HEALTH RISKS OF WATER AND SANITATION

HEALTH RISKS OF WATER AND SANITATION

Vom Himmel kommt es

Zum Himmel steigt es

Und wieder nieder

Zur Erde muss es

Ewig wechselnd

Goethe - Gesang der Geister über den Wassern

The Ministry of Housing, Spatial Planning and the Environment

National Institute of Public Health and the Environment

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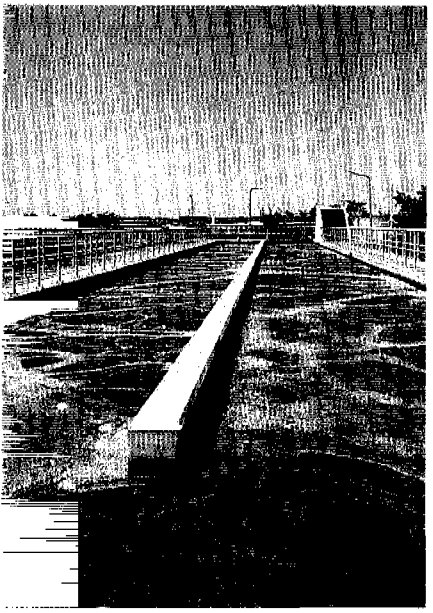
INTRODUCTION



WATER IS THE SOURCE OF LIFE ON EARTH FOR HUMANS, ANIMALS AND PLANTS. A HUMAN BEING WILL SURVIVE WITHOUT DRINKING WATER FOR ONLY A FEW DAYS. IN ANCIENT TIMES THE RELATION BETWEEN WATER SUPPLY AND HEALTH WAS KNOWN BY ASSOCIATING MARSHY PLACES WITH FEVERS. IT LASTED UNTIL THE 19TH CENTURY BEFORE THE PRECISE RELATION BETWEEN INFECTED WATER SOURCES AND DISEASES AS CHOLERA WAS SHOWN BY JOHN SNOW IN LONDON.

Worldwide the supply of drinking water can be very different over the countries. In western Europe, North-America and Australia drinking water is available in sufficient amounts and of relative good quality. In other parts of the world safe drinking water is not always available for all citizens because of scarcity or pollution of the sources.

Drinking water can be polluted with micro-organisms or/and with chemical pollutants. For tropical countries the micro-organisms form the biggest health threat. Diseases, especially diarrhoea and malaria, related to drinking water and sanitation are responsible for at least 5 million deaths per year worldwide.

This document gives information about diseases caused by exposure to unsafe (drinking) water and poor sanitation. Diseases caused by micro-organisms are classified in four categories which are based on the route of transmission of the agent. If it was not so clear in which category a disease should be classified, the classification of the WHO is chosen.

This booklet contains nine chapters, each chapter has got one or more paragraphs. Each paragraph gives information about a single disease, micro-organism or pollutant. So the paragraphs can be read separately. In every paragraph there is a short introduction to the subject. In facts, trends and effects information is given about the prevalence and symptoms of the disease. In causes and pathways information is given about the causing agent of the disease, for instance a micro-organism, the vector or pollutant and the transmission route is described. Every paragraph ends with some actions which can be taken to prevent the transmission of the disease.

This booklet gives information about the highlights of the most important diseases in one or an other way related to water all over the world. The information is derived from the public literature or the internet, mainly from the WHO-site. The authors did their very best to write the paragraphs in a way that the information and the message will be understood by a broad public and especially by the participants of the second World Water Forum in The Hague, March 2000.

1 WATER, SANITATION AND HEALTH

IN THE TIME OF HIPPOCRATES THE RELATION BETWEEN WATER SUPPLY AND HEALTH WAS ALREADY WELL KNOWN BY ASSOCIATING MARSHY PLACES WITH FEVERS. SNOW, IN HIS FAMOUS BOND STREET STUDY IN 1855 SHOWED THE PRECISE RELATION BETWEEN INFECTED WATER SOURCES AND CHOLERA. PRESENTLY, A TOTAL OF 20-30 MAJOR WATER AND SANITATION RELATED DISEASES ARE KNOWN TO BE CAUSED BY MICRO-ORGANISMS. FOR TROPICAL COUNTRIES THESE MICRO-ORGANISMS FORM BY FAR THE BIGGEST HEALTH THREAT, EVEN THOUGH VARIOUS CHEMICAL POLLUTIONS - ARSENIC, FLUOR, LEAD ETC. - CAN ALSO POSE HEALTH PROBLEMS.

FACTS AND TRENDS

Water contaminated by humans or animals and chemical or industrial wastes, can cause a variety of communicable diseases brought on by ingestion or physical contact. More than 20 years ago public health researchers realized the enormous impact if the transmission routes of water related infections and diseases could be broken by the use of safe water supply and hygienic sanitation.

Research about the relations between water supply and sanitation with health proposed to classify water-related diseases according to the transmission routes which resulted in the following now universally accepted four categories for sanitation related diseases: waterborne, water-washed, water-based and water-related diseases (see Table 1).

WATERBORNE DISEASES: caused by the ingestion of water contaminated by human or animal faeces or urine containing pathogenic bacteria, viruses or parasites. These diseases include cholera, hepatitis, poliomyelitis, typhoid fever, amoebic and bacillary dysentery and other diarrhoeal diseases. The classical waterborne diseases are due to highly infectious organisms, where only a few are needed - relative to the levels of pollution that readily occur - to infect the host. The two main diseases, typhoid fever and cholera, associated with high mortality if untreated, are diseases that a community is very anxious to escape. Both of the responsible organisms are relatively fragile and have as their sole reservoir, the human being.

WATER-WASHED DISEASES: caused by poor personal hygiene and skin or eye contact with contaminated water. These include trachoma, scabies, and flea-, lice- and tick-borne diseases. The diarrhoeas are the most important water-washed diseases in tropical areas. They are responsible for the vast morbidity in people of all ages. The diarrhoeal diseases are responsible for the majority of economic losses ascribed to water-related diseases in many countries. They are brought on by a range of infectious agents but fall to a low incidence where the hygiene is good.

WATER-BASED DISEASES: caused by parasitical (worm) infections. The parasites are found in intermediate organisms living in water and include legionellosis, dracunculiasis (guinea worm), disease of Weil, schistosomiasis and other helminths infections. Several diseases are due to flukes or trematodes, whose larvae depend on aquatic snails. The eggs pass from excreta to water, and the larvae emerge from the snails and may be ingested directly via domestic water or via ingestion of food, plants or animals that are carrying encysted larvae from the water.

WATER-RELATED DISEASES: caused by insect vectors breeding in water. Diseases include dengue, filariasis, malaria, onchocerciasis, tryponasomiasis and yellow fever. In rural areas people can be bitten at residual waterholes. Infection may be controlled by selective bush clearing near the water point. Mosquitoes can be killed with a suitable insecticide.

Table 1: Transmission categories, diseases and action

Category		Example	Relevant improvement
I	Water-borne	cholera, hepatitis, poliomyelitis, typhoid, amoebic and bacillary dysentery	water quality
II	Water-washed (a) skin and eyes (b) diarrhoeal	trachoma, scabies, flea and tick borne diseases	quantity
III	Water-based (a) penetrating skin (b) ingested	legionellosis, dracunculiasis (guinea worm), schistosomias	protection user
IV	Water-related insect vectors (a) biting near, (b) breeding in	dengue, malaria, yellow fever sleeping sickness trypanosomiasis, onchocerciasis filariasis	running, piped water
V	Poor sanitation	worms e.g. hookworm	excreta disposal

TRANSMISSION PATHS:

a. human-to-human via the environment

b. human-to-human multiplying in the environment

c. human-to-animal-to-human via the environment

d. animal-to-human via the environment

Current knowledge of tropical epidemiology indicates that low-income communities in hot climates suffer from high morbidity due to non-waterborne faecal-oral infections and to the water-washed infections. In the faecal-oral category, diarrhoeal diseases (especially among children) are a major cause of acute morbidity and mortality throughout developing countries, most of the diseases being non-waterborne. In the water-washed category, skin and eye infections are also major causes of morbidity, and like non-waterborne diarrhoea, are reduced by increasing the quantity, availability and reliability of the water supply almost irrespective of its quality. Therefore a general rule can be postulated that all low-income water supply facilities should strive to transport sufficient quantities of water near to or into homes throughout the year.

In over 50 countries (34 in Africa, 13 in Asia, 5 in Latin America and 1 in Europe) the domestic water consumption is below a Basic Water Requirement level of 50 liters per capita per day (l.c.d.), with 26 countries below 20 l.c.d. and 6 even below 10 l.c.d. The Basic Water Requirement is considered to be 5 l.c.d. for drinking (pure minimum to sustain life), 15 for bathing, 10 for cooking and 20 for sanitation.

Conventional engineering wisdom recommends treatment of all drinking water, except when using high-quality groundwater sources. Universally, drinking water suppliers adhere to drinking water quality standards, mostly the standards issued by

the World Health Organisation. In situations where treatment is not feasible owing to financial or maintenance constraints a choice must be made between supplying water without treatment or abandoning the water supply. Abandoning should be considered only when the rationalised health risks outweigh the rationalised benefits.

Water quality e.g. microbial and chemical quality:

- Microbial causes: viruses, bacteria, parasites, intestinal helminths infections and worms
- Chemical causes: chemical pollution such as fluor, arsenic, nitrates, (heavy) metals, pesticides

The magnitude of the health burden from the most prominent water-related diseases is monitored and regularly reported on by the World Health Organisation. Over 1996 the burden was reported as found in the table below (Table 2).

Table 2: The incidence of water-related diseases over 1996 (source WHO)

Disease	**Morbidity (episodes per year, or as stated)**	**Mortality (deaths per year)**	**Relationship of disease to water supply and sanitation**
Diarrhoeal diseases	1×10^9	3.3×10^6	Highly related to unsanitary excreta disposal, poor personal and domestic hygiene, unsafe drinking water
Infection with intestinal Helminths	1.5×10^{9}*	100,000	Highly related to unsanitary excreta disposal, poor personal and domestic hygiene
Schistosomiasis	0.2×10^{9}*	200,000	Highly related to unsanitary excreta disposal and absence of nearby sources of safe water
Dracunculiasis (Guinea worm)	100,000		Highly related to unsafe drinking water
Trachoma	0.15×10^{9}**		Highly related to not washing the face, often due to absence of nearby sources of safe water
Malaria	0.4×10^9	1.5×10^6	Related to poor water management, water storage, operation of facilities at water points and drainage

Disease	Morbidity (episodes per year, or as stated)	Mortality (deaths per year)	Relationship of disease to water supply and sanitation
Dengue fever	$1{,}75 \times 10^6$	20,000	Related to poor solid waste management, water storage, operation of facilities at points and drainage
Poliomyelitis	114,000		Related to unsanitary excreta disposal, poor personal and domestic hygiene, unsafe drinking water
Trypanosomiasis	275,000	130,000	Related to the absence of nearby sources of safe water
Bancroftian filariasis	72.8×10^6		Related to poor water management, water storage, operation of facilities at water points and drainage
Onchocerciasis	17.7×10^6	40,000	Related to poor water management in large-scale projects

* *people currently infected*
** *excluding Sudan*

Actions

The knowledge about the transition routes in combination with information from epidemiological research about the enormous public health burden caused by water and sanitation related diseases provided a huge challenge for public health engineers to take remedial action. A challenge which led to the Interna-tional Drinking Water Supply and Sanitation Decade (1980-1990), which targeted at adequate water supply and sanitation provision for all by 1990. But, despite the efforts in service provision and construction during the Decade, rapid population growth and lagging investments together with failing operation and maintenance of facilities have left more people without access to basic water supply and sanitation facilities today than in 1990. Presently an estimated quarter of humanity does not avail of access to proper and safe water supply and sanitation. The Decade also learnt that provision of water supply and sanitation facilities is not enough to reduce water and sanitation related morbidity and/or mortality.

Reducing the public health burden of water and sanitation related diseases by breaking the transmission routes of the disease causing agents can be seen as consisting of three interactive elements, that is availability of safe and adequate facilities, their proper use for public health benefits and their upkeep.

The first element is the availability of safe drinking water facilities and hygienic means for excreta disposal. Concretely, this means 20 to 40 litres of water per person per day located within a reasonable distance from the household. Safe water implies the protection of water sources as well as proper transport to and storage in the home. It also means clean facilities with good drainage for bathing, and for washing clothes and kitchen utensils. Sanitary disposal of excreta comprises the isolation and control of faeces from both adults and children so that they do not come into contact with water sources, food or people. A second element is the proper use of the water and sanitation facilities by the users at the household level to obtain the health benefits inherent in them. This means knowing how to protect and store water safely, how to maintain personal and domestic cleanliness, how to maintain sewage disposal facilities and how to avoid or minimise unsanitary environmental conditions. To break the transmission chain of faecal-related diseases, good standards of personal and domestic hygiene, which begin with handwashing after defecation, are essential. Knowledge transfer and personal as well as public attitudes and practises attaching importance and dignity to private and public hygiene are the key factors.

The third of the interactive elements is the public support to improve public health. The public importance attached to public health is expressed by government policies and their institutional as well as financial support for sector development and upkeep. It is even more so expressed by and dependent on community-based efforts, whether in a small village or a large metropolis, in identifying and meeting people's needs.

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2 WATERBORNE DISEASES

WATERBORNE DISEASES ARE CAUSED BY THE INGESTION OF WATER CONTAMINATED BY HUMAN OR ANIMAL FAECES OR URINE CONTAINING PATHOGENIC MICRO-ORGANISMS

EXAMPLES ARE:

CHOLERA, HEPATITIS, POLIOMYELITIS, TYPHOID, AMOEBIC AND BACILLARY DYSENTERY AND OTHER DIARRHOEAL DISEASES

Amebiasis

Entamoeba histolytica is one of the agents causing such amoebic infections as amoebic dysentery. This parasite, found in dysenteric stools of a patient in St. Petersburg, was first described by Lösch in 1875, although he failed to recognise the causal relationship between amoeba and acute colitis. It took many years and experiments to reveal the difference between a slightly larger, morphologically different and harmless commensal, Entamoeba coli and Entamoeba histolytica as being the cause of amoebic dysentery. Recently, Entamoeba histolytica has been differentiated from the morphologically identical non-pathogenic Entamoeba dispar.

Facts, trends and effects

Facts and trends: *Entamoeba histolytica* is an ubiquitous organism. It is more prevalent in the tropics and subtropics than in cooler climates. However, in unhygienic communities found in temperate and subarctic areas, the incidence is, at times, as high as in the tropics.
It is estimated that 10% of the world's population is infected with *Entamoeba histolytica* or *Entamoeba dispar*; this has resulted in about 50 million cases of invasive amebiasis and up to 100,000 deaths. Amebiasis is in many countries a not reported disease. For the WHO it is a Class 3C disease, only reported collectively on a weekly, monthly or sometimes annual basis in recognised endemic areas.

Effects: *Entamoeba histolytica* is named for its lytic effect on tissue. It may act commensally or invade the tissue, giving rise to intestinal or extra-intestinal disease. Most infections are asymptomatic but may become clinically important under certain circumstances: Symptomatic invasive infection develops in about 10% of the asymptomatic individuals.
Intestinal disease may vary from mild abdominal discomfort to fulminating dysentery with fever, chills and bloody diarrhoea. Spreading of the disease to other organs via the bloodstream may occur, producing an abscess in the liver or, less commonly, in the lung or brain. Amebiasis may mimic other diseases like bacterial dysentery or carcinoma.
Case fatality is low in accurately diagnosed and properly treated patients. *Entamoeba dispar* is not reported to cause symptomatic invasive disease.

Causes and pathways

Causes: *Entamoeba histolytica* is a one-celled micro-organism and exists in two forms: the active, growing and potential invasive trofozoite and the surviving state, the cyst. The cysts are in the infective stage.
The life cycle of *E. histolytica* is simple: the infective cyst passes after ingestion through the stomach into the small intestine. There the amoeba emerges from the cyst. In the

lumen of the colon the cysts start to form under conditions which are as yet unknown. The cysts are excreted in the faeces and are infectious at once.

PATHWAYS: Transmission occurs mainly by ingestion of faecally contaminated food or water containing amoebic cysts. The cysts are relatively chlorine resistant. Transmission also occurs in direct human-to-human contact or sexually by oral-anal contact. Asymptomatic cyst passers form the main reservoir, which may continue to pass cysts for years. Acute amoebic dysentery patients do not excrete cysts and therefore only pose a limited danger.
Cysts remain viable in damp soil up to 8 days and in water at 4°C for about 3 months. These figures will depend on bacterial contamination and temperature: the greater the number of bacteria and the higher the temperature, the shorter the period of survival. Cysts are killed by temperatures above 50°C, irradiation and desiccation.
Cysts can be transmitted via flies and cockroaches, the cysts surviving in a fly's excreta for as long as 48 hours.

ACTIONS

- adequate, safe supplies of water for drinking, household purposes and bathing by filtration, sedimentation or sterilisation employing iodinisation;
- disposing human faeces in a hygienic manner;
- protecting food supplies from contamination caused by use of human excreta as fertiliser and by infected food handlers, filth flies or cockroaches;
- thorough washing with potable water and keeping fruit/vegetables dry; disinfectant dips for fruit and vegetables are of unproved value in preventing transmission of *E. histolytica;*
- preventing direct human-to-human transmission in institutionalised groups, as in mental hospitals and prisons;
- treatment of infected persons: both symptomatic and asymptomatic.

There is no vaccine available for *E. histolytica.* Chemoprophylaxis is not recommended because of the risk of drug intoxication and induction of drug resistance.

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Cholera

Cholera is an acute watery diarrhoea caused by the bacillus Vibrio cholerae, originating from the Ganges delta in India and now endemic the world over. It has been responsible for eight successive pandemics; the first one occurring in 1817 and leaving millions dead. The worldwide epidemics have resulted from mutations of the organisms coupled with global travel and mobility. In 1849 Snow demonstrated the importance of infected water in the spread of cholera in London using his famous Broad Street hand pump. The Cholera vibrio (a gram-negative, comma-shaped facultative anaerobic bacillus) was discovered in 1883 by Koch in Egypt.
During the first decades of the 20th century cholera became known as "the great sanitary reformer" in Europe and North America, boosting as no other the large-scale construction of water supply and sanitation systems.

Facts, trends and effects

Facts: Cholera was known as a killer disease from the times of Alexander the Great. Between 1909-1948, 788,000 deaths due to cholera were reported in India alone. Between 1991 and 1998, 700,000 incidences of cholera were recorded in Peru, with some 4600 deaths as a result.
Data from WHO (WHOISIS) on the incidence of water-borne diseases, one of which is cholera, report one billion episodes (morbidity) per year and an estimated 3.3 million deaths per year. Cholera outbreaks are reported almost weekly, for example, in rural and urban areas in India, Bangladesh, Kenya, Malawi, Afghanistan and China, but also in Peru and Colombia.
Cholera seems to be seasonal, with a major peak from September to January and second, smaller, peak from March to May.
Cholera has occurred in almost all countries except those in the northernmost and southernmost parts of the globe.

Trends: Currently, the world is experiencing the eighth cholera pandemic. As the number of clinical cases of cholera El Tor 01 decline, outbreaks of the 0139 type are occurring the world over. The classical biotype had caused the 5th and 6th pandemics and the 7th pandemic by the El Tor 01 biotype.
The present 8th pandemic has been caused by El Tor 0139. Thus, of the four pandemics for which a cause is known, three came through a different organism, suggesting that cholera has been generating mutations in response to changing conditions and pressures.

Effects: Cholera sets in as painless diarrhoea with watery stools, faecal in nature, issuing from the patient in endless streams. The stools quickly lose their faecal character, becoming colourless, or rather like thin rice or barley water, and are soon accompanied by vomiting. Death occurs within hours or a few days. In endemic areas, cholera is mainly problematic for children under 10.

Mortality rates in unprepared communities may be as high as 50%. Where the response is well organised, with an established diarrhoeal disease control programme, this rate can be reduced to less than 1%.

Causes and pathways

Causes: The *Vibrio cholera* is a very minute organism measuring 1.5 to 2 µm. Three cholera vibrios have been identified, the classical vibrio, the El Tor 01 vibrio and the haemolytic El Tor vibrio. Now spread all over the world, El Tor is the harder strain, having replaced the classical cholera vibrio. More recently, the 0139 strain has emerged in Asia. As yet, this strain has not reached Africa, but is expected to do so soon.
Cholera originates in the Ganges Delta in India and Bangladesh, where it is endemic, with the blue-green algae appearing to act as a reservoir for continuous transmission. Other possible continuous reservoirs e.g. animals, humans and the environment, do not seem likely. Factors common in all endemic areas are proximity to rivers, a high population density, low-lying lands and a high absolute humidity. Global warming may enhance endemic areas.

Pathways: Transmission is highly related to unhygienic disposal of excreta, poor personal and domestic hygiene, and use of unsafe drinking water e.g. open wells or rivers (see Figure 1). Transmission of cholera over longer distances (hundreds of kilometres) has been linked to infected traders, bodies of deceased cholera victims (Malawi) and transportation of infected foodstuffs.
Cholera is a disease found where conditions are unsanitary; it can be spread by humans and travels just as quickly. The increasing mobility offered by buses, trains and aeroplanes contributes to the increasing mobility of cholera in an infected community and beyond.

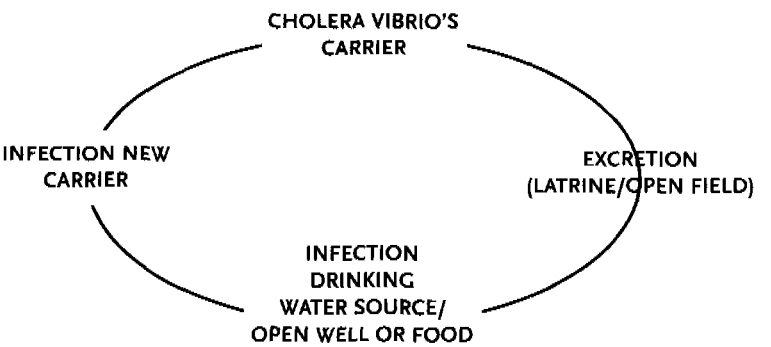

Figure 1: Transmission path of Cholera vibrio

Actions

Information, communication and research

Global level: WHO has designed fact sheets on diseases like cholera and the weekly bulletin of the IRC (Collaborative Council) reports on cholera outbreaks worldwide. The London School of Hygiene and several institutes in developing countries, e.g. the Bangladesh Diarrhoea Research Institute, have research groups working on cholera.

National level: Ministries of Health compile data on cholera outbreaks from hospitals and clinics to monitor and control. Ministries of Health train and support local clinics and their staff during outbreaks.

Local level: Local health staff are charged with hygiene and health education, and control and surveillance for reporting suspected and confirmed cases (age, geographical location/address, measure of hospitalisation and outcome).

Immunity, prevention and treatment: Cholera can only be reliably prevented by ensuring that all populations have access to and make use of adequate sewage disposal systems and safe drinking water.
When cholera appears in a community it is essential to ensure hygienic disposal of human faeces, an adequate supply of drinking water and hygienic handling of food. Travellers to endemic areas are given the advice: "Boil it, cook it, peel it or forget it". Routine treatments with antibiotics and/or restriction of travel and trade have no effect.

Vaccination with two doses of parenterally administered "killer" vaccine will confer only partial protection (50% or less) and that will be limited (3-6 months). Vaccination reduces the incidence of symptoms but not the severity.
In 1973 the World Health Assembly deleted the requirement for presentation of a cholera vaccination certificate from the International Health Regulations. Today, no country requires proof of a cholera vaccination as a condition for entry.
Treatment for cholera is normally done through oral (80-90% of patients) or intravenous (only severely dehydrated patients) administration of salt solutions. Very severe cases can be treated with tetracycline.

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HEPATITIS

HEPATITIS IS DERIVED FROM THE GREEK WORD 'HEPAR' WHICH MEANS 'LIVER' AND THE SUFFIX, '-ITIS', WHICH DENOTES 'INFLAMMATION'. THE TERM 'HEPATITIS VIRUSES' REFERS TO A DIVERSE GROUP OF VIRUSES ALL HAVING THE HUMAN LIVER AS PRIMARY TARGET OF REPLICATION. THESE VIRUSES CAUSE HEPATITIS, CHARACTERIZED BY LIVER CELL DAMAGE AND JAUNDICE. CLINICAL SYMPTOMS CAUSED BY HEPATITIS VIRUSES ARE VERY SIMILAR AND SOME OF THE VIRUSES ONLY EMERGED ON LARGE SCALE IN RECENT YEARS. THEREFORE, DISTINCTION OF UNTIL NOW 7 DIFFERENT AETIOLOGICAL AGENTS WAS ACCOMPLISHED ONLY IN THE 1960'S. HEPATITIS VIRUSES ARE DIVIDED INTO THE PARENTERALLY TRANSMITTED OR BLOODBORNE HEPATITIS VIRUSES AND THE ENTERIC HEPATITIS VIRUSES. THE LATTER GROUP INCLUDES THE HEPATITIS A, E AND F VIRUSES THAT HAVE BEEN REGULARLY ASSOCIATED WITH WATERBORNE OUTBREAKS OF DISEASE.

FACTS AND TRENDS

FACTS: The first two hepatitis viruses distinguished were simply designated as A (HAV) and B. With every newly discovered hepatitis virus, another letter was added. Letter 'G' has already been reached and there are indications of more hepatitis viruses. Taxonomically, all hepatitis viruses belong to different virus families based on their biophysical and biochemical properties. The waterborne hepatitis viruses A and E have been classified as Picornaviruses and Caliciviruses, respectively, though currently, the discussion is whether HEV should be classified as Togaviruses. Hepatitis F viruses are as yet unclassified.

The replication of hepatitis viruses may result in mass destruction of liver cells. Consequences include failure of the liver to fulfill basic functions, such as the removal of bilirubin from the circulatory blood system. Bilirubin is released from red blood cells during the ongoing replacement of these cells by new ones.

The colour of bilirubin is yellow to green. Therefore, accumulation in the blood stream results in excretion through the kidneys (dark urine), the digestive tract (dark stool) and the peripheral blood stream, the latter giving rise to yellowing of the eyes and hand palms. This symptomatic condition of the accumulated bilirubin is known as jaundice.
Another typical consequence of massive liver cell damage is release of liver enzymes into the blood stream. These enzymes include alanine amino transferase (ALT) and aspartate amino transferase (AST), the serum levels of which used to diagnose hepatitis.

Hepatitis A has an incubation period of 2 to 6 weeks, which is somewhat shorter than that of hepatitis E (4 to 8 weeks). HAV infection tends to be mild or without clinical symptoms in children. In contrast, HEV tends to occur more often occur in young adults, many of whom are already immune to HAV. Typical clinical symptoms of HAV infection, such as substantial liver damage, are predominantly seen in adults who rarely develop complications. In contrast, HEV-infected individuals tend to display more prominent cholestasis; progression to acute fulminating hepatitis is commoner than in any other viral hepatitis, at 1 to 2% of cases. This may be even higher in pregnant women for whom fatality rates as high as 20 to 40% have been recorded. Although the general level of mortality is less than 1%, hepatitis may be quite severe and incapacitating. Patients may be confined to bed for up to six weeks or more, feeling ill with apathy to foods, which depend heavily on liver functions for digestion. The severity of illness and mortality may be associated with underlying conditions such as immunodeficiencies, malnutrition and the general state of health.

Trends: HAV typically occurs in all parts of the world, and there is no indication of geo-graphical preferences, other than those related to levels of hygiene and sanitation. In several parts of the world, the virus continues to cause substantial morbidity.
In developing countries most individuals contract infections during early childhood. Fortunately, there seems to be only one antigenic type that provides lifelong immunity. In adult populations of developing countries and communities, immunity to HAV may exceed 95%, in contrast to industrialised communities, where levels of immunity may be less than 50%. Therefore, epidemic HAV outbreaks are relatively common in most industrialised countries. In addition, low levels of endemic HAV transmission prevail. Although HEV is also primarily associated with poor hygiene and sanitation, the epidemiology of HEV also seems to include a geographic element. Clinical infections and outbreaks of hepatitis E have been recorded predominantly in certain parts of the world, such as India, its surrounding countries and parts of Central Asia, China and Africa, but also Mexico. The disease is endemic in many of these countries and is the most common cause of acute hepatitis in adults in parts of India, Asia and Africa.

Causes and pathways

Hepatitis A is very infectious and can spread in a variety of ways. Direct person-to-person transmission by the faecal-oral route is probably the commonest, especially in children. This route of transmission is the most likely cause of most of the school-based outbreaks described in the UK and elsewhere. Foodborne outbreaks caused by an infected food handler or raw shellfish harvested from polluted waters as the source of infection are also common. Hepatitis A is most common in tropical and subtropical countries just as hepatitis E, though the latter is less common. Hepatitis E infection appears to be less infectious since secondary cases are much less prevalent than reported for hepatitis A. HEV appears to be exclusively spread by drinking water, and outbreaks of this pathogen demonstrate many of the problems associated with providing safe drinking water supplies in tropical countries. The limited information available on HFV suggests that it is transmitted by water and food, as are HAV and HEV.

Because of its typical association with epidemics caused by contaminated water and food worldwide, HAV was initially known as epidemic or infectious hepatitis. Small and large outbreaks throughout the world, numbering 3 to 300,000 cases have been associated with drinking contaminated well water, bathing in contaminated spa pools, working at a wastewater treatment plant and eating clams. Large outbreaks of hepatitis E have also been reported. Consumption of drinking water contaminated with sewage affected hundreds to thousands of people in India, Nepal, Pakistan, China, Africa and Asia. Clinical cases of hepatitis E in regions, such as Central Europe, Great Britain, North and South America, Australasia, Japan and South Africa seem limited to imported cases. However, seroprevalence studies now reveal that the virus is actually present in many of these countries, and 2 to 10% of inhabitants may have antibodies, which confirms the exposure to HEV. Reasons for a relatively low incidence of clinical cases and outbreaks in certain parts of the world, despite the presence of the virus, are not yet fully understood. In contrast to HEV, HFV is associated with sporadic cases in certain geographical areas, such as the UK, Italy, the USA and India, and not with out-breaks or epidemics. In some of these areas cases of HAV and HEV are rare or virtually unknown.

Actions

The production of water and food, which is free of enteric hepatitis viruses, at least within acceptable limits, is feasible. However, due to factors such as the presence of available expertise and facilities, and the varying incidence of viruses in sources and the exceptional resistance of at least HAV complicates effective virus removal. Indeed, hepatitis A viruses have been shown to survive treatment and disinfection processes even when sanitary guidelines were met. Recently, HAV vaccines were introduced which may contribute to the control of this virus. There are no vaccines available for HEV and HFV, and there is no indication that vaccines for these viruses are forthcoming. Controlling the spread of enteric hepatitis viruses is particularly important because treatment for the diseases is not yet available as yet.

Additional reading

Fields Virology, Third edition, edited by Fields, Knipe and Howley et al., Lippincott - Raven Publishers, Philadelphia, 1996

Internetsite:
www.who/inf-fs/en/fact112.html

Poliomyelitis

For centuries, poliomyelitis has been a well-known and much feared disease. The oldest reference is an Egyptian engraving from 1500 years BC, showing a crippled priest with a shrunken and deformed leg supported by crutches. The most prominent symptom of poliomyelitis anterior acuta is acute flaccid paralytis (AFP), a consequence of the destruction of motor neuron cells in the spinal cord. In 1908, Karl Landsteiner discovered the causative agent of this disease to be a virus, appropriately called the "poliovirus".

Facts and trends

Facts: Polioviruses, together with echo- and coxsackie-viruses, belong to the *Enterovirus* genus of the *Picornaviridae* family. As the name *Picornaviridae* suggests, polioviruses are small RNA viruses of about 30 nanometres in size that are classified into three serologically distinct types. Although the poliovirus-induced infection does not produce visible symptoms in 90% of the persons affected, clinical manifestations can be quite severe; these include paralysis, meningitis and fever. The incubation period of poliomyelitis is normally 9-12 days (range 5-35 days). Abortive polio presents as an influenza-like illness, a tonsillitis or a gastroenteritis, which can last for just a few hours to 4-5 days at most before recovery. Patients who go on to develop the paralytic disease also usually experience a similar illness.
Unfortunately, after remaining symptom-free for 2-5 days, these patients develop features of meningitis: fever, headache, neck stiffness and vomiting. Muscle pain is also an early feature. After about 1-2 days of suffering from meningitis, patients start to develop muscle weakness and eventually paralysis. Spinal poliomyelitis ranges from weakness of part of a single muscle to complete quadriplegia, whereas bulbar poliomyelitis, affecting the cranial nerves, may lead to dysphlagia, dysarthria or dyspnoea. The illness is more severe in the very young, pregnant, immunosuppressed and otherwise debilitated.

Trends: The reported incidence of polio cases has declined by 82% from the 31,251 cases reported in 1988, since the eradication target was set at 6179 cases for 1995. Of a total of 214 countries, 57 have reported more than one case whereas 150 countries have reported zero cases of polio. The greatest decline, 51%, as compared with 700 cases reported in 1994, was found in the western Pacific region followed by 33% and 27% declines in the Southeast Asia and eastern Mediterranean regions, respectively. The American region, covering the whole of the Western hemisphere, was the first WHO region to eradicate polio. The last case was in August 1991. With the exception of an "imported" case from the Netherlands to Canada in 1993, no wild virus transmission has been detected in the Western hemisphere since.
The stool specimens from tests on 82% of the 1928 Acute Flaccid Paralys is cases detected in this region in 1995 all showed negative for the wild poliovirus. Because epidemiological surveillance is incomplete in many polio-endemic countries, the WHO estimated that approximately 80,000 cases of paralytic polio occurred in 1995.

Causes and pathways

Infection usually follows the ingestion of fecally contaminated water or food. The infective dose is about 10^5 to 10^6 infectious particles. The initial site for replication is the submucosal tissue of the pharynx or distal small intestine. Large amounts of the virus may be excreted in the faeces and oropharyngeal secretions, which will directly or indirectly lead to further spread of the virus. One waterborne outbreak of poliomyelitis involved over 1000 cases of paralytic poliomyelitis in Taiwan in 1982. Logistic regression analysis showed the use of water from non-municipal water supply and inadequate vaccination to be independent risk factors for acquiring the disease.

Actions

To eradicate the disease, the naturally occurring wild poliovirus, which can survive in the environment for about 2 to 3 months must be destroyed. Since humans are the only host of the virus, mass immunisation is the only tool for breaking virus transmission. Two types of vaccines are used, inactivated poliovirus vaccine (IPV) and oral poliovirus vaccine (OPV). The main advantage of IPV is the inability of the virus to replicate and therefore to revert to a pathogenic variant. This advantage at the same time holds a disadvantage since IPV will not booster other individuals in the population since mucosal replication can not occur as is the case with OPV. Global certification of polio eradication requires a three-year control period after the last case of polio worldwide has been identified.

Additional reading

Fields Virology, Third edition, edited by Fields, Knipe and Howley et al., Lippincott - Raven Publishers, Philadelphia, 1996

Internetsite:
www.who/inf-fs/en/fact112.html

ROTAVIRUS INFECTION

DESPITE THE MAGNITUDE OF THE PROBLEM OF INFANTILE DIARRHOEAL DISEASES, THE SEARCH FOR IMPORTANT ETIOLOGIC AGENTS OF DIARRHOEA - BACTERIAL, VIRAL OR PARASITIC - WAS UNREWARDING UNTIL THE 1970'S. THIS WAS ESPECIALLY DISAPPOINTING FOR VIROLOGISTS BECAUSE WITH THE ADVENT OF TISSUE CULTURE TECHNOLOGY, SCORES OF ENTERIC VIRUSES HAD ALREADY BEEN DISCOVERED IN THE 1950'S AND 1960'S, BUT NONE WAS FOUND TO BE AN IMPORTANT ETIOLOGIC AGENT OF INFECTIOUS DIARRHOEA. THIS FRUSTRATION ENDED WITH THE DISCOVERY OF THE NORWALK VIRUS ASSOCIATED WITH DIARRHOEA IN OLDER CHILDREN AND ADULTS, BY KAPIKIAN ET AL. IN 1972. IN 1973 ROTAVIRUSES WERE ALSO FOUND TO BE ASSOCIATED WITH SEVERE DIARRHOEA IN INFANTS AND YOUNG CHILDREN BY BISHOP ET AL. USING THIN-SECTION ELECTRON MICROSCOPY. THESE 70-NM PARTICLES OWE THEIR NAME TO THEIR WHEEL-LIKE APPEARANCE WITH SHORT SPOKES AND A SMOOTH OUTER RIM (ROTA IS LATIN FOR 'WHEEL'). IN A RELATIVELY SHORT PERIOD, INVESTIGATORS FROM MANY COUNTRIES REPORTED THE DETECTION OF ROTAVIRUSES IN THE FEACES OF PEDIATRIC PATIENTS SUFFERING FROM A DIARRHOEAL ILLNESS. IT WAS THEREFORE SOON ESTABLISHED THAT ROTAVIRUSES WERE THE LONG-SOUGHT MAJOR VIRAL ETIOLOGIC AGENTS OF SEVERE DIARRHOEA IN INFANTS AND YOUNG CHILDREN IN BOTH INDUSTRIALIZED AND DEVELOPING COUNTRIES, CONSISTENTLY OUTRANKING OTHER KNOWN ETIOLOGIC AGENTS OF SEVERE DIARRHOEA IN IMPORTANCE.

FACTS AND TRENDS

FACTS: Rotaviruses belong to the *Reoviridae*, which owe their name to the respiratory and enteric infections they can cause (reo = Respiratory, Enteric, Orphan). Three human pathogenic rotavirus species, rotavirus types A, B and C, can be distinguished in the *Reoviridae*. Rotavirus particles are nearly spherical, with a 16 to 21 kbp linear, double-stranded RNA genome. Rotavirus replication takes place in the villus epithelial cells of the small intestine and causes a loss of absorptive cells. This may in part explain the cause of reduced fluid absorption associated with diarrhoea.
As stated above, rotavirus most commonly affects infants and young children. The virus has an incubation period of about 24 hours. The illness starts abruptly, with fever, vomiting and diarrhoea, which normally lasts 24 to 48 hours, although diarrhoea can last for up to five days.

TRENDS: Viral gastroenteritis is a very important cause of diarrhoeal disease, with 3 to 5 billion cases each year and up to 10 million deaths. A wide variety of viruses cause gastroenteritis. Viral gastroenteritis appears to be on the increase, at least in the UK. While some of this increase may be due to better diagnosis, a large portion of the reported increase appears to be realistic. Rotaviruses have been detected around the world wherever they have been sought. Data from epidemiological studies in developing and developed countries have accumulated on rotaviruses as the major etiological agents of severe infantile diarrhoea in every country where

this disease has been studied using appropriate techniques. Although rotavirus diarrhoea occurs with high frequency in the developed countries, mortality is low. The burden of rotavirus diarrhoea in the USA in 1 to 4 year-olds is estimated to include over 1 million cases of severe diarrhoea and caused up to 150 deaths (0.015%). The burden of rotavirus diarrhoeal disease in infants and young children under five years of age in developing countries has been estimated at over 125 million cases of which over 18 million were considered moderately severe to severe. In addition, it was estimated that 873,000 infants and young children 1 to 4 years of age die from rotavirus diarrhoeal illness each year (0.70%). Rotaviruses display a very marked seasonal prevalence, reaching epidemic peaks in temperate countries such as the Netherlands during the coldest months of the year.

Causes and pathways

Oral administration of rotavirus positive stool specimens induces diarrhoeal illness in volunteers, confirming the suggestion that rotaviruses are transmitted through the oral-fecal route. In 1992, an outbreak of severe waterborne gastroenteritis due to contamination of the main water supply occurred in a small Brazilian town, affecting 132 adults and children. The outbreak of the diarrhoeal disease had an abrupt onset and afflicted all age segments of the population. Group A rotavirus was the only pathogen associated with the epidemic. Severe dehydration was common among the adults and older children, and 35% of all the patients registered were hospitalised to receive parenteral rehydration. In 1988, a mass outbreak of diarrhoea consisting of 296 cases, occurred in the Fukuoka Prefecture. Fecal samples from afflicted patients were cultured for bacteria, but all samples were found negative. The outbreak appeared to be caused by group C rotaviruses, based on the pattern of electrophoretic migration of RNA genome segments seen when apply-ing polyacrylamide gel electrophoresis and immuno electron microscopy. In the winter of 1982, two very large outbreaks of waterborne rotaviral gastroenteritis affected more than 12,000 adults in coal mining areas of China. Both epidemics showed an explosive onset. The main water supply was found to be heavily polluted with feaces. The outbreak quickly subsided after control of the water hygiene.

Actions

Rotaviruses are fairly resistant to many disinfectants, except chlorine, to which they are relatively sensitive. Therefore disinfection by chlorination of drinking water can be effective in limiting rotavirus infection. Furthermore, disinfection with chlorine and careful handwashing constitute important measures for containing rotavirus infection, especially in a hospital or institutional setting.

Additional reading

Fields Virology, Third edition, edited by Fields, Knipe and Howley et al., Lippincott - Raven Publishers, Philadelphia, 1996

Typhus (Salmonella)

Typhoid fever is a disease of great historical interest to the water industry, considering that in the early part of the 20th century it was the commonest known cause of waterborne outbreaks in both the UK and the USA. Much of the water legislation and practice dating back to the early and mid-20th century was aimed at preventing this disease. Although now uncommon in the western world, it is still a major problem in many less prosperous countries. When outbreaks occur, they can still cause many deaths in affected individuals. Salmonella owes its name to the American veterinary surgeon, Daniel E. Salmon, who first discovered the bacteria in 1885.

Facts and trends

Facts: The genus of gram-negative *Salmonella* bacteria consists of two species: *Salmonella enterica* and *Salmonella bongori*. Of these two species, *Salmonella enterica* is subdivided into six subspecies, *Salmonella enterica* subsp. These are: *enterica, salamae, arizonae, diarizonae, houtenae* and *indica*. A total of 2213 different *salmonella* strains have been identified. Human pathogenic species include S. *arizonae*, S. *typhi* and several S. *enteritidis* serotypes. S. *arizonae* can cause human salmonellosis, which is usually characterized by acute onset of fever, abdominal pain, diarrhoea, nausea and sometimes vomiting. In some cases, particularly in the very young and the elderly, dehydration can become severe, and lifethreatening. S. *enteritidis* can cause gastroenteritis. S. *enteritidis* serotype *paratyphi* A can cause paratyphoid fever, S. *typhi*, typhoid fever and S. *enteritidis* serotype *typhimurium*, both food poisoning and paratyphoid fever.

Trends: Over the years, antibiotic-resistant strains have developed that are difficult to control and there is a body of evidence in the scientific literature suggesting the possibility that some of these strains may have emerged due to use of antibiotics in intensive animal husbandry. Recent years have seen a dramatic rise in cases of human salmonellosis, both in terms of incidence and severity. Compared to 1980, some countries in Europe have witnessed a staggering 20-fold increase in incidence in the last 10 - 15 years. Where the epidemic has been further studied, the majority of cases has been caused by strains or, more correctly, serotypes of *Salmonellosis enteritidis* and *Salmonellosis typhimurium*. The bad news is that since the beginning of the 1990's, strains of S. *typhimurium*, which are resistant to a range of antibiotics, have emerged and are threatening to become a serious public health problem. This is part of a general trend. The incidence of bacterial resistance has increased at an alarming pace in recent years and is expected to continue rising at a similar or even greater rate in the future as antimicrobial agents or antibiotics lose their effectiveness. The disease-reporting system in England and Wales has revealed a ten-fold increase in the number of human cases of multi-drug resistant *Salmonella typhimurium* DT 104 in the period 1990-1996, rising from 300 to 3500 cases per

year. This specific strain is second only to *Salmonella enteriditis*, and was the most common salmonella infection in humans in England and Wales in 1995. More than 55% of cases of *Salmonella typhimurium* in humans were caused by the multi-drug resistant, DT 104, which has developed a stable resistance to some of the most common antibiotics: ampicillin, chloramphenicol, streptomycin, sulfonamides and tetracycline. Since 1994, an increasing number of isolates with additional resistance to trimethoprim, and a few with additional resistance to ciprofloxacin, have been reported. Infection with multi-drug resistant *Salmonella typhimurium* DT 104 has been associated with hospitalization rates twice that of other zoonotic food-borne salmonella infections and with case-fatality rates ten times as high. An increase in the overall number and percentage of multi-drug resistant *Salmonella typhimurium* DT 104 is also reported from other European countries. In Germany, it accounted for up to 10% in more than 10,000 salmonella samples from human sources examined in 1995; and 18% of those examined in 1996. Almost all DT 104 isolates were multi-drug resistant, with the same resistance pattern shown as in England and Wales, although resistance to ciprofloxacin has not yet been observed. *Salmonella typhimurium* DT 104 was recently detected in the USA, but little is known about its prevalence and means of transmission. Preliminary evidence suggests that DT 104 may have spread widely in the United States during the past 2 to 3 years, which may result in a marked increase in human illness in the near future. Over the past three decades, practically all countries in Europe have reported a sharp rise in the incidence of salmonellosis. The same pattern could be observed in a number of countries in the Middle East and Southeast Asia.

Causes and pathways

The primary route of infection for humans is by consumption of contaminated food of animal origin. Unlike *Salmonella enteritidis*, which is mainly associated with poultry and eggs, multi-drug resistant *Salmonella typhimurium* DT 104 can be found in a broad range of foodstuffs. Outbreaks in Great Britain and Northern Ireland have been linked to poultry, a variety of meats and meat products, and unpasteurized milk. In addition to acquiring infection from contaminated food, humans who had contact with infected cattle have also contracted the infection. A small proportion may have become infected through contact with pets such as cats and dogs, who can also be infected with this strain of *Salmonella*. Pets probably acquire the infection like humans, in other words, through consumption of contaminated raw meat, poultry or poultry-derived products.

Actions

Control of multi-drug resistant *Salmonella typhimurium* DT 104 requires a reduction of the infection in animals for slaughter (for food production) and lowering the risk of contamination during all stages of the food production chain. In addition, the avoidance of unnecessary antibiotic use in animals for slaughter should be combined with high-quality animal husbandry, abattoir

practice and hygiene at all stages in the food production chain, from processing plants to kitchens to food service establishments.

Additional reading

Internetsite:
www.who/inf-fs/en/fact112.html

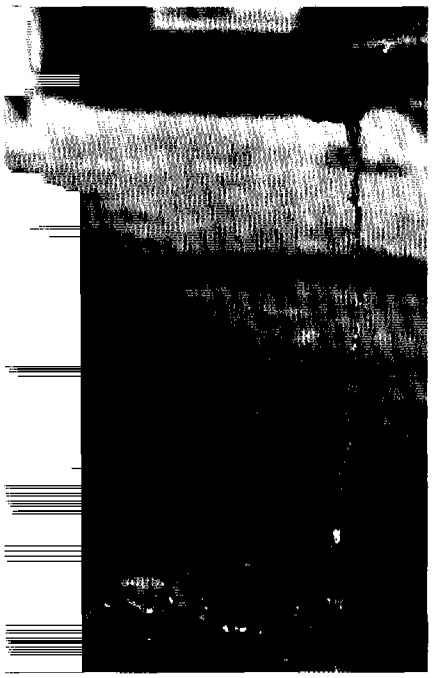
3 WATERWASHED DISEASES

WATERWASHED DISEASES ARE CAUSED BY POOR HYGIENE AND SKIN OR EYE CONTACT WITH CONTAMINATED WATER

EXAMPLES ARE:

TRACHOMA, SCABIES AND FLEA, LICE AND TICK-BORNE DISEASES

Trachoma



Trachoma is one of the oldest infectious diseases known to humankind and means roughness. The agent is Chlamydia trachomatis - a micro-organism resembling both bacteria and viruses. It is responsible, at present, for 15% of the blindness in the world. Worldwide, about 6 million people have been largely irreversibly blinded by trachoma and an estimated 146 million people suffering from the active form of the disease are in need of treatment if blindness is to be prevented.

Facts and trends

Facts: The Chlamydiaceae constitute a family of small coccoid, gram-negative, parasitic micro-organisms that have a unique, obligately intracellular developmental cycle and are incapable of synthesising their own source of energy. They induce their own phagocytosis by host cells, in which they then form intracytoplasmic colonies. The family contains a single genus, *Chlamydia*. *C. trachomatis* serotypes A - C can cause trachoma, which starts with provocation of an inflammatory reaction in the eye followed by formation of follicles in the conjunctiva. After years of repeated infections, the inside of the eyelids may be scarred so severely that the eyelid turns inwards, with eyelashes rubbing on the eyeball. If untreated, this condition will lead to blindness. *C. trachomatis* serotypes D - K can also cause inflammation of the urethra and rectum, which can lead to Hodgkin's disease or ectopic pregnancies. *C. psittaci* strains may cause psittacosis, ornithosis and a variety of diseases in animals.

Trends: Today, the disease is found mainly in poor rural areas of most African countries, some countries in the eastern Mediterranean, and in certain parts of Central and South America. Trachoma is still endemic in several Asian countries, but due to a lack of updated information from some major populations, such as India and China, the extent is not known.

Causes and pathways

Chlamydia trachomatis spreads through contact with eye discharge from the infected person (on towels, handkerchiefs, fingers, etc.) and through transmission by eye-seeking flies.

Actions

Trachoma and related blindness can be prevented by the implementation of the SAFE strategy, standing for Surgery for trichiasis (inward growing eyelashes), Antibiotics, Facial cleanliness and Environmental improvement. This strategy is based on a combination of community-targeted public health interventions.

Recent field trials of a new and apparently effective drug, azithromycin, have been very encouraging. This long-acting antibiotic has been tested in a number of countries. The initial results look very promising: one dose of azithromycin per year could eliminate the blinding propensity of trachoma. Once its efficacy has been proven, the challenge will be to find the means to make this drug available to all those in need.

Additional reading

Internetsite:
www.who/inf-fs/en/fact112.html

4 WATERBASED DISEASES

WATERBASED DISEASES ARE

CAUSED BY PARASITES FOUND IN INTERMEDIATE

ORGANISMS LIVING IN WATER

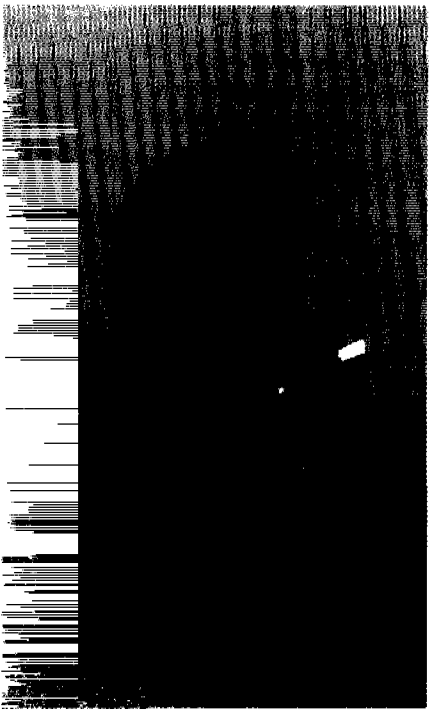
EXAMPLES ARE:

LEGIONELLOSIS,

DRACUNCULIASIS (GUINEA WORM),

DISEASE OF WEIL, SCHISTOSOMIASIS AND

OTHER HELMINTHS INFECTIONS



Dracunculiasis (Guinea worm)

Dracunculiasis, commonly known as the 'Guinea worm', is the only parasitic water-based disease that may be fully eradicated in the near future through the improvement of water sources, in other words, by preventing water infected by Guinea worm carriers from spilling back into water sources. Dracunculiasis was widespread at the beginning of the 20th century, and even just more than a decade ago there were over three million cases of dracunculiasis worldwide. Active foci of the disease are now found only in Africa south of the Sahara and in Yemen. A global campaign is aiming at eradication early in the next century.

Facts, trends and effects

Dracunculiasis is still endemic in 19 countries, of which 17 are African (mainly Sudan, Ghana and Nigeria), however, India and Yemen have also not yet been declared dracunculiasis-free. From an 'estimated' 48 million cases in 1947, the incidence dropped to 3.3 million cases in 1986, less than 80,000 reported cases in 1997 and 50,000 in 1998, of which over 50% were in the Sudan, 20% in Nigeria and 10% in Ghana.

The guinea worm kills few victims but causes its human hosts debilitating pain, usually in the planting season, and so can have far-reaching economic effects if a majority of the population cannot work at that time. In some areas the worm is transmitted chiefly through domestic water supplies. The body of the mature female worm migrates after being ingested to under the skin in the leg of the human host, where it nestles with its genital pore beneath a blister near the ankle. When this blister is immersed in water or when water splashes over it, for example, when drawing water from a well or water hole, it bursts and releases a cloud of *Dracunculiasis* larvae, which are likely to be washed down into the well or pool. The larvae then infect cyclops, tiny crustaceans found in many small bodies of water, and develop inside their new hosts. The cylops, being only 2 mm long, are easily inadvertently consumed by humans drinking water from an infected well. The cyclops are not dangerous in themselves but the worms they contain develop further in the human host, with a female *Dracunculiasis* making its way to the host's legs to form a blister a year later, when a new cycle is started.

Causes and pathways

Dracunculiasis is caused by the parasite *Dracunculiasis medinensis*, the largest of tissue parasites affecting humans. The female has the thickness of a needle and is usually 60 - 100 cm in length (the male is only 2 cm long). It lives in the host's connective tissue and does no harm until it is about to produce its offspring.

At this point it is drawn through the tissue towards the earth (geotropism), mainly through the limbs, eventually emerging from the body (via the feet in 90% of the cases), causing intense pain, a blister and an ulcer. People infected with this worm are sick for several months.

The full grown worm begins to migrate throughout the infected person's body within about one year after ingestion. In endemic countries, the disease typically reappears every year during the planting season, with farmers in particular being infected. There are no drugs to treat the disease.

Actions

In the early 1980's, dracunculiasis was identified as a disease which could be eradicated by simple preventive measures. In 1997, the World Health Assembly adopted a resolution reiterating its request that dracunculiasis be eradicated as soon as possible. The eradication strategy adopted by WHO is based on the approach: interruption of transmission and surveillance, combining a variety of interventions including:

- an integrated community-based surveillance system;
- intensified case-containment measures;
- provision of safe water, including appropriate water supply systems, filtering
- health education;
- monitoring the dracunculiasis situation through the use of geographical information systems (GIS);
- interagency collaboration.

WHO, UNICEF, Global 2000, agencies operating bilaterally, several NGOs and the countries affected contribute to the interruption of transmission, programme monitoring and surveillance processes. Certification of eradication falls directly under the WHO mandate. The Centres for Disease Control and Prevention (CDC) of the US Department of Health and Human Services issues monthly Internet bulletins on the guinea worm eradication campaign results.

Additional reading

Peries H, Cairncross S. Global eradication of guinea worm. Parasitology Today 13 (11): 431-437, 1997

Cairncross S, Feachem R. Environmental Health Engineering in the Tropics, John Wiley & Sons, Chichester, 1983

Manson's Tropical Diseases, 18th (ed.). Bailliere Tindall, London, 1983

Internetsites:
www.who.int/inf-fs/en/fact098.html
www.cdc.gov/ncidoddpd.list_drc.htm

Legionellosis

Legionellae were first detected after a spectacular outbreak of pneumonia in a hotel on the occasion of a meeting of US army veterans in 1976. Legionellae have been identified as an important cause (5-15%) of community- and hospital-acquired pneumonia. Common to natural freshwater sources all over the world, Legionella is often found during random sampling of water systems and services.

Facts and trends

Facts: The *Legionellae* consist of one monophyletic family, the Legionellacae, containing one genus: *Legionella*, with 42 subspecies. The subspecies *L. pneumonophila* is responsible for about 90 percent of the infections caused by *Legionellae*. *L. pneumophila* contains 14 serogroups, but serogroup 1, 4 and 6 account for the overwhelming majority of strains implicated in human infection. *Legionellae* are small, motile, slow-growing bacteria, proliferating at temperatures ranging from 25 to 55°C, with an optimum growth temperature of 42°C. In clean water, *Legionellae* can survive for one year and in aerosols a few hours. A typical feature is the ability to multiply inside of amoebae, which inhabit water or soil. Different amoebal species support the growth of *Legionella*, with some cyanobacteria genera promoting relative rapid growth.

Two kinds of disease have been observed in humans:

- Legionnaire's disease (incubation time of 2 to 10 days) takes the form of severe pneumonia, with a lethality of about 15%. This disease is often accompanied by extrapulmonary manifestations, e.g. renal insufficiency, colonic abscesses and myocarditis. The pathogenity for humans is controlled, to a large extent, by the host's susceptibility. Children and young people are rarely affected, while immunocompromised patients, especially transplant recipients, run a very high risk.
- Pontiac fever is a febrile illness of 2 to 6 days duration (incubation time: 1 to 2 days) without pneumonic symptoms or any other local manifestation. In contrast tolegionnaire's disease, it affects children or healthy adults to the same extent as compromised patients.

There are no specific clinical symptoms which distinguish infections caused by *Legionella* from pneumonias or local infections of other origin. It is commonly accepted that probably more people die of legionnaire's disease than documented due to the fact that not all *Legionella* infections are recognised as such. Based on two prospective studies in two counties in Ohio, USA, it was shown that the vast majority of *Legionella*-caused pneumonia per year is about ten times more than the number reported to the health authorities.

Trends: Since the description of the outbreak in 1976 many similar incidents have been published, although mostly on a smaller scale. Very frequently, hospitals have been involved. Outbreaks or clusters of cases have often been traced to the colonised part of air conditioning plants, while recurrent single cases are associated with contaminated hot water systems.
The overall frequency of *Legionella* in domestic water supplies and plumbing systems may be lower than in hospitals, but investigations in different German cities have shown that apartment blocks may be as heavily contaminated as hospitals, while private homes are perhaps less affected.
A recently documented (1999) major outbreak in the Netherlands involved 242 patients of which 28 succumbed.

Causes and pathways

In natural surroundings only low concentrations of *Legionella* are found, as can be expected from the small replication rates at temperatures below 25°C. The bacteria are introduced from surface water, soil and subsoil into water serving as a source for drinking-water preparation or other purposes. The storage of unpurified water in reservoirs does not bring about reduction in numbers; elevated temperatures during storage are even expected to increase growth. Treatment will affect the concentration, just as with other bacteria. Growth inside filters used for drinking-water preparation is conceivable if microbial colonisation, including amoebae, occurs, but will be controlled by low temperatures. Significant concentrations will develop in situations were temperatures reach and remain at levels above 20°C for prolonged periods and where microbial growth is possible. *Legionellae* are frequently found to colonise man-made water systems at temperatures between 20 and 50°C. The presence of assimilable organic carbon influences the growth of *Legionellae* in biofilms, which can be enhanced by materials found in the plumbing system.
The main mode of transmission is inhalation of *Legionella*-containing aerosols generated by technical devices, e.g. cooling towers of air conditioning systems, warm water installation of buildings or hot whirlpools. Aspiration following ingestion of contaminated water or food has also been incriminated in some cases as the route of infection. The infective dose is supposed to be very small, maybe only a few or single microorganisms.
In the lungs of susceptible persons, the high carbon dioxide and oxygen concentrations, combined with a high body temperature, humidity and the presence of nutrients result in the growth of this organism. After and during growth the microorganism starts producing toxins which destroy the cells of the lungs, leading to the legionnaire's disease.

Actions

It does not seem feasible to attempt to prevent contamination of water supply systems and reservoirs with *Legionellae* for extended periods of time; however, the entry of single bacteria or bacteria-carrying amoebae should always be considered possible. Keeping the water below the preferable 15°C and flowing, or keeping it hot (at least above 55°C) and flowing, will be the best way to prevent growth. Addition of chlorine to 0.2 mg/l will keep drinking waters free of the bacteria. To prevent colonisation of plumbing systems or other water-containing devices, elevated temperatures are recommended (50°C UK; 60°C NL). Even with these measures, it is sometimes not possible to keep warm water systems permanently free of *Legionellae* due to peculiarities in the plumbing system, which either cannot be identified or repaired .
Continuous surveillance and disinfection should be implemented in water supply systems. Transplant patients and other immunocompromised patients should be fed and washed with only sterilised water.

Additional reading

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5 WATERRELATED DISEASES

WATERRELATED DISEASES ARE CAUSED BY INSECT VECTORS WHICH BREED IN WATER

EXAMPLES ARE:

CRYPTOSPORIDIOSIS, DENGUE, GIARDIASIS, MALARIA AND YELLOW FEVER

Cryptosporidiosis

Members of the genus Cryptosporidium are small parasites that infect the digestive and respiratory tract of vertebrates. Several species have been described; infections in humans are almost exclusively caused by Cryptosporidium parvum. The first human cases of cryptosporidiosis were reported in 1976 and cases are still current. C. parvum is recognised as one of the most commonly identified intestinal pathogens worldwide. This species is also frequently found in cattle and causes infections in a wide range of other mammal species.

Facts and trends

Facts: *Cryptosporidium* is shed in faeces by infected hosts in the form of oocysts, the environmental-resistant transmission stage of the parasite. These oocysts are infectious and may remain in the environment for long periods without losing their infectiousness.
Studies on infections with healthy human volunteers show good agreement between the probability of infection and the ingested dose; even the ingestion of a single oocyst may result in an infection.
Watery diarrhoea is the most prominent symptom, with others being nausea, abdominal cramps, weight loss, vomiting and mild fever. In immunocompetent persons the infection is limited by the immune response that eventually clears the host of the parasite. In immunocompressed persons, however, the infections are often persistent and severe. The duration of the infection is generally 7-14 days for the immunocompetent patient, for whom mortality is low. However, in immunodeficient persons, the infection can result in severe dehydration and high mortality. To date, no consistently effective therapeutic agent has been found.
This protozoa can only be detected using specific colouring techniques (acid-fast), which is the reason it was not found earlier.

Trends: The reported prevalence of *Cryptosporidium* in patients with gastroenteritis is 1-4% in Europe and 3-20% in the rest of the world. Peaks in industrialised countries are observed in late summer and spring. Oocysts are ubiquitously present in the aquatic environment. They have been found in most surface waters and are persistent in water at 0.01-100 per litre. Furthermore, they are resistant to disinfectants commonly used in drinking-water treatment. Combining all these facts with the low numbers of oocysts required for infection make these organisms a critical pathogen when considering production of safe drinking water from surface water. Many water-related outbreaks have been reported, most attributed to contaminated drinking water (surface water and groundwater), along with recreational water and swimming pools (recently in the United Kingdom). In a significant number of outbreaks, the water quality complied with the legislation or WHO guidelines for bacteriological parameters *(Escherichia coli)* and turbidity.

Causes and pathways

A major route of exposure is person-to-person transmission, as illustrated by outbreaks in daycare centres and the spread of the infection in households of the children attending. Sexual practices implying oral-anal contact also pose a high risk for exposed persons. Transmission from animals, especially the new-born, to humans can occur. Many infections have derived from contact with infected calves and lambs. Indirect transmission may occur by contamination of water used for recreation or drinking, or by food contamination. Water-related outbreaks have been caused by contamination of source water meant for drinking-water production due to heavy rainfall or melting snow, or to contamination of wells with sewage, inadequate treatment practices, or treatment deficiencies or both. Leakage or crossed connections in the distribution system have also caused outbreaks. The number of people affected by a cryptosporidiosis outbreak via the drinking water ranges from several up to 400,000.

Actions

Preventing transmission of *Cryptosporidium* requires a multiple-barrier approach: protection of watersheds and installation of adequate treatment facilities, coupled with verification by monitoring.
Recognising the local sources of contamination and controlling these as much as possible through treatment of discharge and reduction of faecal input can protect the watershed. Treatment of sewage or agricultural waste forms an important barrier to environmental transmission (90-99.7% removal).
For production of safe drinking water only a few techniques can be used to effectively decrease the numbers of oocysts or deactivate them. Disinfecting by chlorine or UV is not very effective; a filtration step (slow sand filtration, membrane filtration, coagulation/filtration) or soil passage is better. Sometimes ozone may be effective. A well-designed treatment plant is the most effective action.
An annual infection risk level of 1×10^{-4}, as proposed by the EPA is currently used in the USA, Canada and the Netherlands as the basis for determining the appropriate removal efficiencies of the drinking-water production systems. The implementation of guide values is still hampered by the difficulty of determining source water quality and treatment efficiencies accurately.

Additional reading

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Dengue

Dengue, dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS) are mosquito-borne, viral infections, which in recent years have come to represent a major international public health concern in over 100 countries. These dengue infections threaten the health of more than 2.5 billion people living in urban, peri-urban and rural areas of tropical and subtropical regions. Dengue haemorrhagic fever (DHF), a potentially lethal complication, was first recognised during the 1950's and is today a leading cause of childhood mortality in several Asian countries. The average mortality rate is 5%, with some 24,000 deaths per year.
The rising significance and continued spread of dengue led to the approval of a resolution on dengue prevention and control at the World Health Assembly in 1993. This resolution was followed by the adoption of a global strategy on dengue and DHF in 1995.

Facts, trends and effects

Facts and trends: Before 1970 only nine countries had experienced DHF epidemics; however, this number had increased more than fourfold by 1995. In 1995 the WHO reported the morbidity due to dengue at 1.75 million episodes per year and the mortality at 20,000 per year. In November 1998 the same WHO estimated that some 2500 million people were at risk - two-fifths of the world population, with every year 50 million cases of Dengue infections worldwide.
The disease is now endemic in more than 100 countries in Africa, the Americas, the eastern Mediterranean, Southeast Asia and the western Pacific, with Southeast Asia and the western Pacific most seriously affected. In 1998 alone, there were more than 616,000 cases of dengue in the Americas, of which 11,000 constituted DHF. The disease is also rapidly spreading to new areas. Between January and October 1998 nearly 475,000 cases were reported in Brazil. During epidemics of dengue, attack rates among those susceptible to the disease are often 40-50%, but may even reach 80-90%. An estimated 500,000 cases of DHF require hospitalisation each year; a large population of the afflicted consists of children, and roughly 5% of those hospitalised die.

Effects: Dengue is a severe, flu-like illness that affects infants, young children and adults. The clinical features of dengue vary according to the age of the patient. Infants and young children may have a non-specific febrile illness with rash. Older children and adults may have either a mild febrile syndrome or the classical incapacitating disease, characterised by abrupt onset and high fever, severe headache, pain behind the eyes, muscle and joint pains, and rash.
Dengue haemorrhagic fever is a potentially deadly complication that is characterised by high fever, haemorrhagic (bleeding) phenomena - often with enlargement of the

liver - and in severe cases, circulatory failure. The illness commonly begins with a sudden rise in temperature accompanied by facial flush and other non-specific constitutional symptoms of dengue fever. The fever can continue for 2-7 days, body temperature rising as high as 40-41°C. After a few days the patient's condition may suddenly deteriorate; the temperature drops, followed by signs of circulatory failure and either possible death within 12-24 hours or, with as a result of appropriate volume replacement therapy, the patient may quickly recover.

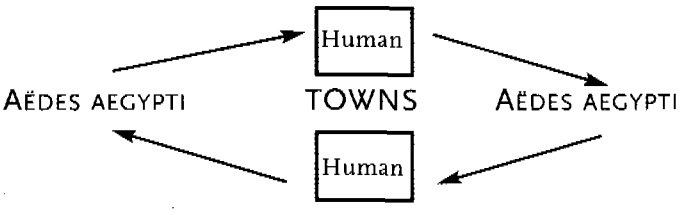
Causes and pathways

Dengue viruses are transmitted to humans through the bites of infective female *Aëdes* mosquitoes. Mosquitoes generally acquire the virus while feeding on the blood of an infected person. Once infective a mosquito is capable of transmitting the virus to susceptible individuals for the rest of its life during probing and blood feeding. Humans are the main amplifying host of the virus, although studies have shown that in some parts of the world monkeys may become infected and perhaps serve as a source of virus for uninfected mosquitoes. The virus circulates in the blood of infected humans for 2-7 days, at approximately the same time as they have fever; *Aëdes* mosquitoes may acquire the virus when they feed on an individual at this time.

There are 6 arboviruses known to cause dengue. These are types 1, 2, 3, 4, TH-36 and TH-SMAN, all transmitted through the bites of the *Aëdes aegypti* mosquito (also the transmitter of Yellow Fever), causing haemorrhagic fever and lesions in various organs. In recent years *Aëdes albopictus* has emerged as a secondary global dengue vector.

The transmission pathway is short, from humans via the invertebrate anthropod mosquito, *Aëdes aegypti*, and back to humans (see Figure 1).

Figure 1: Transmission route Aëdes aegypti



Factors in the transmission by anthropods include:

- susceptibility of the anthropod to infection
- breeding habits of the anthropod
- biting habits of the anthropod
- anthropod longevity, dependent on temperature, humidity and the availability of hosts to feed on
- abundance of anthropods

Actions

Global

The WHO, UNDP, World Bank and other agencies provide information, monitor the spreading of the disease, provide training and maintain contact with centres for disease control and prevention. The WHO regional offices have conducted regional seminars to develop regional strategies, and in Bangladesh have assisted with the development of a national strategy.
The WHO has also issued the brochure, "Preventing Dengue and dengue haemorrhagic fever, a fact sheet for municipal and community leaders" (CDT/FIL(DEN)/94.4).

Immunity, prevention and treatment

Immunity: Vaccine development for dengue and dengue haemorrhagic fever is difficult since any of the known viruses may cause the disease. Furthermore, protection against one or two of the dengue viruses may increase the risk of a more serious disease. Nonetheless, progress is gradually being made in the development of vaccines that may protect against all four dengue viruses. Such products could be commercially available within several years.

Prevention and control: The only method at present to control the spread of the disease is to combat the vector (mosquito). In Asia and America the *Aëdes Aegypti* breeds primarily in water from man-made water/food containers, reservoirs, tree holes, leaf axils and even old tyres. The rapid geographical spread of the species *Aëdes albopictus* has been largely attributed to the international trade in used tyres. Proper solid waste disposal and improved water storage practices, including covering containers to prevent access by egg-laying female mosquitoes are among the methods encouraged through community-based programmes.
Application of appropriate insecticides to larval habitats e.g. water vessels, prevent mosquitoes breeding for several weeks but must be re-applied periodically. Regular monitoring and surveillance of the natural mosquito population should accompany control efforts so as to determine the impact of the measures e.g. insecticides.

Treatment: There is no specific treatment for dengue fever. Maintenance of the circulating body fluid volume is the central feature of DHF case management. Appropriate supportive therapy may reduce mortality to less than 1%.

Additional reading

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Internetsites:
www.who.int/ctd/html/dengue.html (1999)
www.who.int/inf-fs/fact107.html (1998)

Giardiasis

Giardia is a flagellated protozoa, which has been found in more than 40 animal species, for example: Giardia muris in rodents, birds and reptiles, G. intestinalis (duodenalis, lamblia) in mammals (including humans), rodents, reptiles and birds, G. agilis in amphibians, G. ardea in the blue heron and G. psittaci in the budgerigar.
G. intestinalis causes gastroenteritis in humans; besides this, it is ubiquitous in the aqueous environment and therefore of concern for the (drinking-)water supply. This protozoa was already seen by Antonie van Leeuwenhoek.

Facts and trends

Facts: The parasite, *Giardia*, is shedded in the faeces of infected hosts in the form of cysts, the environmental-resistant transmission stage of the parasite. These cysts are infectious and may remain in the environment for long periods without losing their infectious nature. A high degree of genetic heterogeneity is found in human and animal isolates; however, it is still uncertain if this is related to host specificity and pathogenicity.
Studies on infection with healthy human volunteers show a good relationship between the probability of infection and the ingested dose; ingestion of ten cysts containing *G. intestinalis* resulted in 100% infection in the volunteers. Non of the infected volunteers, however, developed symptoms of giardiasis. The infection-to-illness ratio varies between isolates. Asymptomatic carriage appears to be common. The time between infection and the occurrence of *Giardia* cysts in the faeces ranges from 12 to 19 days. The occurrence of symptoms varies between 1 and 75 days. The most predominant symptoms are: diarrhoea (fatty, yellowish), weakness, weight loss and abdominal pain. The infection is generally self-limiting to 2-4 weeks.

Trends: *Giardia* infections are common in children in developing countries. Also occurring in industrialised countries, *Giardia* shows prevalence peaks in children of age 1-4 years; a second peak occurs in the 20-40 age group (possibly because of caring for small children). In developing countries the percentage of patients with gastroenteritis is around 20% and in the industrialised countries about 3-7%.
Cysts are ubiquitously present in the aquatic environment. Water-related outbreaks of giardiasis have been reported for almost 30 years. In the US *Giardia* is the most commonly identified pathogen, with more than 100 waterborne outbreaks (based on epidemiological evidence). Other waterborne outbreaks have been reported in Canada, Australia, New Zealand, the United Kingdom and Sweden. Most of these outbreaks have been linked to consumption of untreated surface water or groundwater contaminated by sewage and wild rodents. Drinking surface water which is only disinfected or for which filtration is ineffective, or where there is damage in the distribution system, may also contribute to outbreaks.

Causes and pathways

Person-to-person transmission is the major route for spreading infection. The role animals play in transmitting human giardiasis is still controversial. As previously mentioned, several water-related outbreaks have been described around the world.

Actions

Preventing transmission requires a multiple-barrier approach: protection of watersheds and installation of adequate treatment, along with verification of treatment by monitoring.
Recognising the local sources of contamination and controlling these as much as possible by treatment of discharge and reduction of faecal input can protect the watershed. Treatment of sewage or agricultural waste is an important barrier to environmental transmission (90-99.7% removal). For production of safe drinking water only a few techniques can be used to effectively decrease the numbers of cysts or deactivate them. Disinfecting with chlorine or UV is not very effective; it is better to use a filtration step (slow sand filtration, membrane filtration, coagulation/filtration) or soil passage. Sometimes ozone may be effective. A well-designed treatment plant is the most effective action.
An annual infection risk level of 1×10^{-4}, as proposed by the EPA, is currently maintained in the USA, Canada and the Netherlands as the basis for determining the appropriate removal efficiencies of drinking-water production systems.
The implementation of guide values is still hampered by the difficulty in accurately determining source water quality and treatment efficiencies.

Additional reading

Betts et al. Protozoan parasites and water. The Royal Society of Chemistry, United kingdom 1995

Medema GJ. Cryptosporidium and Giardia: new challenges to the water industry, 1999

Malaria

Malaria is by far the world's most important tropical parasitic disease. It is a public health problem today in more than 90 countries and has a worldwide prevalence of 300-500 million clinical cases each year with a mortality that is estimated to be over 1 million deaths each year. Mosquitoes transmit malaria. The number and type of Anophiline mosquitoes in a given area determine the extent of transmission. Transmission is affected by climate and geography and often coincides with the rainy season.

Malaria was already described in ancient Chinese medical literature in 2700 B.C. The disease came to Europe much later, in about 600 BC, and by the time of Hippocrates (ca. 460-377 B.C.) had become a major health problem. Hippocrates connected this disease, characterised by intermittent fever (malaria), with swampy areas. The next development was in 1880, when the parasite was seen in human blood by the French army doctor, Lavaran. The complete life cycle and the role of the mosquito was finally discovered in 1898 by the British army surgeon, Ross.

Facts and trends

Facts: Malaria is caused by four different parasites of the same genus, *Plasmodium vivax, Plasmodium ovale, Plasmodium malariae* and *Plasmodium falciparum*. They cause different diseases, of which the most serious is malignant malaria or malaria tropica, brought on by *Plasmodium falciparum. Plasmodium* is a small parasite, one cell in total, living in the red blood cells. The vector is a mosquito, *Anopheles*, of which there are many different species able to transmit malaria. The efficiency of the vector depends on the species. *Plasmodium falciparum* is the most prevalent species in the tropics and subtropics. *Plasmodium vivax* has been successfully eradicated in the USA and Europe. Worldwide, this species is more common in temperate zones, therefore not generally occurring in sub-Saharan Africa. *Plasmodium ovale* is mostly found in tropical Africa and *Plasmodium malariae* occurs in tropical Africa and Southeast Asia. Malaria belongs to a WHO class 1 reportable disease.

Trends: Malaria is still a very common disease and one of the great child killers in Africa. It kills one child every 30 seconds. *P. vivax* seems to be returning to some areas where it had already been irradicated, for example, in the Central Asian Republics of Tajikistan and Azerbaijan and in Korea. In Africa, Asia and Latin America there is an increase in drug resistance of *Plasmodium falciparum*, and so a growing number of patients suffer from infections due to a multi-resistant *P. falciparum* strain. Chloroquine-resistant strains of *P. vivax* have been seen in Iryan Jaya, and this problem seems to be spreading in Asia.

In malaria endemic parts of the world, a change of risk of malaria can be the unintended result of economic activity or agricultural policy that changes the use of land (e.g. creation of dams, irrigation schemes, commercial tree cropping and deforestation). "Global warming" and other climatic events such as "El Niño" also play their role in increasing the risk of this disease.

Causes and pathways

Causes: Malaria tropica (caused by *Plasmodium falciparum*) may exhibit quite a varied clinical picture, including fever, chills, sweating, cough and diarrhea up to shock, coma and death. The fatality rate among cases of untreated children and non-immune adults exceeds 10%. Malaria is particularly dangerous during pregnancy. It causes severe anemia, and is a major factor contributing to maternal deaths in endemic areas.
Malaria tertiana (caused by *P. vivax* and *P. ovale*) and malaria quartana (caused by *P. malariae*) are generally not life threatening, except in the very young, very old and immunocompromised. Typical are the periods of fever, shaking chills, headache and nausea, ending in profuse sweating, alternating with a period free of symptoms.

Pathways: The life cycle is fairly complicated, with a sexual stage in the mosquito and an asexual stage in humans. The infection starts with a bite of the female *Anopheles*, who injects the parasites into the bloodstream through her saliva. After a few days to weeks the parasites in the liver burst during an episode and infect the red blood cells. They divide asexually in these red blood cells and burst every 2 or 3 days, thus causing the typical malaria attacks of fever. The mosquitoes are infected by taking a blood meal, in which they suck up the infected red blood cells. Malaria can also be transmitted by transfusion of infected blood.

Actions

- Elimination or reduction of the Anopheles' breeding places by improvement of hygiene. Larvacides and biological control with larvivorous fish can be useful here;
- Prevention of mosquito bites by using impregnated bed nets, insect repellents and protective clothing (long sleeves and pants after dusk);
- Proper treatment of both acute and chronic patients;
- Chemoprophylaxis for travelers, depending on geographical distribution of malaria and drug sensitivity;
- Screening blood donors for malaria;
- Applying insecticides to aircraft, ships and other vehicles.

UNICEF recognises that malaria is one of the five major causes of under-five child mortality. The agency has made the disease a top priority, supporting malaria control programmes in 32 countries, 27 of which are in Africa.

Additional reading

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WHO Fact Sheets Malaria no 94; http://www.who.int/inf-fs/fact094.html Revised October 1998

Yellow Fever

Yellow fever is a viral, water-related disease, probably originating in West Africa and transported to the Americas by ships carrying mosquitoes. Known from historical writings going back 400 years, it has caused large epidemics in Africa (Zimbabwe, Sudan, Ethiopia) and the Americas (Brazil, USA, Argentinia) but was never reported in Asia and Australia. This is the reason for the strict control measures for (im)migrants and aircraft. Just as dengue (Aedes aegypti), yellow fever is transmitted by mosquitoes.

Facts, trends and effects

Facts and trends: The virus is constantly present as an endemic disease, characterised by low levels of infection, in some tropical areas of Africa and the Americas. Thirty-three countries, with a combined population of 468 million, are at risk in Africa. In Latin America yellow fever is endemic in nine countries. Formerly, yellow fever was also endemic in Europe, but nowadays the vector (mosquitoes) is absent. There are 200,000 estimated episodes of yellow fever per year.

Effects: Yellow fever has an incubation period of 3 to 6 days, followed by two phases. The first, "acute", phase is normally characterised by fever and muscle pain (with prominent backache), headache, fever (shivers), loss of appetite, nausea and/or vomiting. After a few days most patients improve, but about 15 % enter a "toxic" phase when jaundice develops; bleeding can occur from the mouth, nose, eyes and/or stomach. Half of these patients die between 10 and 14 days. The others recover without signifcant organ damage.

Yellow fever is difficult to diagnose. It can be easily confused with malaria, typhoid and other arboviral infections. Laboratory tests require a highly trained laboratory staff using specialised equipment and materials.

Causes and pathways

Yellow fever is caused by a virus of the flavivirus group. In Africa there are two distinct genetic types; Latin America also has two types, but only one has been identified as the cause of disease outbreak.
There are three types of transmission cycles for yellow fever depending on the breeding of the virus carrying mosquitoes: sylvatic, intermediate and urban.
All three cycles occur in Africa, but in South America, only sylvatic and urban yellow fever occur. The sylvatic cycle passes from infected monkeys in the African rain forests via mosquitoes to men working in the forest. Urban yellow fever is introduced by infected migrants moving into urban areas and is transmitted through mosquitoes in the domestic areas.
The mosquito can also pass the virus via infected eggs to its offspring (vertical transmission). The eggs produced are resistent to drying and lie dormant through dry conditions, hatching when the rainy season begins. Therefore, the mosquito is the true reservoir of the virus, ensuring transmission from one year to the next. Several different species of the Aëdes and Haemogogus (S. America only) mosquitoes transmit the yellow fever virus.

Actions

Immunity: Yellow fever vaccination is safe and highly effective. A single dose provides protection within a week for a period of 10 years to 95% of those vaccinated. Vaccination campaigns proved very effective between 1939 and 1952, when yellow fever virtually vanished from West Africa. To prevent an epidemic at least 80% of the population must have immunity to yellow fever.

Prevention: Mosquito control measures are an effective means for controlling the spread of yellow fever.

Treatment: There is no specific treatment for yellow fever.

Additional reading

Internetsite:
www.who.int/inf-fs/en/fact100.html

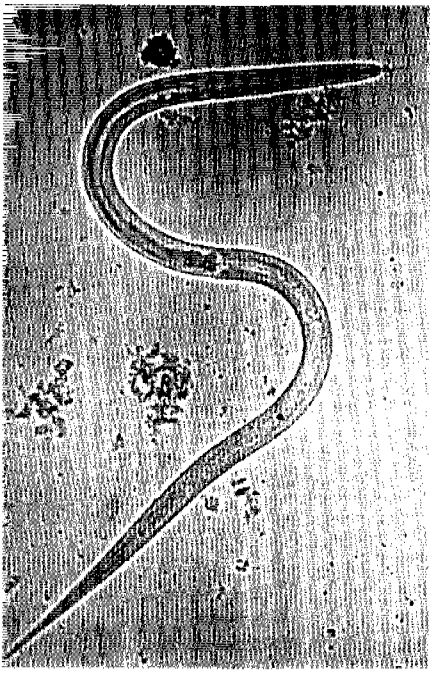
6 POOR SANITATION

POOR SANITATION PROVIDES A TRANSMISSION-PATH TO WATER-RELATED DISEASES THROUGH INEFFECTIVE WASTEWATER AND EXCRETA DISPOSAL

EXAMPLE:

HOOKWORM INFECTION

Hookworm infection



The global burden of intestinal nematodes, one of which is hookworm, amounts to an estimated number of infections of over one billion. Manure and, in particular, human excreta contain the organisms (pathogens) causing the infection. Direct application of night soil to the land can exacerbate the spread of such diseases as typhoid, cholera, and hookworm. Hookworm infections are transmitted mainly through defective and/or unsanitary faecal disposal.
Treating human solid waste/animal manure by means of anaerobic digestion in the biogas plant before using it as fertiliser lessens the chances of disease transmission since this digestion process is capable of destroying most pathogens. Studies have shown that less than 10% of the eggs pass through the digestion process into the effluent. The hardiest worms, e.g. roundworm or ascaris, do, however, manage to pass relatively unscathed through treatment plants.

Facts, trends and effects

Estimates of the number of infections in different regions of the world are still calculated by extrapolating from prevalence surveys. The most recent estimates show over 1.2 billion infections with hookworms, virtually the same infection rate as 50 years ago. This is due to the large increase in the world population. However, the rate of infections has decreased greatly in North America, Europe, Japan and Australia, although infection can be contracted in Europe and The Netherlands in mines and tunnels where temperatures and humidity are favourable and sanitation absent.
Infection is highest in children of school and pre-school age. It is estimated that up to 22 million DALYs (= disability adjusted life years) are lost per year because of hookworm. In comparison, an estimated 35 million DALYs are lost due to malaria, 34 million due to measles, 4.5 million due to schistosomiasis and 11.8 million due to Vitamin A deficiency.
Intestinal helminths infections, such as hookworms, cause many non-specific symptoms, with very serious conditions being much rarer. Worm infections are known to reduce physical fitness, growth and cognitive abilities.

Causes and pathways

The Hookworm family knows two genera. The genus *Necator americanus*, or the New World hookworm, is found in the small intestine of man, as well as in the gorilla, monkey, rhinoceros and dog (puppy). The genus *Ancylostoma* of which the eggs are larger than of the *Necator americanus* is found in Africa and Asia.
Transmission of hookworm infections seems to occur between households,

through the soil rather than the domestic environment. Hookworms are latent and persistent, can live for 10-20 years and produce eggs which in 5-8 days develop into infectious larvae. Larvae remain infectious for a couple of weeks in the ground. Hookworm has no intermediate hosts. They can contaminate yards/ gardens and pastures and croplands, via communal defecation, the latter if manure has not been treated prior to land application. The main carriers are children, mainly of pre-school and school age.

Actions

Global: Helminths infection control programmes are more operational than medical. Safe, effective and cheap drugs are available but the challenge is to deliver programmes on a sustainable basis, with low per capita cost and to those who need them most, i.e. children. Control can be stimulated by promoting the provision and universal use of toilets with clean floors.

Prevention and treatment

Prevention: It is now generally believed that helminths infections are difficult to eradicate through eradication campaigns, both for practical and ecological reasons. Rather, control concentrates on reducing the worm burden, thus controlling morbidity. Some research indicates that improved domestic hygiene, particularly in relation to children, can be an effective means of control. Other research shows the observed decline in child mortality in Nicaragua since 1960, of which 5% is due to worms (incl. hookworms), to be related to the increase in the numbers of health workers.

Treatment: Since the discovery of benzimidazole the potential for chemotherapeutic treatment has increased greatly. The most commonly used drugs are albendazole and mebendazole.
Cost analyses of school-based intervention programmes show these programmes to be highly cost-effective, turning out as low as US$ 0.16 per child per year.

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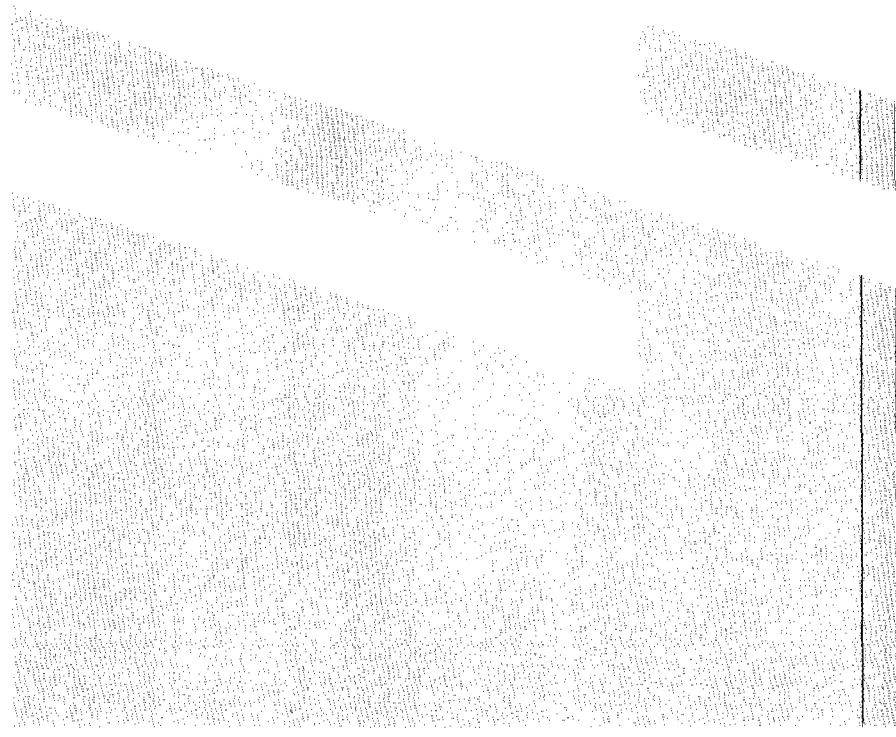


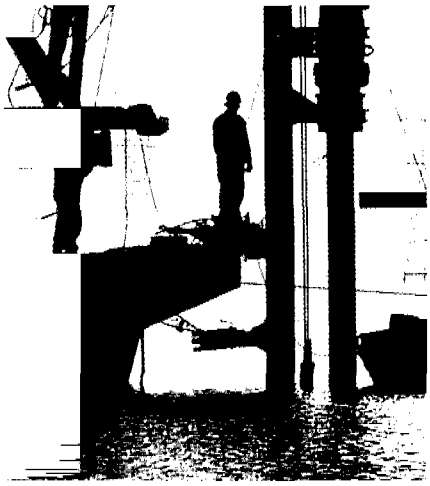
7 METALS

METALS OCCUR IN THE WATER-SYSTEM AND CAN BE PRESENT IN AN ORGANIC OR INORGANIC FORM

EXAMPLES ARE:

ALUMINIUM, ARSENIC, CADMIUM, COPPER, FLUORIDE, LEAD AND MERCURY

Aluminium



Aluminium is widespread and one of the most abundant metals, accounting for 8% of the earth's crust. It is always found combined with other elements such as minerals and rocks. Aluminium is found as a normal constituent of soils, plants and animal tissues. It is used in the manufacture of many products, such as aircraft, electrical conductors and dental fillings. It is also used in combination with other chemicals to serve many purposes like use in medicinals, catalysts and dyes, and for fireproofing and tanning. Its compounds are also used as antacids, antiperspirants, food additives and vaccine adjuvants. Aluminium salts are widely used in water treatment as flocculants.
Aluminium is not a necessary element for the functioning of the human body.
In the last decade aluminium has been linked to Alzheimer's disease, however until now there is not enough evidence to support this.

Facts and trends

Facts: Aluminium can dissolve in lakes, streams and rivers, depending on the quality of the water. Acid rain may dissolve aluminium from soil and rocks. Aluminium is usually present in drinking water in the form of reactive species of low relative molecular mass. In natural waters, it is usually associated with particulate matter of organic complexes of high relative molecular mass.
Some aluminium is insoluble in water, while some aluminium complexes, however, are soluble in water. Aluminium solubility in water is dependent on the acidity of the water.

Trends: Aluminium may be present in natural waters as a consequence of leaching from soil and rocks. In the USA aluminium in groundwater was found in concentrations ranging from 14 to 290 µg/l and in surface water in concentrations ranging from 16 to 1170 µg/l.
Aluminium salts are used as coagulants in water treatment; here residual aluminium will be formed. When the residual aluminium concentrations are high, high levels of aluminium in tap water (drinking water) may result.
The presence of aluminium in water may produce discoloration of the water. The incidence of discoloration in the water distribution system increases if the aluminium level in the tap water (drinking water) exceeds approximately 0.1-0.2 mg/l of drinking water, which is similar to the frequency level of consumer complaints.
The drinking-water standard for aluminium in Europe is 0.2 mg/l. The guideline value used by the WHO for aluminium concentration in drinking water is

0.2 mg/l. This is not a health-based guideline value; the concentration of 0.2 mg/l forms a compromise between the practical use of aluminium salts in water treatment and discoloration in distributed water.

Causes and pathways

Causes: Exposure to aluminium takes place through water and drinking water, air and mainly food. There is very little exposure through aluminium cooking utensils. The daily aluminium intake is estimated to be within the range of 5-20 mg per day. Aluminium in drinking water will usually contribute only a very small proportion of the total daily intake; about 2% of the total daily aluminium intake originates in drinking water.

Pathways: The human body absorbs only less than 1% of the ingested aluminium. Once absorbed, aluminium binds to proteins and is eliminated by the kidneys. Individuals with renal insufficiency are unable to excrete aluminium via the kidneys so tend to accumulate it in their bodies.
Patients on dialyses are known to develop the "dialysis dementia" syndrome. The incidence of this syndrome is correlated with the aluminium concentration in the water used to prepare the dialysate fluid. The syndrome is characterized by an insidious onset of altered behaviour, dementia, speech disturbance, muscular twitching and convulsions, and usually has a fatal outcome.
Aluminium is thought to have an important role in some neurodegenerative diseases: amyotrophic lateral sclerosis (neurologic disease which leads to muscular atrophy) and Parkinson's dementia. A chronic nutritional deficiency of calcium and magnesium might increase absorption of aluminium, resulting in deposition of aluminium in the neurons. Interference with the structure and formation of the brain neurons characterize these diseases. Parkinson's dementia has a very high incidence among the Chamorro people of Guam.
Aluminium has also been linked to Alzheimer's disease. The symptoms of this disease are memory lapses, disorientation, confusion and, frequently, depression, all of which will lead to progressive mental deterioration. Patients with Alzheimer's disease have elevated levels of aluminium in their brain tissue. It is not known whether aluminium causes the disease or whether the accumulation of aluminium is an effect of the disease. In some studies, aluminium has appeared to be associated with the brain lesions that characterize Alzheimer's disease. In only in a few studies has the incidence of the disease been associated with aluminium in drinking water. There is not enough evidence to support a causal role for aluminium in Alzheimer's disease. Aluminium is not known to be carcinogenic.

Actions

Aluminium salts are used as coagulants in water treatment. Residual aluminium concentrations in drinking water are a function of the aluminium levels in the source water, the amount of aluminium coagulant used and the efficiency of filtration of the aluminium floc. When aluminium is not used as a coagulant, the amount of residual aluminium in drinking water will decrease.
Efficient drinking-water treatment technologies to remove aluminium are:

- coagulation/filtration with ironsalts
- control of the acidity

Prevention of high concentrations of aluminium in drinking water and its sources is the best option.

Additional reading

WHO (1996)
Guidelines for drinking water quality.
Health criteria and other supporting information, 2nd ed., vol. 2, World Health Organisation, Geneva, 1996

Journal AWWA
Bioavailability of Al in alum-treated drinking water, November 1999, Vol. 91, No. 11, page 84-93

Internetsites:
http://mail.odsnet.com/TRIFacts/196.html
http://www.atsdr.cdc.gov/tfacts22.html

Arsenic

Arsenic is a naturally occurring element widely distributed throughout the earth's crust, most often in compounds with oxygen, chlorine and sulfur. Its use is mainly commercial and industrial, in alloying agents and wood preservatives, but also, although to a limited extent, in insecticides, weed killers and pharmaceuticals.
There is no evidence that arsenic is an essential trace element for the human body.
Chronic exposure to high levels of arsenic causes severe skin problems followed by disablement.

Facts and trends

Facts: Arsenic, introduced into water through the dissolution of minerals and ores from industrial effluents and atmospheric deposition, is found especially in waters flowing through arsenic-rich rocks.
High concentrations of arsenic in drinking water are found in various parts of the world including Argentina, Bangladesh, Chile, China (Province of Taiwan), Hungary, India (West Bengal), Mexico and the USA.
Human arsenic intake is more associated with food, e.g. seafood, than with drinking water. However, arsenic in fish is mostly low-toxicity organic arsenic. Recently drinking water represents by far the greatest hazard since arsenic in groundwater, the main source of drinking water, is predominantly inorganic and of much higher toxicity. Most arsenic compounds can dissolve in water, although some high-toxicity inorganic arsenic compounds are insoluble in water.
Most arsenic compounds have no odour or special taste.

Trends: The concentration of arsenic in natural waters generally varies between 1 and 2 µg/l. In areas containing natural sources, however, concentrations may be elevated up to 12 mg/l.
In surface waters arsenic concentrations fall within a range of few micrograms per litre. In groundwater used for drinking water, especially water abstracted from anaerobic aquifers, very high levels of arsenic are common. Concentrations of arsenic in drinking water prepared from surface water sources are generally lower than in water prepared from groundwater.
In Bangladesh and West Bengal (India) arsenic in drinking water presents a serious problem for public health. Here, the source of the arsenic contamination of groundwater is natural. The levels found are higher than seventy times the national standard of 0.05 mg/l in both Bangladesh and India. In these countries the number of people drinking water contaminated with arsenic has increased over the past 25 years due to well-drilling and population growth. The number of persons affected may therefore increase further. It has been estimated that in Bangladesh and India some 30 million people may be at risk of arsenic exposure.

In the past drinking water was prepared from surface water in this region. Because of the contamination of these sources people moved to groundwater wells for their drinking water.
The drinking water standard for arsenic in Europe is recently reduced from 0.05 mg/l to 0.01 mg/l.
The WHO established 0.01 mg/l as a provisional guideline value for arsenic in drinking water, with a view to reducing the concentration of arsenic in drinking water. This guideline used to be 0.05 mg/l, which many countries, e.g. the USA, have upheld as the national standard for arsenic in drinking water.

Causes and pathways

Causes: Exposure to arsenic can take place through contaminated drinking water, food and air. The estimated mean daily intake of arsenic from drinking water will generally be less than 10 µg; this is based on a concentration of arsenic in drinking water in areas with no natural sources of less than 5 µg/l, and is found where the average daily consumption of drinking water is two litres.

Pathways: Ingested arsenic is poorly absorbed and largely unchanged on excretion. Soluble arsenic compounds are rapidly absorbed from the gastrointestinal tract; organic arsenic is rapidly and almost completely eliminated via the kidneys. Inorganic arsenic may accumulate in skin, bone and muscle; its half-life in humans is between 2 and 40 days.
Inorganic arsenic is a human poison. Organic arsenic is less harmful. Exposure to high levels of arsenic in food or water, and drinking water, can cause several acute intoxication effects like poor appetite, nausea, abdominal pain, vomiting, diarrhoea, muscle cramps and heart complaints.
Chronic exposure to arsenic includes melanosis (abnormal black-brown pigmentation of the skin), de-pigmentation, (hyper)keratosis (thickening) of palm and sole, gangrene of the lower extremities, peripheral vascular disorders, cardiovascular and neurological diseases, and skin cancer. Ingesting inorganic arsenic increases the risk of skin cancer and tumours of the bladder, kidney, liver and lung. Since inorganic arsenic compounds are carcinogenic in humans, there can be no safe level of exposure to arsenic.

Actions

Several drinking water treatment technologies may be effective for the removal of arsenic. These include:

- coagulation/filtration
- lime softening
- ion exchange
- adsorption on activated alumina
- membrane technologies (reverse osmosis, nanofiltration, electrodialysis)

Chemicals for household treatment may be effective too in reducing the risk of further complications due to arsenic in drinking water.
However, in Bangladesh or West Bengal (India) for instance, most drinking-water

treatment technologies are much too expensive. Therefore only a few reliable options are available to provide safe drinking water:

- shallow hand pumps for zones where arsenic is undetected
- arsenic-free water from deeper aquifers (> 100 or 200 m)
- rainwater harvesting
- pond-sand filtration
- bucket-type household treatment
- piped water supply from safe or treated sources.

Additional reading

WHO (1996)
Guidelines for drinking water quality
Health criteria and other supporting information, 2nd ed., vol.2. World Health Organisation, Geneva, 1996

Internetsites:
http://www.who.int/inf-fs/en/fact210.html
http://www.atsdr.cdc.gov/tfacts2.html
http://mail.odsnet.com/TRIFacts/204.html
http://www.epa.gov/OGWDW/ars/trtmt.html

Cadmium

Cadmium is a natural element in the earth's crust. It is usually found as a mineral combined with other elements such as oxygen, chlorine or sulfur.
All soils and rocks have some cadmium in them. Cadmium is used mainly as an anticorrosive, primarily for metal plating and coating operations. It is also used for batteries, pigments, plastics, electronic components and nuclear reactors.
Cadmium has no known biological functions, although it competes with zinc for binding sites and can therefore interfere with some of zinc's essential functions.
Chronic exposure to cadmium causes kidney disease; high exposure to cadmium can cause bone disorders ("itai-itai" disease).

Facts and trends

Facts: Cadmium is introduced into the water via waste streams, spills or leaks at hazardous waste sites and leaching of landfills. Cadmium also occurs naturally in zinc, lead, copper and other ores, which can serve as cadmium sources for groundwater and surface waters, especially when in contact with soft, acidic water. A particular characteristic of cadmium is its release into drinking water from the corrosion of some galvanized plumbing and water-main materials.

Cadmium does not break down in the environment, but can change forms.
Some cadmium compounds are able to leach through soils to groundwater.
When cadmium compounds bind with river sediments, they can be more easily bio-accumulated or re-dissolved when sediments are disturbed, such as during flooding.
Fertilizers produced from phosphate ores constitute a major source of diffuse cadmium pollution.
Cadmium in slightly soluble in water. The solubility of cadmium in water is influenced to a large degree by its acidity. In fresh waters, cadmium toxicity is influenced by water hardness: the harder the water, the lower the toxicity.
Cadmium does not have a definite taste or odour.

Trends: Cadmium concentrations in natural waters are usually below 1 µg/l. Median concentrations of dissolved cadmium found at 110 stations around the world amounted to <1 µg/l, with a maximum concentration of 100 µg/l in Rio Rimao (Peru).
In 1988 average cadmium concentrations in the Rhine were found to be 0.1 µg/l, and in the Danube the average cadmium concentration found was 0.025 µg/l.
Contamination of drinking water may occur as a result of cadmium being present as an impurity in the zinc of galvanized pipes or cadmium-containing solders in fittings, water heaters, water coolers and taps. In some areas in Sweden drinking water from shallow wells where the soil has been acidified contained concentrations of cadmium of about 5 µg/l. In Saudi Arabia, mean concentrations of 1-26 µg/l were found in samples of potable water.
The drinking water standard for cadmium in Europe and the USA is 5 µg/l.
The WHO maintains a guideline value of 3 µg/l for cadmium in drinking water.

Causes and pathways

Causes: Exposure to cadmium can take place through contaminated water and drinking water, and food, air and cigarette smoke. Cadmium is ingested mainly via food; only a very little (a few percent) cadmium is ingested via drinking water. The intake from drinking water is usually less than 2 µg per day. Smoking will increase the daily intake of cadmium.

Pathways: Cadmium is mainly absorbed via the gastrointestinal tract into the kidneys. In healthy persons only about 3-7% of the cadmium ingested is absorbed. Excretion takes place mainly via the faeces; only a small part is excreted in the urine. The absorbed cadmium is accumulated primarily in the kidneys, but also in the liver.
Exposure to high levels of cadmium through drinking water for relativily short periods of time can, potentially, cause nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.
Lifetime exposure to cadmium at high levels can, potentially, cause kidney, liver, bone and blood damage.
Long-term exposure to lower levels of cadmium leads to a build-up of cadmium in the kidneys and may possibly lead to kidney disease.
Cadmium also affects the bones. It has been known to cause bone and joint aches,

and pains. This syndrome, termed "itai-itai" disease, was first described in Japan. In some areas in Japan people were chronically exposed to cadmium through contaminated food and drinking water. The daily intake of cadmium varied between 100 and 2000 µg per day. Cadmium remains in the body for a very long time and can accumulate on many years of exposure to low levels; the biological half-life in humans is 10-35 years.
Cadmium and cadmium compounds may reasonably be anticipated to be carcinogenic, but there is no evidence that drinking cadmium-contaminated water causes cancer. There is also no evidence that cadmium is genotoxic.

Actions

Cadmium can be effectively removed by applying a number of drinking-water treatment technologies:

- coagulation/filtration
- lime softening
- ion exchange
- reverse osmosis

Several of these drinking-water treatment technologies are, however, very expensive to conduct. Therefore prevention of high concentrations of cadmium in drinking water and the sources of drinking water is the best option.

Additional reading

WHO (1996)
Guidelines for drinking-water quality.
Health criteria and other supporting information,
2nd ed., vol. 2. World Health Organisation, Geneva 1996

Internetsites:
http://www.atsdr.cdc.gov/tfacts5.html
http://mail.odsnet.com/TRIFacts/207.html
http://www.epa.gov/OGWDW/dwh/c-ioc/cadmium.html
http://www1.ldc.lu.se/iiiee/impacts/health/health_home.html#Cd

Copper

Copper is a commonly occurring element in our natural waters. This metal is found in natural deposits in the form of ores containing other elements, and is an important heat and electrical conductor. It is also used for roof coverings, household goods and chemical equipment, both as an ore and in many alloys. Copper oxides, chlorides, sulfates, ethanolates, bromides and carbonates are widely used in pest control, as inorganic dyes and food additives, in photography, seed desinfectants, fungicides and algicides, as well as in electroforming. Copper is also widely used in household plumbing materials (water pipes). Copper is an essential trace element for the human body. The average adult has between 75 and 150 mg copper in his/her body; half of it is contained in the skeleton and muscles.

Facts and trends

Facts: Although copper is rarely found in source water, copper mining, melting operations and municipal incineration may serve as sources of contaminated water. Surface water can contain high concentrations of copper due to sewage water discharges. Some aquatic organisms are sensitive to copper at micro-gram level. Copper may occur in drinking water, either by contamination of the source water used by the water system, or by corrosion of copper plumbing. Corrosion of plumbing is by far the greatest cause of concern.
Monovalent copper is unstable in aqueous solutions. Some copper (I) compounds are insoluble in water and other copper (I) complexes are stable in aqueous environments. Copper (II) forms complexes with both inorganic and organic ligands like ammonium and chloride ions, and humic acids. All water is corrosive for copper to some degree, very acidic water being the most extreme.
Most copper and copper compounds are soluble in water. Dissolved copper imparts a colour and an unpleasant astringent taste to drinking water; the taste threshold exceeds 5 mg/l.

Trends: Natural concentrations in drinking water are around a few micrograms per litre. Factors such as hardness, pH, anion concentrations, oxygen concentration, temperature and the technical condition of water pipelines influence the copper concentration in drinking water; water from copper pipes may contain several milligrams of copper per litre up to 22 mg/l. The drinking water standard for copper in Europe is recently reduced from 3 mg/l to 2 mg/l.
The Environmental Protection Agency (EPA) maintains an 'action limit' for copper in drinking water of 1.3 mg/l in the USA; if copper is present at levels above 1.3 mg/l in more than 10% of all homes tested by a water supplier, the system must continue to monitor this contaminant twice a year. If the copper concentrations are consistently found to be above this action level, treatment methods will be needed to reduce the copper concentration.

In view of the remaining uncertainties on copper toxicity in humans, the WHO maintains a health-based provisional guideline of 2 mg/l for copper in drinking water. This guideline should include taste problems too.

Causes and pathways

Causes: Exposure to copper takes place through contaminated water and drinking water, food and air. Copper is ingested by humans mainly via food and drinking water. Drinking water, however, can contribute a significant proportion of the daily copper intake if it has passed through copper installations.
In the Netherlands the average daily copper intake found per person is between 1.2 and 1.4 mg. In the western and eastern parts of Germany average daily intakes of 1.82-2.38 mg per person and less than 0.95 mg per person have been found, while in the USA, the average daily intake found per person is between 2 and 4 mg.

Pathways: Copper is absorbed in the stomach and upper intestine. It is transferred across the gut wall and carried to the liver where it is stored. The copper stored in the liver is incorporated into bile, secreted into the intestine and excreted in the faeces. Only about 30% of the copper intake will be absorbed.
Copper accumulates in the bones, muscles and liver, and is most concentrated in the brain and liver.
Toxic to high concentrations of copper can cause acute toxic effects such as nausea, vomiting, abdominal pain, diarrhoea, muscle pain, heart complaints, immune suppression and abnormal mental states. The estimated concentration of copper(II) in drinking water that can lead to symptoms of this type is 30 mg/l.
Especially in infants and young children, short-term exposure to high concentrations of copper can cause early childhood liver cirrhosis and death.
Chronic exposure to copper can cause Wilson's disease, a genetic disorder in which the liver is unable to remove copper from the body. Excessive amounts of copper accumulate in the body after several years of exposure, and lead to symptoms of liver disease and loss of brain function. Drugs to remove excess copper, and zinc, to promote excretion of copper, can be used to treat Wilson's disease. Zinc and copper compete with each other for absorption.
Copper is not known to be carcinogenic.

Actions

The following treatment technologies can effectively remove copper from drinking water:

- coagulation/filtration when copper is present in the source
- corrosion control of the distribution network

Prevention of high concentrations of copper both in drinking water and its sources, and corrosion control are the best options.

Additional reading

WHO (1996)
Guidelines for drinking water quality.
Health criteria and other supporting information,
2nd ed., vol.2. World Health Organisation, Geneva, 1996

Internetsites:
http://mail.odsnet.com/TRIFacts/210.html
http://www.epa.gov/OGWDW/dwh/c-ioc/copper.html
http://www.biospectron.se/copper_human.html

Fluoride

Fluorine is a natural element, accounting for about 0.3 g/kg of the earth's crust. Fluorine does not occur in the elemental state in nature because of its high reactivity but exists in the form of fluorides in a number of minerals such as flourspar, cryolite and fluorapatite. Fluorine combines with oxygen to make hydrogen fluoride and with metals to make fluorides like sodium, aluminium and calcium fluorides.
Fluorine, an essential trace element for humans, is present in the teeth, bones, thyroid gland and skin. Fluoride makes up about 2.6 g of the average body weight of humans.
Fluorine is used in rocked fuels, glass and the glass fibre industries, as well as in enamel, bricks and the production of phosphate fertilisers. Hydrogen fluoride goes mainly into the making of aluminium and chlorofluorocarbons (CFCs).
Fluorides are used to manufacture steel, chemicals, ceramics, lubricants, dyes and plastics. Fluorides also prevent dental caries, while fluorosilic acid is used in municipal water fluoridation schemes.
The main effects of high fluoride concentrations in drinking water are dental fluorisis and skeletal fluorisis.

Facts and trends

Facts: Fluorides erode from rocks and leach from phosphorus fertilisers into the water. Industrial discharges from mining are a source of contamination of river waters, with fluoride being introduced into the drinking water by contamination of the source entering the water system. The addition of fluorides to drinking water low in fluoride to prevent dental caries is a source of contamination. Hydrogen fluoride dissolves in water to form hydrofluoric acid; sodium fluoride is soluble in water, but aluminium and calcium fluoride are only sparingly soluble. Fluorine has a strong, sharp odour; hydrogen fluoride a biting odour.

TRENDS: Fluorides are present in many waters, e.g. 1.3 mg/l in seawater. In groundwater, fluoride concentrations are found of up to 10 mg/l, depending on the type of rock that the water flows through.
In the Netherlands the Rhine contains fluoride levels of below 0.2 mg/l; in the Meuse fluoride levels are between 0.2-1.3 mg/l. In 1990 fluoride concentrations in the groundwater of some villages in China were found to be more than 8 mg/l. In Canada fluoride levels vary from <0.05 to 0.2 mg/l in non-fluoridated up to 0.6-1.1 mg/l in fluoridated drinking water areas. Fluoride concentrations up to 3.3 mg/l were found in drinking water prepared from well water. Year-round averages for all drinking-water plants in the Netherlands are below 0.2 mg/l.
In areas rich in fluoride-containing minerals like some African countries, well water may contain about 10 mg/l fluoride; in Tanzania fluoride levels in drinking water are found to be 8 mg/l. The drinking-water standard for fluoride in Europe is 1.5 mg/l. The Environmental Protection Agency (EPA) uses a maximum contaminant level (MCL) of 4 mg/l in the USA. The WHO uses a guideline value of 1.5 mg/l for fluoride in drinking water; climatic conditions, volume of water consumed and intake from other sources should be considered when setting national standards.

CAUSES AND PATHWAYS

CAUSES: Intake of fluoride is equally divided between drinking water, food, other beverages and dental products like toothpaste. The total daily intake of fluoride is estimated to be between 5-7 mg per day in optimally fluoridated areas. Fluoride is ingested mainly via food; about 10% can be attributed to drinking water.
In Tanzania, where the soil is rich in fluoride-containing minerals, the daily exposure to fluoride may be as high as 30 mg/l for adults, mainly from drinking water intake.

PATHWAYS: Approximately 50% of the ingested fluoride will be absorbed in the gastrointestinal tract. The absorbed fluoride is transported via the blood and accumulates in teeth and bones. Excretion is mainly through the kidneys, but also via faeces and sweat.
High concentrations of fluoride of more than 2500 times the recommended dose can be fatal. Low concentrations of fluoride provide protection against dental caries, especially in children. The protective effect increases with concentrations up to about 2 mg/l.
Signs of fluoride toxicity include dental fluorisis by children during the tooth calcification period and skeletal fluorisis by mainly the elderly. Manifest dental fluorisis (dermatitis and mottling of the teeth as the enamel becomes infiltrated by yellow-brown staining) occurs at fluoride concentrations above 1.5-2.0 mg/l in drinking water. Skeletal fluorisis (stiff and painful joints, denser bones, causing adverse changes in the bone structure) is observed when drinking water contains 3-6 mg/l of fluoride. Crippling skeletal fluorisis occurs when the fluoride level in drinking water is more than 10 mg/l.
Osteoporosis and arthritis are also thought due to the fluoridation of drinking water; in central India fluoride-poisoned water from untested wells caused severe arthritic damage to millions of people.

Exposure to fluorides is known to lower the mental (intelligence) capacity of humans; children are especially susceptible to early fluoride toxicity. Children, the elderly and persons with renal impairment, are in the high risk group for fluoride poisoning.
In several studies a relationship has been found between bone and uterine cancer, and the fluoride concentration in drinking water, but there is not enough evidence to classify fluoride as a carcinogen.

Actions

In fluoridated areas, the addition of fluoride to the drinking water should be reduced or stopped. The following treatment technologies can effectively remove fluoride from drinking water:

- lime and alum coagulation
- gypsum and fluorite filters
- filtration by clay or bone material

A disadvantage of these relatively cheap methods for developing countries is that they do not achieve a fluoride reduction till the WHO guideline level. In areas where fluoride occurs naturally in the scarce sources it remains expensive to reduce the fluoride concentrations to save levels.
Prevention of high concentrations of fluoride in drinking water and the sources of drinking water is the best option.

Additional reading

WHO (1996)
Guidelines for drinking-water quality, Second edition, Volume 2
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Council Directive 98/83/EC, on the quality of water intended for human consumption, pb. L330,32

Frencken J.E. (ed)., Endemic fluorosis in developing countries, causes, effects and possible sollutions, 1992

Internetsites:
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http://www.biospectron.se/fluorine_human.html
http://www.bruha.com/fluoride/index.html

Lead

Lead and its compounds form one of the metals known since ancient times. Being the commonest of the heavy elements, it accounts for 13 mg/kg of the earth's crust. Lead dissolves from rocks and minerals into surface waters.
Lead is used in many different ways, such as in solders, pipes, alloys, cable sheathing, paint pigments, ammunition, ceramics, electronic devices and plastics. However, the most important use is in the production of batteries. Lead is also used as an anti-knock compound in petrol, but the introduction of lead-free petrol to reduce the atmospheric emission of lead, has almost completely phased out this use of lead in North America and western Europe, though not in eastern Europe or many developing countries. Lead is of universal importance in household plumbing materials like lead-soldered fittings and lead pipes in (especially older) water distribution systems; this is an important consideration from a drinking-water perspective. Lead can cause many different symptoms in humans depending on duration and level of exposure, and age. Especially young children and pregnant women are at risk.

Facts and trends

Facts: Although lead is rarely found in source water, lead mining and melting operations may be sources of contamination. Lead itself does not break down, but lead compounds can be changed by sunlight, air and water. Lead clings to soil particles and in water binds to sediments. Lead does not move from soil to groundwater or drinking water unless the water is acid or soft. Lead's presence in drinking water is, to some extent, due to the contamination of the source water entering the water system, but primarily to the corrosion of lead plumbing, by far the greatest cause for concern. PVC pipes also contain lead compounds that can be leached from them, resulting in high lead concentrations in drinking water. The amount of lead dissolved in the plumbing system depends on such factors as the presence of chloride and dissolved oxygen, the acidity, temperature, water hardness and standing time of the water. In the course of time, lead-containing materials in the plumbing system will usually develop a scale that minimises further corrosion of the pipes.
Lead and its compounds have water solubilities ranging from highly soluble to practically insoluble. Lead has no special taste or odour.

Trends: Lead is introduced into tap water (drinking water) as a result of its dissolution from natural sources, but primarily from household plumbing systems that contain lead (pipes, solders and fittings).
In 1989 the geometric mean of lead found in drinking water in the USA was 2.8 µg/l. In 1987 the median level of lead in drinking water samples from five

cities in Canada was found to be 2.0 µg/l. In 1992 the mean concentration of lead in drinking water consumed during a one-week sampling period in Ontario (Canada) was found to be in the range of 1.1-30.7 µg/l, with a median of 4.8 µg/l. In 1990 lead levels were above 50 µg/l in 10% of the households in the United Kingdom and 33% in Scotland.
In Europe the drinking water standard for lead is recently reduced from 50 µg/l to 10 µg/l. The introduction of programmes for renewing lead pipes to solve this problem have been in preparation for some years.
The Environmental Protection Agency (EPA) uses an 'action limit' for lead in drinking water of 15 µg/l in the USA; if lead is present above 15 µg/l in more than 10% of all homes tested by a water supplier, the system must continue to monitor this contaminant twice a year. If the lead levels are found to be consistently above this action level, treatment methods to reduce the lead concentration will be needed.
The WHO uses a guideline value of 10 µg/l for lead in drinking water. This value was established as the amount of lead a bottle-fed infant consuming 0.75 litres of drinking water per day can tolerate; since infants are the most sensitive subgroup of the population, this value would also provide protection for other age groups.

Causes and pathways

Causes: Exposure to lead takes place through contaminated water and drinking water, food, air and tobacco smoke. Lead is ingested by humans mainly via food and dust; about 15% of the total daily intake can be attributed to drinking water. Ingested lead via drinking water forms a relatively small proportion of the total daily intake of children and adults, but a significant one of bottle-fed infants. Since corrosion of plumbing systems is an important source of excessive lead in drinking water, lead levels in water should be measured at the tap rather than at the drinking-water source when estimating human exposure. It is not known, however, to what extent the general population flushes the system before using tap water; in addition, the stagnation time (and hence the lead level) is highly variable. If the concentrations of lead in dust and air decrease, the exposure via drinking water distributed by lead pipes relatively increases.

Pathways: About 10% of the ingested lead will be absorbed by the human body. Absorption of lead is increased when the dietary intake of calcium and phosphorus, but especially iron, is low. Lead binds to the haemoglobin in the red blood cells and is transported from the intestine to the liver, lungs, spleen, kidneys and bone marrow. From here more than 90% of the absorbed and transported lead is slowly transferred to the skeleton; the half-life of lead in the skeleton being approximately 17-27 years. A steady-state condition arises between the concentration of lead in blood and in various soft tissues like the kidneys and brain; the blood lead concentration can therefore be used as a reasonably good indicator of exposure to lead from all sources.
Inorganic lead is not metabolised in the body. Unabsorbed lead is eliminated in the faeces, absorbed but not retained lead is excreted unchanged via the kidneys or biliary tract.

Acute intoxication includes dullness, restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, hallucinations and loss of memory, with encephalopathy occurring at lead levels of 10-20 µg/l in adults and 8-10 µg/l in children.
Chronic exposure to lead can cause fatigue, sleeplessness, irritability, headaches, joint pains and gastrointestinal symptoms; all effects may appear in adults at blood lead levels of 5-8 µg/l. Prolonged exposure to increased lead levels can also cause stroke, kidney disease, aggressive behaviour, hyperactivity and learning problems. In adults, increased lead levels have been linked to high blood pressure and damaged hearing. Serious lead poisoning can lead to blindness, brain damage and mental retardation. The biosynthesis of haem and the functions of the kidneys central nervous system have been found to be by far the most sensitive biological functions to long-term exposure at relatively high levels of lead. Lead interferes with the activity involving the biosynthesis of haem, causing anaemia.
A number of studies have been carried out to investigate the effects of exposure of young children to lead on their intellectual abilities and behaviour. Some of these studies have found a significant association between blood lead levels of 3 µg/l or more and IQ deficits of about four points. Young children, infants and pregnant women are particularly vulnerable to unsafe lead levels, which can emerge and impair a child's mental and physical development, reduce a baby's birth weight and cause premature birth.
Some studies found that lead may decrease fertility in both males and females. Lead is a probable teratogen in humans. There is inadequate evidence to clearly determine lead's carcinogenicity in humans, although an association between the ingestion of lead salts and renal tumours has been demonstrated experimentally.

Actions

The level of lead in drinking water can be reduced by corrosion-control measures such as the addition of lime and the adjustment of the pH in the distribution system from <7 to 9.
The following treatment technologies can effectively remove lead from drinking water:

- corrosion control
- coagulation/filtration
- lime softening

Prevention of high concentrations of lead in drinking water and the sources of drinking water, and corrosion control are the best options. Some of the ways available to reduce the concentration of lead in drinking water are listed below:

- flushing the cold water system before using the water for drinking or cooking since water standing in a pipe for several hours is more likely to contain lead than flowing water;
- using cold water when cooking and preparing baby formula; hot water can dissolve lead more quickly than cold water.
  The removal of the plumbing and fittings containing lead is, however, by far, the best way to reduce the lead concentration in drinking water.

Additional reading

WHO (1996)
Guidelines for drinking water quality,
Health criteria and other supporting information, 2nd ed., vol. 2. World Health Organisation, Geneva, 1996

Versteegh J.F.M. et al. Waterleidingbesluit 2000,
Voorstel voor implementatie van de EG-Drinkwaterrichtlijn; onderdeel kwaliteitseisen. RIVM, Bilthoven, 1999. Report 734301018

Council Directive 98/83/EC, on the quality of water intended for human consumption, pb. L330,32

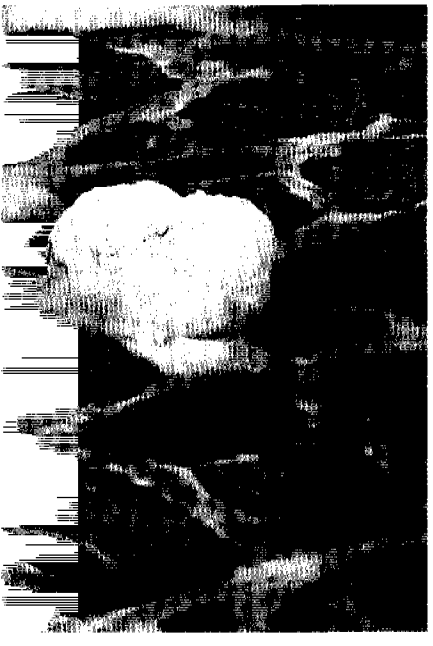
Internetsites:
http://mail.odsnet.com/TRIFacts/197.html
http://www.epa.gov/OGWDW/dwh/c-ioc/lead.html
http://www.atsdr.cdc.gov/tfacts13.html
http://www.everpure.com/issues/iowq009.html
http://www.everpure.com/issues/iowq003.html

8 MACROCONTAMINANTS

EXAMPLES ARE:

NITRATE/NITRITE AND SULPHATE

Nitrate and nitrite



Nitrate and nitrite are naturally occurring ions that are part of the nitrogen cycle. Nitrate (NO_3) is the stable form of nitrogen for oxygenated systems; nitrite (NO_2) contains nitrogen in a relatively unstable oxidation state.
Nitrate is used mainly in inorganic fertilisers. It also serves as an oxidising agent and is used in the production of explosives and purified potassium nitrate for glass-making. Sodium nitrite is used as a food preservative, especially in cured meats.
In the human body, nitrates are partly converted into nitrites. High levels of nitrate in drinking water together with diarrhoea can cause methaemoglobinaemia ("blue-baby syndrome") in bottle-fed infants.

Facts and trends

Facts: The most prominent use of nitrates is in fertiliser. In the soil, fertilisers containing inorganic nitrogen and wastes containing organic nitrogen are converted to ammonium or nitrate. Some nitrogen fertilisers contain nitrogen already in the form of nitrate, while in other fertilisers, nitrogen is present in the form of ammonium, which is rapidly converted to nitrate. Nitrogen is necessary for plant growth. When the nitrate supply is greater than the amount used by plants, potential accumulation of nitrates in the soil and leaching from the system into the groundwater occurs.
Nitrate can enter groundwater from the natural ecosystem, agricultural runoff, runoff from lawns and gardens, intensely fertilised agricultural areas, livestock facilities, livestock manure and human sewage. Primary sources of organic nitrates include human sewage and livestock manure. The primary inorganic nitrates and potential contaminants of drinking water are potassium nitrate and ammonium nitrate, both of which are widely used as fertilisers. Leaching of nitrate is of primary concern in the contamination of groundwater.
Since nitrate and nitrite are very soluble in water and do not bind to soil, nitrates have a high potential to migrate to groundwater. Nitrates can be denitrified by denitrifying bacteria and certain chemical compounds present in the underground.

Trends: The natural nitrate concentration in groundwater under aerobic conditions is a few milligrams per litre and depends strongly on soil type and the geological situation. Under aerobic conditions, (surplus) nitrate percolates in large quantities into the aquifer because of the small extent to which degradation or denitrification occurs. Under anaerobic conditions, nitrate may be denitrified or degraded almost completely to nitrogen. In the USA natural nitrate levels do not exceed 4-9 mg/l and nitrite levels, 0.3 mg/l. As a result of agricultural activities, the nitrate concentrations can easily reach several hundred milligrams per litre; concentrations

up to 1500 mg/l have been found in one of the agricultural areas in India.
The nitrate concentration in surface water is normally low (0-18 mg/l). It can, however, reach high levels as a result of agricultural runoff, runoff from refuse dumps, or contamination with human or animal wastes.
Due to the increasing use of artificial fertilisers, the disposal of wastes (particularly from livestock farming) and changes in land use, nitrate concentrations have increased over the last 20 years, in surface water but also especially in groundwater. In most countries, nitrate levels in drinking water derived from surface water do not exceed 10 mg/l. However, it has been found that in 15 European countries, 0.5 to 10% of the population is exposed to nitrate levels in drinking water of more than 50 mg/l. And in agricultural areas throughout the world, individual wells are especially vulnerable and nitrate concentrations in the water often exceed 50 mg/l.
The standard in Europe for nitrate in drinking water is 50 mg/l. For nitrite in drinking water, this is 0.1 mg/l; the standard for nitrite levels in tap water after distribution is 0.5 mg/l.
The WHO uses a guideline value for nitrate in drinking water of 50 mg/l.
A provisional guideline value of 3 mg/l has been proposed for nitrite in drinking water; because of the possibility that nitrate and nitrite will occur simultaneously in drinking water, the sum of the ratios of concentration to a guideline value for each should not exceed 1: i.e. [nitrate]/50 + [nitrite]/3 = 1.

Causes and pathways

Causes: Exposure to nitrate and nitrite can take place through water and drinking water, as well as food (especially vegetables) and air. Where nitrate levels in drinking water exceed 50 mg/l, drinking water will be the major source of total nitrate intake, especially for bottle-fed infants. The mean dietary intake of nitrate has been determined in the range of 43 to 131 mg per day; for nitrite this is 1.2 to 3 mg per day. Estimates of the total nitrate intake range from 39 to 268 mg per day, the higher values applying to vegetarian and nitrate-rich diets. Babies consume large quantities of water in proportion to their body weights, especially when water is used to mix powdered or concentrated formulas. Daily intake from formula made with water containing nitrate at 50 mg/l water would average about 8.3-8.5 mg of nitrate per kg body weight per day.

Pathways: Ingested nitrate is readily and completely absorbed from the upper small intestine. Nitrite may be absorbed directly both from the stomach and the upper small intestine. Nitrate is rapidly distributed throughout the tissues. Approximately 25% of the ingested nitrate is actively secreted into saliva, where it is partly (20%) reduced to nitrite by the oral microflora; bacterial reduction of nitrate may also take place in other parts of the human gastrointestinal tract. Absorbed nitrite is rapidly oxidised to nitrate in the blood. Nitrite in the bloodstream is involved in the oxidation of haemoglobin (oxygen-carrying red blood pigment) to methaemoglobin. Nitrate is excreted in the urine as nitrate, ammonia or urea, faecel excretion being negligible. Only a bit of nitrite is excreted.
Short-term exposure to excessive levels of nitrate in drinking water can cause serious illness and sometimes death. The toxicity of nitrate to humans is thought to be solely the consequence of its reduction to nitrite. Especially infants of less than six months of age

are the most affected by excess nitrate levels in drinking water used to prepare formulas. Serious illness in infants is due to the conversion of nitrate to nitrite by the body, which can interfere with the oxygen-carrying capacity of the child's blood: i.e. haemoglobin is oxidised to methaemoglobin, which is unable to transport oxygen to the tissues. This condition is called methaemoglobinaemia and causes shortness of breath and blueness of the skin ("blue baby syndrome"), cyanosis and, at higher concentrations, asphyxia. Children with diarrhoea are especially at risk for this syndrome. It is known that infantile methaemoglobinaemia does not occur at drinking-water levels of 50 mg nitrate per litre or less.
Chronic exposure to relatively high levels of nitrates and nitrites can cause effects like diuresis, increased starchy deposits and haemorrhaging of the spleen.
Nitrate, and especially nitrite, cause a possible increased risk of cancer in humans due to the reaction of nitrite with nitro-stable compounds in the stomach to form *N*-nitroso compounds. The association between dietary nitrate and cancer, however, is insufficiently proven.

Actions

Contamination of a water supply with nitrate is very difficult and costly to treat. Nitrate and nitrite can be effectively removed by the following drinking-water treatment technologies:

- ion exchange
- reverse osmosis
- electrodialysis

Additional actions, such as providing an alternative drinking-water supply, may be required to prevent serious risk to public health. When nitrate levels are above 50 mg/l for bottle-fed infants, it is advised to use water with a low nitrate level.

Additional reading

WHO (1996)
Guidelines for drinking water quality,
Health criteria and other supporting information, 2nd ed., vol. 2. World Health Organisation, Geneva 1996

Versteegh J.F.M. et al. Waterleidingbesluit 2000, Voorstel voor implementatie van de EG-Drinkwaterrichtlijn; onderdeel kwaliteitseisen. RIVM, Bilthoven, 1999. Report 734301018

Council Directive 98/83/EC, on the quality of water intended for human consumption, pb. L330,32

Internetsites:
http://www.epa.gov/OGWDW/dwh/c-ioc/nitrates.html
http://www.ces.uga.edu/pubcd/c819-5W.html

9 Concluding remarks and actions

Unsafe drinking water, poor sanitation and a shortage of water form by far the biggest health threat especially in developing countries all over the world. In over 50 countries the domestic water consumption is below a Basic Water Requirement level of 50 liters per capita per day, with 26 countries below 20 and 6 even below 10 liters per capita per day. The Basic Water Requirement is considered to be 5 liters per capita per day for drinking, 15 for bathing, 10 for cooking and 20 for sanitation.

In general it is recommended to treat all drinking water, except when using high-quality groundwater sources. Universally, drinking water suppliers adhere to drinking water quality standards, mostly the WHO standards. In situations where treatment is not feasible owing to financial or maintenance constraints a choice must be made between supplying water without treatment or abandoning the water supply. Abandoning should be considered only when the rationalised health risks outweigh the rationalised benefits.

For each category of disease related to water and sanitation the most relevant actions are summarised below.

Waterborne diseases

- adequate, safe supplies of water for drinking, household purposes and bathing by filtration, sedimentation or disinfection employing f.i. chlorination and iodinisation;
- disposing human faeces in a hygienic manner;
- protecting food supplies from contamination caused by use of human excreta as fertiliser and by infected food handlers, filth flies or cockroaches;
- thorough washing with potable water and keeping fruit/vegetables dry; disinfectant dips for fruit and vegetables are of unproved value in preventing transmission;
- if vaccines are available mass immunisation is a very important tool for breaking virus transmission.

Waterwashed diseases

- trachoma and related blindness can be prevented by the implementation of the SAFE strategy (Surgery, Antibiotics, Facial cleanliness and Environmental improvement). This strategy is based on a combination of community-targeted public health interventions. Recently a new drug, azithromycin, has been very promising in field trials.

Waterbased diseases

- for Legionellosis the best way to prevent growth of the micro-organism is keeping the water below the preferable 15°C and flushing, or keeping it hot (at least above 55°C) and flushing;
- for Dracunculiasis (guinea worm) actions are:
  - an integrated community-based surveillance system including health education;
  - intensified case-containment measures;
  - provision of safe water, including appropriate water supply systems, filtering devices and the chemical treatment of water to eliminate the vector (cyclops);
  - monitoring the dracunculiasis situation through the use of geographical information systems (GIS).

Waterrelated diseases

- adequate, safe supplies of water for drinking, household purposes and bathing by techniques as soil passage, (membrane) filtration, sedimentation or disinfection employing f.i. chlorination, ozonisation and iodinisation;
- for diseases transmitted by a vector (f.i. a mosquito) the vector has to be combatted by good water storage practices and proper waste disposal;
- regular monitoring and surveillance of the natural mosquito population should accompany control efforts so as to determine the impact of the measures e.g. insecticides;
- prevention of mosquito bites by using impregnated bed nets, insect repellents and protective clothing;
- vaccination if there is a vaccine available.

Poor sanitation

- improve domestic hygiene, particularly in relation to children, can be an effective means of control.

Metals and nitrate/nitrite

- prevention of the source if possible;
- corrosion control and the removal of lead pipes;
- water treatment techniques as acidification, coagulation and filtration, ion exchange;
- in house filtration techniques using local filter materials especially for fluoride in countries with lack of alternative water sources.

In general there are solutions for many of the diseases caused by unsafe water and poor sanitation, however support in different ways from both developing and developed counties worldwide is needed.

Core tasks RIVM

The National Institute of Public Health and the Environment is a research institute for policy support relating to public health and the environment. It also serves as the government centre of expertise, incorporating a policy assessment function for both the environment and nature conservation.

Core tasks, as laid down by legislation are:

a. to conduct research geared to policy support and supervision in the fields of public health, the environment and nature conservation;
b. to publish periodic reports on the current status and future trends of public health and the environment;
c. to carry out any other activities that may be required by the Minister.

The RIVM is professionally autonomous in carrying out these tasks. Other activities untertaken by the RIVM may not impinge upon the tasks explicitly assigned to it by legislation.

Besides its core tasks, the RIVM carries out self-supporting activities commissioned by both public authorities and international organisations (e.g. WHO, EU, ECE, UNEP, UNICEF and the World Bank), providing these activities can be carried out in synergy with core activities and/or are statutorily assigned to the institute. Included here are assignments pertaining to vaccines, the assessment of existing substances and products, and the provision of information to healthcare and environmental management professionals.

The RIVM is accountable to the Minister of Health, Welfare and Sport. RIVM's products (information, advice, measurement data, research reports, status reports, long-range forecasts, journal articles, vaccines) find their primary use in the support and implementation of government policy on public health, the environment and nature conservation. The RIVM comprises approximately 30 laboratories and centres divided into four divisions. The divisions work closely together on an project basis. Their main activities are described in our brochure.

National Institute for Public Health and the Environment (RIVM)

Calling address: Antonie van Leeuwenhoeklaan 9
Postal address: P.O. Box 1, 3720 BA Bilthoven, The Netherlands

Telephone: +31 30 274 91 11
Telefax: +31 30 274 29 71
e-mail: info@rivm.nl
www.rivm.nl

Colophon

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www.minvrom.nl/water

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