbol α) is a type of ionising radiation emitted by radioactive atomic nuclei during nuclear transformations. Alpha radiation is produced during alpha decay and is a stream of helium ^{42}He nuclei (composed of 2 protons and 2 neutrons, with electric charge +2e = 2 electrons = 21.602×10-19 C), moving at a speed of a few percent of the speed of light (e.g. an alpha particle with energy of 5.5 MeV has a speed in vacuum of 15,000 km/s). Alpha radiation is strongly absorbed by matter - its range in air is only 1-10 cm centimetres, in tissue 1/10 mm and less. However, it can be extremely dangerous to eat food or inhale air containing substances that produce alpha radiation. Due to its relatively higher mass (e.g. in comparison to β-, β+ radiation) and electric charge, alpha radiation strongly ionises tissues of living organisms leading to serious damage and radiation sickness (ARS).

Figure 27 - Alpha radiation (scheme)

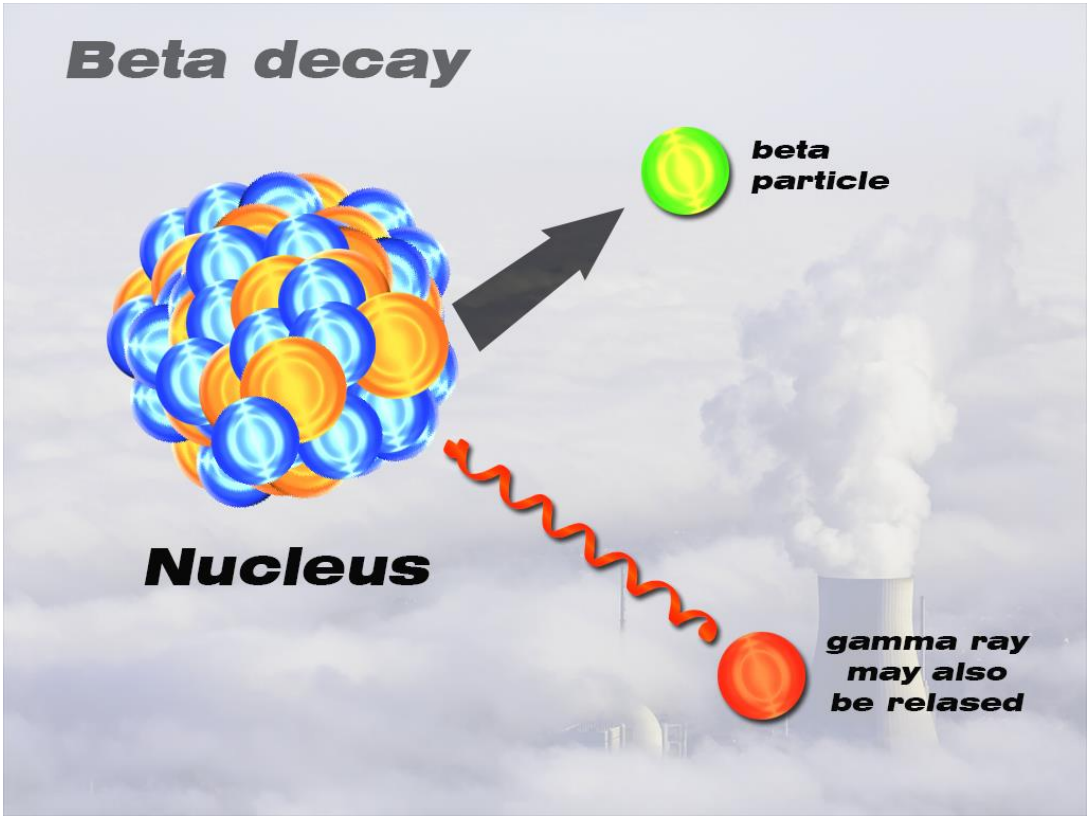


Source: schematic picture, own collaboration

Beta radiation - (denoted by β- or β+) is a type of ionizing radiation emitted by radioactive atomic nuclei during nuclear transformations. Beta radiation is produced during beta decay, it is a stream of electrons or positrons (with charge -1e = -1.602×10-19 C and +1e = 1.602×10-19 C respectively) moving at a speed close to the speed of light (e.g. an electron with energy of 5.0 MeV has a velocity in vacuum close to 299.000 km/s). β-radiation consists in the emission of an electron from the decay of a neutron in the nucleus of the element undergoing transformation. Atomic nuclei in which there are more neutrons than protons undergo this transformation. During the β- transition the neutron is transformed into a proton, an electron and an electron antineutrino. The β+ radiation is based on the emission of a positron from the decay of a proton in the nucleus of the element that is being transformed. Atomic nuclei in which there are more protons than neutrons undergo this transformation. During the β+ transition, the proton decays into a neutron, a positron and an electron neutrino. Beta radiation is strongly absorbed by matter, but not as strongly as alpha radiation - its range in air is of the order of a

few to several tens of metres, in water of the order of a few cm, in metal of the order of a few mm. Like alpha radiation, beta radiation ionises the tissues of organisms leading to damage, but this ionisation is weaker than that caused by alpha radiation.

Figure 28 - Alpha radiation (scheme)

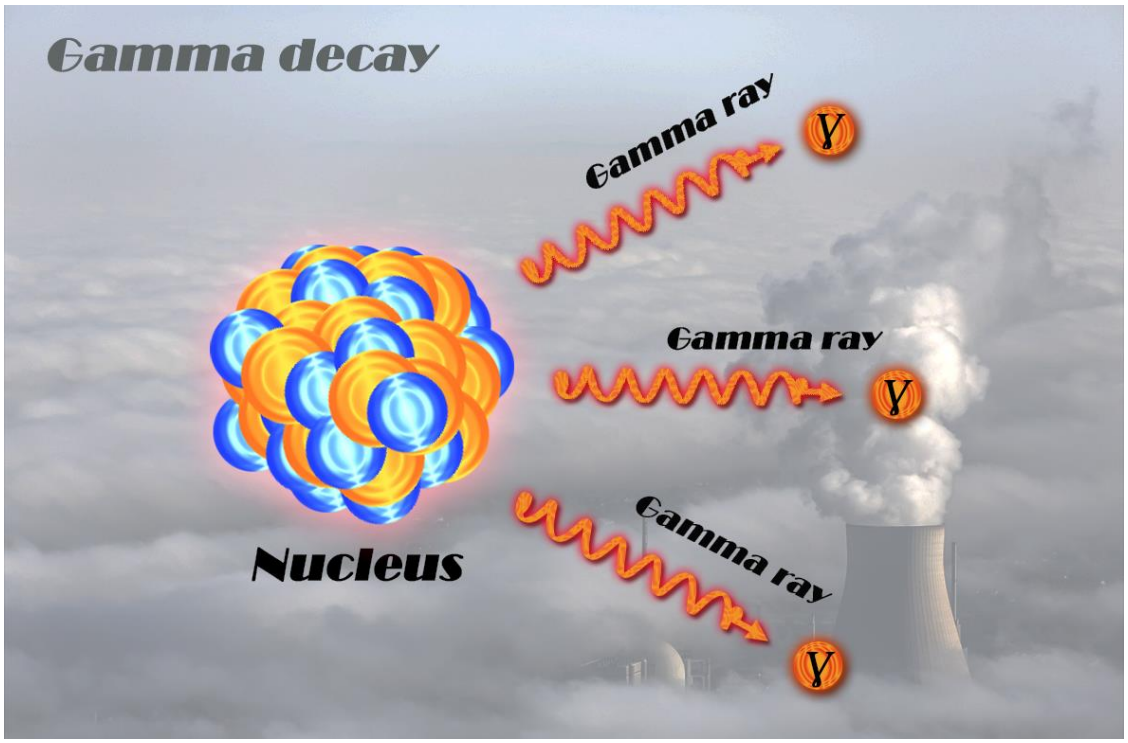


Source: schematic picture, own collaboration

Gamma radiation - (designated by the symbol γ) is a high-energy form of electromagnetic radiation. Gamma radiation is considered to be radiation with a quantum energy greater than 50 keV. This range partially overlaps with that of X-rays, hence in some scientific publications the distinction between gamma radiation and X-rays is based on their sources and not their wavelengths. Gamma radiation is produced by nuclear transformations or collisions of nuclei or subatomic particles and X-rays are produced by collisions of electrons with inner shell electrons or their scattering in the field of atomic nuclei. Gamma radiation is ionising and penetrating radiation. Unlike alpha radiation and beta radiation, gamma radiation has no maximum range in any material. The use of shields, according to the law of absorption, weakens the gamma radiation beam the thicker the shield. The heavier the shielding material used, the less thickness is required to provide the desired attenuation multiplication factor. Typical shielding materials used to attenuate gamma radiation are lead, depleted uranium, steel and concrete. The phenomena responsible for the absorption of gamma radiation in matter are: intrinsic photoelectric effect, whereby gamma radiation gives up energy to electrons, pulling them away from atoms or transferring them to higher energy levels; Compton scattering - weakly bound or free electrons experience acceleration in the direction of radiation propagation (there is a small change in the energy of a gamma quantum in a single act of interaction; as a result of interaction with many electrons the gamma quantum loses its energy); creation of electron-positron pairs - the gamma quantum, striking the atomic nucleus, causes creation of particle-antiparticle pairs (the condition of the phenomenon occurrence is the energy of the gamma quantum larger than twice the rest energy of the electron, i.e. 1.02 MeV); photo-nuclear reactions - in this interaction gamma radiation gives energy back to the atomic

nuclei, exciting them and, with a sufficiently high photon energy, producing new particles (the excited atomic nucleus can radiate a gamma quantum, undergo disintegration or fission). The contribution of the photoelectric phenomenon and Compton scattering to the total gamma-ray absorption in matter decreases with increasing energy in favour of an increasing contribution from pair creation. At low energies the photoelectric phenomenon dominates.

Figure 29 - Gamma radiation (scheme)



Source: schematic picture, own collaboration

Neutron radiation - is a form of ionising radiation that occurs in the form of free neutrons. The typical phenomena responsible for the emission of neutron radiation are nuclear fission or nuclear fusion causing the release of free neutrons, which then react with the nuclei of other atoms to form new isotopes, which in turn can release further neutron radiation. Neutrons can also be emitted from other nuclear reactions such as radioactive decay or particle interactions with cosmic rays. Neutron radiation is a penetrating radiation, much harder to absorb by shielding than alpha radiation or beta radiation. As with gamma radiation, the use of shielding only weakens the neutron beam. In order to protect against neutron radiation, materials are used which consist of substances having atomic nuclei which absorb neutrons (e.g. cadmium, hafnium, gadolinium, varieties of concrete containing barium salts or 3Hel which becomes tritium - a heavy isotope of hydrogen).

10.2 Definitions of physical quantities used for measuring the effects of ionising radiation

Source (radioactive) activity - A, is defined as the number of nuclear transformations occurring in a source per unit time:

$$A = -\frac{\mathrm{D}N}{\mathrm{D}t}$$

where:

- N is the number of nuclear transformations,
- t is the time interval.

The unit of activity is s^{-1} called becquerel (Bq).

Exposure dose - X, is a measure of the ionisation that occurs in air under the influence of X-ray or γ radiation:

$$X = \frac{\mathrm{D}Q}{\mathrm{D}m}$$

where:

- X is the exposure dose,
- Q - the absolute value of the sum of ion charges of one sign produced by photons in dry air when all electrons released in the air are completely inhibited,
- m - mass of air, in which complete braking of the created electrons takes place.

The unit of exposure dose is the coulomb per kilogram (C/kg). Formerly, the unit of exposure dose was the roentgen (R). The exposure dose was equal to 1 R if $2.08 \cdot 10^9$ ion pairs were formed in 1 cm3 of dry air.

Absorbed dose - D is a measure of the absorption of ionising radiation by various materials. More specifically, it is the ratio of the energy that is lost and absorbed by the medium through which the radiation passes, calculated per unit mass of that medium. This quantity makes sense for all types of ionising radiation and also for any medium in which the radiation is absorbed. The absorbed dose is calculated from the formula:

$$D = \frac{E}{m}$$

where:

- D is the absorbed dose,
- E is the mean energy of radiation transferred to the matter through which it passes,
- m - mass of the medium.

The unit of absorbed dose is joule per kilogram J/kg, called Gray (Gy). The previously used unit was rad (rd).

As can be seen, however, neither activity nor absorbed or exposure dose are sufficient to determine the extent of the biological effects that ionising radiation may cause in the human body. This is because these effects do not depend solely on the absorbed dose, but other factors also have a significant impact: type of radiation, ionisation density (i.e. the number of ions formed per unit distance travelled by the radiation in matter), sensitivity of organs and tissues, as well as age and general condition of the human being. For this reason, so-called "weighting factors" have been introduced, characterising the types and energies of radiation, as well as the types of tissues and organs, which make it possible to

take these factors into account in the calculation of the biological effects of radiation. In order to quantitatively describe the biological effects of radiation on living organisms, the concepts of equivalent dose (H_T), effective dose (E), committed equivalent dose ($H_T(\tau)$) and effective committed dose ($E(\tau)$) were also introduced.

Equivalent dose - H_T is determined as the product of the absorbed dose in a tissue or organ and the radiation weighting factor (w_R) according to the formula:

$$H_T = w_R \cdot D$$

where:

- H_T is the equivalent dose,
- w_R is the radiation weighting factor taking into account the energy and type of ionising radiation,
- D is the tissue or organ averaged absorbed dose.

If irradiation of tissues and/or organs occurs with different types of ionising radiation or with radiation of different energies, then the total equivalent dose is determined from the sum of the respective radiation weighting factors multiplied by the corresponding tissue or organ absorbed dose averaged over the different types of radiation. The weighting factors are unmeasured quantities. The unit of equivalent dose is $J \cdot kg^{-1}$, called sievert (Sv). The unit of equivalent dose formerly used was the rem.

The equivalent dose takes into account only the weighting factors and the energy of the ionising radiation, but does not take into account the sensitivity of the tissues and organs being irradiated. For this reason, the effective dose, also called the effective dose, was introduced.

The effective dose - E is the sum of the products of the equivalent doses and the corresponding weighting factors of the irradiated tissues and/or organs. In this way, not only the type and energy of radiation but also the sensitivity of the organs and tissues irradiated are included in the effective dose. In this way, it became possible to describe the total exposure of the body to radiation from external and internal sources. The effective dose is determined from the formula:

$$E = \sum_T w_T \cdot H_T = \sum_T w_T \sum_R w_R D_{R,T}$$

where:

- D_R is the absorbed dose from ionizing radiation R, averaged over tissue or organ T,
- w_R is the weighting factor for the type of ionising radiation R,
- w_T - weighting factor of tissue or organ type T.

The unit of effective dose is, as in the case of equivalent dose, the Sievert (Sv).

10.3 Half-life time

Half-life - (denoted by $T_{1/2}$) is the time during which the number of unstable nuclei decreases by half. The half-life is a parameter that characterizes a given radioactive isotope independently of external factors such as temperature, pressure, chemical form, and state of matter, and is a concept applicable to any type of radioactive decay. The time must satisfy the condition:

$$N(t) = N_0 \left(\frac{1}{2}\right)^{\frac{t}{T_{1/2}}}$$

where:

- N(t) is the number of atoms of the radioactive isotope remaining after time t,
- N_0 is the initial number of atoms of the radioactive isotope.

10.4 History of invention of nuclear fission reaction

The nuclear fission reaction is a process in which a nucleus splits into two or more fragments comparable in size. This phenomenon occurs for heavy nuclei and is more likely to occur in the excited state of the atomic nucleus than in the ground state, fission into two fragments of comparable mass being the most likely. Due to the fact that the fissioning heavy nuclei are rich in neutrons (the number of neutrons in the atomic nucleus is significantly larger than the number of protons), the fission fragments are also rich in neutrons. These fragments are created in highly excited states, emitting neutrons immediately after their creation (so-called immediate neutrons) in an average number of about 2.5 per one fission act, and also after decay (so-called delayed neutrons). This leads to the very important phenomenon that the emission of neutrons, which can in turn cause the fission of other nuclei, creates the possibility of a chain reaction. In 1905. Albert Einstein formulated the special theory of relativity, one of whose of its most revolutionary conclusions was the equivalence of mass and energy according to the formula:

$$E = mc^2$$

where:

- E is the rest energy (of the particle),
- m is the rest mass,
- c is the speed of light in vacuum.

Mass of the system of protons and neutrons bound in the atomic nucleus is smaller than the sum of their free (rest) masses by the value equivalent to the energy of their binding, i.e. energy needed for their complete separation. Therefore, the processes of creating bound systems are accompanied by the release of energy. Due to the fact that the binding forces of nucleons (protons and neutrons) in the atomic nucleus are much higher than the binding forces of electrons in the atom, the energies given off in nuclear reactions are about a million times higher than the energies given off in chemical reactions.

The phenomenon of forced fission of the atomic nucleus under the influence of neutrons was first observed in 1938 by Otto Hahn and Fritz Straßmann and the first more complete description of this phenomenon appeared in the work of Lise Meitner and Otto Robert Frische. They found that in a fission reaction the atomic nucleus almost always splits into two fragments and free neutrons, with an average of more than two neutrons per fission act.

10.5 The Chernobyl and Fukushima nuclear accidents

The Chernobyl and Fukushima nuclear accidents are considered to be the biggest accidents that have occurred in the world to date. They reached the highest possible level - 7 - on the International Nuclear and Radiological Event Scale (INES).

As a result of the Chernobyl accident on 26 April 1986, an area of about 135,000 km² on the border between Belarus, Ukraine and Russia was contaminated with radioactivity and the radioactive cloud emitted from the damaged reactor spread across Europe. Several hundred thousand people were evacuated and resettled as a result of the contamination. The deterministic effects of the Chernobyl disaster include:

- death of 3 plant personnel as a direct result of the explosion and fire;
- death of 28 firefighters from acute radiation sickness as a result of receiving an effective dose of 4 to 16 Sv;
- acute radiation sickness in 134 people who, out of 237 people (in addition to those mentioned above), were hospitalized as a result of radiation exposure during the fire activities - all of whom were treated.

To this must be added the stochastic effects of receiving higher radiation doses in a group of people who refused to evacuate and the people employed to remove radioactive material that was scattered in and around the plant as a result of the explosions. Between 600.000 and 800.000 people were assigned to such work. The average dose to these people was about 120 mSv. According to the International Atomic Energy Agency in Vienna, by the end of 2004 a total of 56 people had died as a result of the Chernobyl accident, including nine children from thyroid cancer. In addition to the direct effects, the Chernobyl accident had significant psychological effects. Research carried out in the most exposed areas of Russia, Belarus and Ukraine indicated that anxiety, lack of hope, depression and inadequate nutrition were the most serious problems and were the source of the actual physical ailments.

In the case of the Fukushima nuclear power plant, the cause of the accident on 11 March 2011 was an earthquake that triggered tsunami waves, which in turn led to damage to the emergency power systems. This resulted in the failure of the emergency cooling systems of some of the power units. The result was overheating and damage to the reactor cores and spent fuel pools. The Fukushima accident involved four reactors that contained almost ten times more radioactive substances than the Chernobyl reactor. Due to the appearance of large quantities of iodine (^{131}I) and caesium (^{137}Cs) in the air, the administrative authorities ordered the evacuation of the population, first from the three-kilometre zone directly adjacent to the nuclear power plant, then within a 20-kilometre radius, and finally within a 30-kilometre radius zone. The accident resulted in contamination of soil, drinking water and food. During the entire nuclear accident, it is estimated that significant amounts of caesium ^{137}Cs with an activity of 1017 Bq were released into the atmosphere. In addition, according to official data, by the end of March 2011, 21 power plant workers had received an effective dose of more than 100 mSv. The amount of radioactive substance that entered the atmosphere during the Fukushima accident is estimated to have been ten times less than during the Chernobyl accident.