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INTEGRATED CRITERIA DOCUMENT ARSENIC

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Mailing list Integrated criteria document Arsenic

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SUMMARY

This document contains data on arsenic with regard to sources and distribution, the risks based on a consideration of exposure levels and harmful concentrations and the technical possibilities to reduce these risks.

Arsenic is a metalloid. With regard to its properties it belongs to the group of elements between the (heavy) metals and the non-metals. Arsenic occurs naturally; the natural sources are sometimes considerably greater than the anthropogenic ones. In the Netherlands arsenic is mainly applied in wood preservation and also as a softening agent in PVC, in alloys and in drugs.

The arsenic load in soil, surface water and air has decreased in the past few years. Since 1980 this decrease has amounted to approximately 70% for the agricultural land where arsenic was previously used as a pesticide. At present roughly 50% of the emission to the soil takes place by deposition, the extent of which is mainly determined by sources abroad. Other substantial sources are manure and chemical fertilizer (farmland), pellets of shot and purification sludge (remaining land). In the big rivers the arsenic contents have decreased by over 50%, particularly as a result of reduced emissions abroad. The transfrontier emissions (mainly natural) amount to approximately 200 tonnes. The total emission in the Netherlands at present amounts to approximately 17 tonnes, 50% of which is discharged in the New Waterway (fertilizer industry) and is as such little damaging to the fresh surface water. In the Netherlands the emissions to the air have decreased by approximately 80% through the application of dustfilters and amount to well over 1 tonne. This load is also relatively small, considering the estimated transfrontier emissions which amount to approximately 24 tonnes maximally.

A global overview of the fluxes in and through the Dutch environment is shown in figure A. The accumulation in the soil is estimated at approximately 10 tonnes per year; in the sediment at approximately 12 tonnes per year.

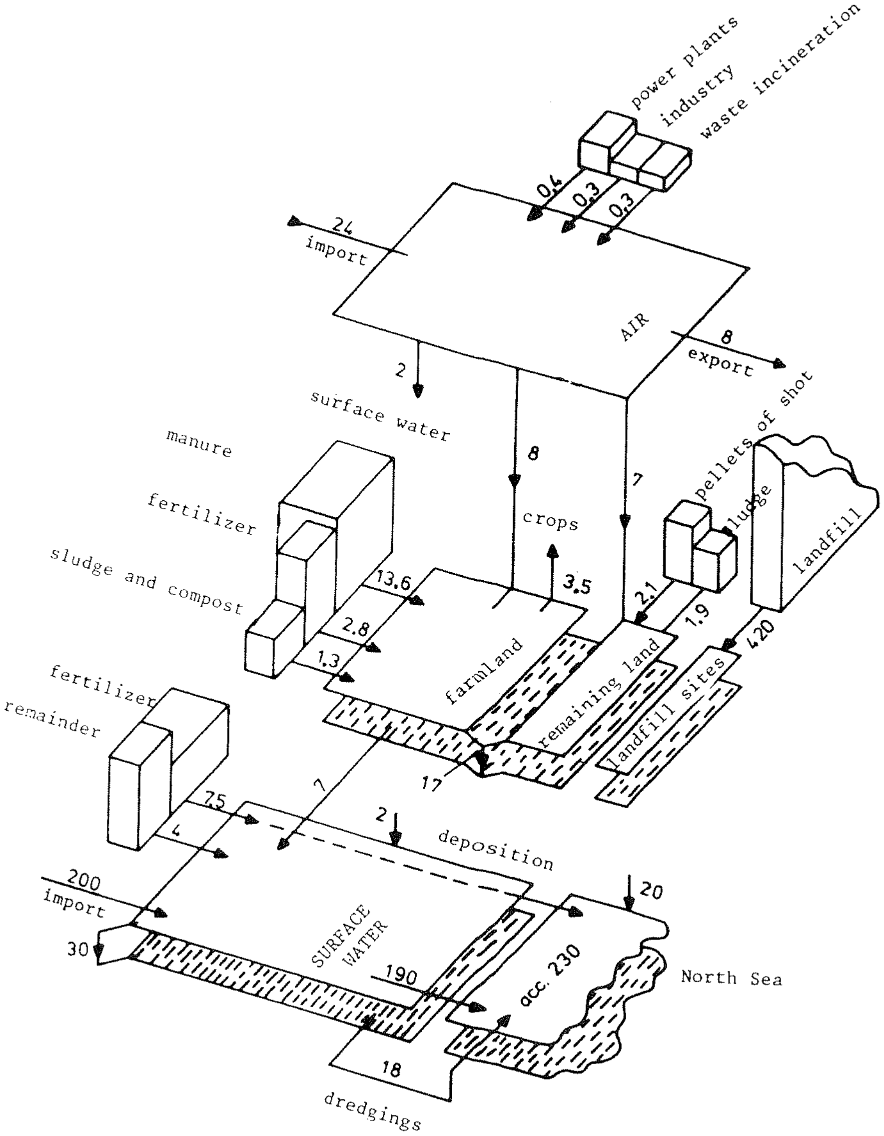


Figure A *Schematic overview of the annual arsenic emissions in the Netherlands, the contributions from abroad and the fluxes and reservoirs of arsenic in the Dutch environment (in tonnes annually; for further details see figure 4.6, page 66 et seq.)*

With regard to the risks for man it should be noted that no international consensus has (yet) been reached. The EPA considers arsenic as a carcinogen without a threshold value, the WHO assumes a threshold value for oral exposure (increases the risk of skin cancer), but not for inhalatory exposure (increases the risk of lung cancer). In this report with respect to the oral exposure the WHO approach is adopted. For the inhalatory exposure, however, the information is considered insufficient to determine a well-founded threshold level.

Based on epidemiological studies, in which exposure took place through drinking water, WHO has arrived at a provisional maximally permissible oral dose of 2 μg of inorganic arsenic per kg body weight. The application of a lower daily drinking water consumption and a lower body weight, as is common practice in the National Institute of Public Health and Environmental Protection, results in a slightly higher maximally permissible dose: 2.5 μg inorganic arsenic per kg body weight. This quantity may not be considered as an ADI for the reason that after long-term daily intake of this dose the probability of effects cannot be ignored. For organic arsenic compounds it is assumed that the toxicity is at least a factor 10 lower. The current contamination of food and drinking water with arsenic in the Netherlands is therefore far below the maximally permissible dose.

The toxicological recommended value for inhalatory exposure is based on the (toxic) effects observed in occupationally exposed groups. Assuming a concentration at which marginal effects were still observed in the working environment, and using an extrapolation factor of 100, a concentration of 0.5 $\mu g.m^{-3}$ is derived, which is expected to cause no effects in the general population. The large-scale annual average arsenic concentrations in the Netherlands are more than a factor 100 below this and near sources a factor 25 or more.

Adverse effects on human health caused by the current oral and inhalatory exposure in the Netherlands are therefore not likely to occur.

For the protection of the aquatic environment (fresh water and seawater) a toxicological risk limit of 6 $\mu g.l^{-1}$ (dissolved inorganic arsenic) was determined in accordance with the procedure of the Health Council. The concentrations in Dutch surface water have decreased in the past few years to a level of 1 - 3 $\mu g.l^{-1}$. Adverse effects on the aquatic ecosystems may therefore be ruled out, possibly with the exception of the immediate vicinity of discharge points.

With regard to the terrestrial environment insufficient data are available to arrive at a well-founded recommended value. Following the addition of 30 mg $As.kg^{-1}$ dry matter to sandy soil growth inhibition was still observed in one wild plant species. The highest concentration at which no effect was observed (wheat, sandy soil) is 20 mg $As.kg^{-1}$ d.m. (background- and added concentration). In most soils these toxicological limits are not exceeded. Although the contents in river clay of the Rhine and the Meuse (30 - 70 mg $As.kg^{-1}$ d.m.) and in harbour sludge polders (35 - 135 mg $As.kg^{-1}$ d.m.) are higher, the biological availability of arsenic in these soils is lower. High contents occur naturally in areas rich in iron (80 - 390 mg $As.kg^{-1}$ d.m.). It is assumed that the elevation of the arsenic content in the soil in the Netherlands through anthropogenic sources does in general not give rise to the occurrence of unacceptable effects neither on terrestrial ecosystems nor on agricultural crops and livestock.

It may be concluded that the risks connected with the current and future arsenic concentrations in the environment of the Netherlands are in general limited. The current standards and guidelines which for soil are at the same level (29 $mg.kg^{-1}$ d.m. for standard soils) and for water (12.5 $\mu g.l^{-1}$ dissolved) higher than the values deduced in this report, are generally not exceeded. It is suggested to change the quality requirement for soil and to lower that for surface water. Besides it should be noted, however, that the concentrations in the Dutch environment are to a high degree determined by the natural background and/or by emissions abroad. The effects of positive autonomous developments and (possible) measures on a national scale on the concentration levels in the Netherlands are therefore extremely limited.

INTRODUCTION

On a national level the environmental policy is in the first place aimed at achieving and maintaining an environmental quality which guarantees the health and well-being of people and the preservation of plants and animals as well as a sustainable social development (Nationaal Milieubeleidsplan, 1989). Adequate knowledge lacking it will, however, not be possible for some time to fully define the general environmental quality aimed at. Therefore, attention is first concentrated on factors which may present great risks such as substances hazardous to the environment. A selection has been made of the many substances that are important because of emission or usage and a priority list has been drawn up. For most priority substances Integrated Criteria Documents are written.

Arranged by substances or groups of substances Integrated Criteria Documents contain data on the sources and the distribution pattern (soil, water, air, biota), the riks of current and future concentrations for man, (parts of) ecosystems and materials, as well as the technical possibilities and the business economic consequences of reducing these risks. This information serves as a scientific basis for the formulation of the effect-oriented policy. This policy is aimed at achieving as great a risk reduction as possible, the ultimate goal being the desirable level. This value is the concentraton in the environment at which no adverse effects (the risks of which may be considered negligible) occur in man, plants, animals and materials. If the desirable level cannot be realized within a reasonable period of time, a tolerable level will be determined for a limited period, the risks ranging from the maximally permissible to the desirable level. In determining the tolerable level also economic and social factors may be decisive. This document is restricted to providing the information necessary for the policy-oriented determination of environmental quality requirements mentioned. The information supplied may also result in general terms of reference for the emission reduction per kind of source. No policy statement, therefore, is mentioned in any section of this document.

Integrated Criteria Documents are drawn up under the auspices of the National Institute of Public Health and Environmental Protection (RIVM). The Institute for Soil Fertility, the Hydrodynamic Laboratory and the firm of consulting engineers HDV contributed to the realization of this document. Support in the preparation (such as scoping meeting, hearing prior to the working document, sources of information) was rendered by the government, industry, environmental groups and representatives of scientific institutes. The content of this document has been (integrally) checked by a Review Committee of the RIVM, whereas a Counselling group composed of staff from the Ministry for Housing, Physical Planning and the Environment, the Department of Inland Waterways/National Institute for Wastewater Research (DBW/RIZA) and the Ministry of Agriculture and Fisheries. The industry supplied important, partly confidential, information through the ad hoc Working Group on Integrated Criteria Documents of the office of Environment and Physical Planning of the Council of Dutch Employers' Unions, VNO and NCW. In the event of differences of opinion an addendum drawn up by the ad hoc Working Group may be added to the document. This is also possible for environmental groups through the Foundation Nature and the Environment. At a later stage the Health Council will advise in brief on the document, including any possible addenda.

In this document arsenic is discussed. In view of the problematic nature and the gaps in knowledge special attention is given to the sources and emissions, speciation and accumulation, the human toxicity and the ecotoxicity. The effects on agricultural crops and livestock will be touched upon. In complience with the wishes of the commisioning body, recent outline articles were used as far as possible with regard to the impact elements. Although the original literature was only consulted in the case of inconsistent data or conclusions in the outline articles, it was used as a basis for deriving a toxicological recommended limit value. Furthermore, articles were evaluated of a more recent date than the outline articles consulted.

1. PROPERTIES AND EXISTING STANDARDS

1.1. PROPERTIES

1.1.1. Occurrence

Arsenic (symbol As, atomic number 33, molecular weight 74.92 and valancies -3, +3 and +5) occurs naturally in various compartments, usually in compounds with sulfur or other metals (copper, cobalt, lead, zinc etc.) and in methylated compounds (di- and tri-methylarsine) as a result of biological transformation in the soil.

Elementary arsenic may be found in various allotropic forms. The yellow and the grey (metallic) forms occur most frequently. The most important naturally occurring arsenic-containing compounds (minerals) are arsenopyrite (FeSAs), copper-, nickel- or iron-arsine and arsenic sulphide and arsenic oxide. The main commercial product is arsenic(III)oxide (As_2O_3 or As_4O_6). In air arsenic mainly occurs in the inorganic form (WHO, 1981; CCRX, 1988).

1.1.2. CAS-registration numbers

Table 1.1. Molecular formulas and CAS-registraton numbers of the main arsenic compounds

Compound	*Molecular formula*	*CAS-reg.number*
Trivalent inorganic arsenic		
Arsine	AsH_3	*7784-42- 1*
Arsenic(III)oxide	As_2O_3	*1327-53- 3*
Arsenious acid		*13464-58- 9*
Salts of arsenious acid		*13768-07-05*
Arsenic(III)chloride	$AsCl_3$	*7784-34- 1*
Arsenic(III)sulphide	As_2S_3	*1303-33- 9*
Sodium arsenite	$NaAsO_2$	*7784-46- 5*
Pentavalent inorganic arsenic		
Arsenic(V)oxide	As_2O_5	*1303-28- 2*
Arsenic acid		*7778-39- 4*
Salts of arsenic acid		*10102-53- 1*
Sodium arsenate	NaH_2AsO_4	
	Na_2HAsO_4	*7778-43- 0*
	Na_3AsO_4	*7631-89- 2*
Lead arsenate	$Pb_3(AsO_4)_2$	*3687-31- 8*
Iron arsenate	$FeAsO_4$	*38296-95- 6*

Table 1.1. continued

Aluminium arsenate	$AlAsO_4$	*60112-04- 1*
Calcium arsenate	$Ca_3(AsO_4)_2$	*89067-81- 2*
<u>Organic arsenic</u>		
Sodium cacodylate	$(CH_3)_2AsO(NaO)$	
Methyl arsonic acid		*124-58- 3*
Sodium methyl arsonic acid	$CH_3AsO(OH)Na$	
Di-sodium methyl arsenate		
Sodium dimethyl arsenate		
Dimethyl arsenic acid		*75-60- 5*
Trimethyl arsenic oxide		*4964-14- 1*
Methyl arsine		*593-52- 2*
Dimethyl arsine		*593-57- 7*
Trimethyl arsine		*593-88- 4*
Arsanilic acid		*98-50- 0*
Arseno betaine		
Arseno choline		

1.1.3. Physico-chemical properties

Arsenic is a metalloid, which with regard to its properties belongs to the group of elements between the heavy metals and the non-metals. It can occur in four oxidation states as arsine (-3), arsenic, arsenite (+3) and arsenate (+5).

When heating arseno pyrite the arsenic sublimes leaving iron sulphide (FeS). When heating elementary arsenic oxidation occurs, producing arsenic oxide (As_2O_3), which readily dissolves in hydrochloric acid or in lye. In oxygenated water arsenic usually occurs as arsenate and under reducing conditions, such as in still deep waters, as arsenite. The boiling point of As_2O_3 is 465 °C, the vapour pressure at 25 °C amounts to 0.6 $\mu g.m^{-3}$ and the solubility at 25 and 98 °C amounts to 20 and 82 $g.l^{-1}$, respectively. Arsenic and arsenic(III)oxide sublime at relatively low temperatures, 613 and 193 °C, respectively.

Alkali earth metals combine with arsenate anions to form salts that are only slightly soluble. Arsenic tends to form a precipitate frequently in association with phosphates.

Reported pKa values for arsenious and arsenic acids are: $HAsO_2$ pKa 9.23, H_3AsO_4 pKa_1 2.20, pKa_2 6.97 and pKa_3 11.53. Arsenates and arsenic acid are strong oxidants.

Lead arsenate, copper arsenate, copper(II)acetate meta-arsenate (Paris Green) and calcium arsenate are compounds which have been used as insecticides and are only slightly soluble in water (EPA, 1980; CCRX, 1988).

The solubilities of the arsenic compounds are shown in table 1.2.

Table 1.2. Solubilities of arsenic compounds

Arsenic compound	*Molecular formula*	*Solubility (g/l)*
Sodium arsenite	$NaAsO_2$	*highly soluble*
Arsenic trioxide	As_2O_3	*37*
Arsenic sulphide	As_2S_3	*0.0005*
Sodium arsenate	Na_3AsO_4	*389*
	$Na_2HAsO_4.7H_2O$	*55*
	NaH_2AsO_4	*soluble*
Lead arsenate	$Pb_3(AsO_4)_2$	*disintegrates*
Aluminium arsenate	$AlAsO_4.8H_2O$	*insoluble*
Calcium arsenate	$Ca_3(AsO_4)_2$	*0.13*
Arsenic pentoxide	As_2O_5	*1500*

1.2. EXISTING STANDARDS AND GUIDELINES

1.2.1. Soil and groundwater

In the Environmental Programme Progress Reports 1988 - 1991 (MPV88, 1987) the reference values for arsenic in soil and groundwater are 15+0.4(L+H) $mg.kg^{-1}$ dry matter and 10 $\mu g.l^{-1}$, respectively, L being the weight percentage lutum (<2 μm) and H the percentage organic matter. For a standard soil containing 10% organic matter and 25% lutum the reference value is, therefore, 29 $mg.kg^{-1}$ dry matter. According to current views, at this value or lower ones, the soil may in general be considered multi-functional, meaning that no adverse effects from arsenic are to be expected. In assessing the soil quality the technical guide yet to be published need to be used. For the application of purified soil, without any restrictions, the same value is maintained in the draft working paper Standard Application Purified Soil (DGM, 1988).

For the assessment of the concentration levels in the soil a testing framework has been presented (table 1.3.) in a draft Guideline on Soil Sanitation (VROM, 1987). The values mentioned in this guideline should not be considered as standards but as an assessment framework (trigger values). At a high lutum and organic matter content the A-value may be higher than the B-value.

Table 1.3. Testing table for the assessment of the concentration levels of arsenic in the soil

	A	B	C
Soil (mg per kg dry matter)	*15+0.4(L+H)*	*30*	*50*
Groundwater (µg per liter)	*10*	*30*	*100*

L = weight percentage lutum of the soil
H = weight percentage organic matter of the soil
A = reference value (standard soil 29 mg/kg dry matter), equals MPV88-value
B = trigger value for the purpose of further studies
C = trigger value for the purpose of rehabilitation studies

- Purification sludge, compost and black soil

The quality of animal and other organic fertilizers are currently controlled under the Fertilizer Decree and their use under the Soil Protection Decree. According to the "Draft resolution quality other organic fertilizers" (draft 27 October 1988) the standards for the purification sludge, compost and black soil will be tightened in phases the following years (table 1.4.).

Table 1.4. Standards for arsenic with respect to the quality of other organic fertilizers (mg/kg dry matter)

Fertilizer	*Period*	
	up to 1/1/91	*1/1/91 - 1/1/95*
Purification sludge	*25*	*25*
Compost	*25*	*25*
Black soil	*15 + 0.4(L + H)*	

L = % lutum
H = % organic matter, in the calculation this percentage should not be higher than 15

Application of purification sludge or compost is only allowed if (compulsory) soil studies show that the reference values for heavy metals and arsenic are not exceeded. Exception to this rule is the application of "clean" compost from vegetable waste which is considered as recycling and does not require soil studies. The standards for the use of purification sludge, compost and black soil are summarized in table 1.5.

Table 1.5. Standards for the use of other organic fertilizers (tonnes dry matter/ha)

Soil usage	Purification sludge		Compost			Black soil
	liquid	solid	sludge only	mixed waste	vegetable waste only	
Grassland	1 t/yr	prohibited	prohibited	prohibited	unrestricted	unrestricted
Agricultural land and green maize land	2 t/yr	4 t/2 yrs	4 t/2 yrs	12 t/4 yrs	unrestricted	unrestricted
Nature reserve	prohibited	prohibited	prohibited	prohibited	unrestricted	unrestricted
Remaining land	prohibited	prohibited	4 t/2 yrs	12 t/4 yrs	unrestricted	unrestricted

The issue will be further elaborated and final regulations are expected to be introduced in 1990. Considerable changes may occur, also based on this integrated criteria document.

1.2.2. Water and sediment

Surface water for the preparation of drinking water may contain 20 $\mu g.l^{-1}$ arsenic maximally (Staatsblad 606, 1983).
In the IMP-Water 1985-1989 (1984) the standard for surface water (basic quality) is 50 $\mu g.l^{-1}$ arsenic (total).
In the MPV-89 (1988) the formulation of recommended standards for sediments is initiated, point of departure being that the general environmental quality sediments and the reference value soil quality in principle indicate an identical protection level. For the general environmental quality of sediment the arsenic content may amount to 29 $mg.kg^{-1}$ maximally. The provisional C-value for sediment, the value above which studies into the necessity of sanitation is imperative, is determined at 100 mg $As.kg^{-1}$ and the provisional trigger value is 45 mg $As.kg^{-1}$.

In the Third White Paper Water Management (1989) the general environmental quality (quality objective 2000) for water and sediment and the trigger value and signal value for sediment are shown. In measuring and testing the general environmental quality a distinction is made between compounds on the M-list and those on the I-list. The M-list contains the most relevant problem compounds, the contents of which require frequent measuring. The general environmental quality of water and sediment is 15 $\mu g.l^{-1}$ (I-list) and 85 $mg.kg^{-1}$ (I-list), respectively. The provisional trigger value and signal value for sediment are 85 and 150 $mg.kg^{-1}$, respectively.

- European Community

In the EC (1975) surface water meant for the preparation of drinking water has to meet the standards shown in table 1.6.

The EC (1979) regulates for the required quality of shellfish water that the arsenic concentration be limited thus that it contributes to a good quality of shellfish products.

Table 1.6. EC standards (mg/l) for arsenic in surface water meant for the preparation of drinking water

Kind of purification	*Guideline*	*Limit value*
Simple treatment and disinfection	*0.01*	*0.05*
Normal physical and chemical treatment and disinfection		*0.05*
Thorough treatment, refinery and disinfection	*0.05*	*0.1*

1.2.3. Air

The quality requirements at the workplace and for outdoor air are given in tables 1.7 and 1.8, respectively (IDC, 1985).

- Germany

Emission limit values have been determined for arsenic, arsenic compounds, arsenic trioxide, arsenic pentoxide, arsenic acid and arsenic acid salts, arsenious acid and arsenious acid salts, as As. At a mass flow of more than 5 $g.hour^{-1}$ the emission concentration may amount to 1 $mg.m^{-3}$ maximally and at a mass flow of more than 25 g.hour^{-1} continuous measuring of the emission concentration is required (TA-Luft, 1986).

Table 1.7. *Air quality at the workplace [MAC-values, time weighted average (TWA) 8 hours per day, a total of 40 hours per week] in mg.m*$^{-3}$

Country	*MAC-values*	*Remarks*	*Reference*
The Netherlands	*0.2*	*arsine*	*National MAC-list (1989)*
	0.05	*arsenic and -compounds (as As): combination of all inorganic compounds*	
	0.25	*water soluble inorganic compounds*	
FRG	*0 (?)*	*arsenic and compounds (as As)*	*SCMO/TNO (1977)*
	0.2	*arsine*	
GDR	*0.3*	*arsenic and compounds (as As)*	*SCMO/TNO (1977)*
	0.2	*arsine*	
Sweden	*0.05*	*arsenic and compounds (as As)*	*SCMO/TNO (1977)*
	0.05	*arsine*	
America	*0.01*	*inorganic; OSHA*	*NIOSH/OSHA(1981)*
	0.002	*inorg.; 15 min.; NIOSH*	
	0.2	*arsenic and soluble compounds*	*ACGIH (1985)*
	0.2	*arsine*	
CSSR	*0.3*	*arsenic and compounds (as As)*	*SCMO/TNO (1977)*
	0.2	*arsine*	
USSR	*0.3*	*arsenic and compounds (as As)*	*SCMO/TNO (1977)*
	0.2	*arsine*	

Table 1.8. *Air quality requirements for outdoor air (mg.m*$^{-3}$*; IDC, 1985)*

Country/province	*Concentration*	*Remarks*
USSR	*0.003*	*daily average; sampling time 20 min., inorganic compounds, as As (except for AsH3)*
Czechoslovakia	*0.003*	*annual average of daily values; inorganic compounds, as As (except for AsH3)*
Israel	*0.006*	*daily average; may be exceeded 3 days per year*
China	*0.003*	*max. daily average; arsenic and compounds, as As*

1.2.4. Food and drinking water

For arsenic no standards have been formulated for accepted contents in foodstuffs. In the Residue Order of the Pesticides Act for arsenic compounds, however, a residue value of 0.1 $mg.kg^{-1}$ for fruit, vegetables and patatoes is mentioned. According to the Salt Decree maximally 1 mg $As.kg^{-1}$ may be present in salt and according to the Substances Decree Cosmetics neither arsenic nor its compounds may occur in cosmetics.

In the VREK-monitoring programme (VREK = "Varkens, Runderen, Eieren, Kippen": Swine, Cattle, Eggs and Chickens), which falls under the responsibility of the Agricultural Advisory Committee on Substances Hazardous to the Environment (LAC) of the Ministry of Agriculture and Fisheries, initiative thresholds have been in force since 1981. In the event of this threshold being exceeded, an investigation will be carried out into the source of the contamination. For arsenic in meat, liver and kidneys these thresholds are 0.03; 0.15 and 0.15 $mg.kg^{-1}$, respectively (LAC, 1982). For arsenic acceptable levels apply for meat and liver/kidneys from cattle and swine. The acceptable levels amount to 0.1 $mg.kg^{-1}$ for meat and 0.5 $mg.kg^{-1}$ for liver and kidneys (CCRX, 1988).
In England the limit value for food is 1 $mg.kg^{-1}$, except for fish and seaweed (WRC, 1984).
The accepted daily intake (ADI) for man is, according to the WHO/FAO (Contaminantenboekje, 1984), 2 $\mu g.kg^{-1}$ body weight for inorganic arsenic.
In the event of the amount of 5 $\mu g.l^{-1}$ in drinking water when leaving the pumping station being exceeded, the sanitary inspector has to be notified (Waterleidingbesluit, 1985).
In table 1.9 the standards for drinking water are shown.
The standard for surface water meant for the preparation of drinking water is 20 $\mu g.l^{-1}$.
Natural mineral- and source water may contain 50 $\mu g.l^{-1}$ maximally.

Table 1.9. *Drinking water standards* ($\mu g.l^{-1}$)

Country	*Standard*	*Reference*
The Netherlands	*50*	*Waterleidingbesluit (1985)*
Germany	*40*	*SCMO/TNO (1977)*
EC	*50*	*EG (1980)*
US	*50*	*EPA (1979)*
USSR	*100*	*Zielhuis (1981)*

1.2.5. Remaining

The Commodity Board for Cattle Feed dictates a limit value of 2 $mg.kg^{-1}$ for single animal feed and green fodder, based on 88% dry matter.

In accordance with the Chemical Waste Act waste is considered chemical waste when the concentration of arsenic and arsenic compounds is higher than 50 $mg.kg^{-1}$. When it concerns waste containing water the concentration limit should be based on the dry matter content of the chemical waste, this content amounting to 0.1 weight percentage or more. In case the dry matter content in the waste is below 0.1 weight percentage it is considered chemical waste when the arsenic content of the total batch, including water, is greater than 50 $\mu g.kg^{-1}$.

The Packaging and Commodities Regulation contains a specific migration limit of 0.01 $mg.dm^{-2}$.

2. PRODUCTION, APPLICATIONS, SOURCES AND EMISSIONS

In this chapter an overview is given of the production, applications, sources and emissions of arsenic, arsenic compounds and arsenic-containing substances. Unless stated otherwise, the data refer to the year 1987. In 2.1. the production (in the instance of As, the import) and the applications of arsenic are described. For a number of relevant sectors an overview is given in 2.2. for the arsenic flows within these sectors. The accuracy of the data mentioned is generally low. Besides, the annual variation in the extent of the flows is in a number of cases considerable.

2.1. PRODUCTION AND APPLICATIONS

The annual import of arsenic (compounds) is appr. 600 tonnes (table 2.1.) (CBS, 1989a). The greater part of the import takes place in the form of zinc ore, which contains arsenic as a contamination. This arsenic is obtained from the zinc ore mainly as a metal. Part of the arsenic is again exported in the form of products.

In the Netherlands, arsenic is mainly applied in the preservation of wood. An estimated 40 - 50 tonnes of arsenic is used to this purpose annually. Furthermore, arsenic is used in alloys, in drugs and as a softener in PVC (Natuur en Milieu, 1984). How much arsenic is involved is not known.

On a global scale 80 - 90% of the arsenic produced is applied as pesticides in agriculture (Nriagu and Pacyna, 1988).

2.2. SOURCES AND EMISSIONS

2.2.1. Natural sources

With regard to the extent of arsenic emissions from natural sources there is great uncertainty. Globally the greatest emissions possibly occur to air (Hutchinson and Meema, 1987). It is estimated that the natural emission to air on a global scale is appr. 50% greater than the anthropogenic emission. The main natural sources are volcanoes and biological methylation. The total global emission to air amounts to an estimated 46 kilotonnes of arsenic per year.

Table 2.1. Import and export of arsenic (compounds) in 1986 in tonnes (CBS, 1989a)

Type of arsenic	*Import*	*Export*	*Application*
Arsenic compounds			
Arsenic, As-containing products	*37*	*44*	*xerographic industry*
Arsenic trioxide	*3*	-	
Arsenic pentoxide and - acids	*59*	-	*wood preservation;* *pesticides**; softeners*
Arsenates and arsenites	*1*	-	*wood preservation*
Organic arsenic compounds	*9*	*1*	
SUBTOTAL	*109*	*45*	
Arsenic-containing raw materials			
Zinc ore (790 ppm)	*313*	*0.1*	*non-ferro metal industry*
Iron ore (4-10 ppm)	*30-75*	*0*	*pig iron- and steel industry*
Phosphate ore (10-20 ppm)	*20-40*	*0.1*	*chemical fertilizer industry*
Phosphate fertilizers	*3*	*15*	*chemical fertilizer industry*
Phosphoric acid/phosphorus	*5*	*9*	
Coal(prod.) incl. coke	*49*	*15*	*fuel for power plants*
Crude oil (0.08-0.67 ppm)	*4-31*	*0-1*	*petrochemical industry*
Raw material for animal feed	*12*	*<1*	*concentrate*
Fish and fish products	*2.2*	*1*	
SUBTOTAL:	*438-530*	*40-41*	
TOTAL:	*547-639*	*83-86*	

**) since 1984 the application of arsenic-containing pesticides is prohibited in the Netherlands*

2.2.2. Industry

- Fertilizer industry

As mentioned in table 2.1. the fertilizer industry imports arsenic in the form of a contamination in phosphate ore. On the extent of the arsenic flow as well as on its final destination there are great uncertainties. Reports from the phosphate ore processing industry show that its arsenic content may vary between 5 and 25 mg.kg^{-1}, most values are between 10 and 20 mg.kg^{-1}, however (CBS, 1989b).

In 1987 over 2 Mtonnes of phosphate ore was imported (CBS, 1989b). The fertilizer industry uses part of this phosphate ore for the production of phosphoric acid by means of a wet procedure, subsequently this phosphoric acid is processed to fertilizer and feed phfosphate. The remaining part of the phosphate ore is converted into phosphorus by the chemical industry (see below). The arsenic flows are shown in figure 2.1.

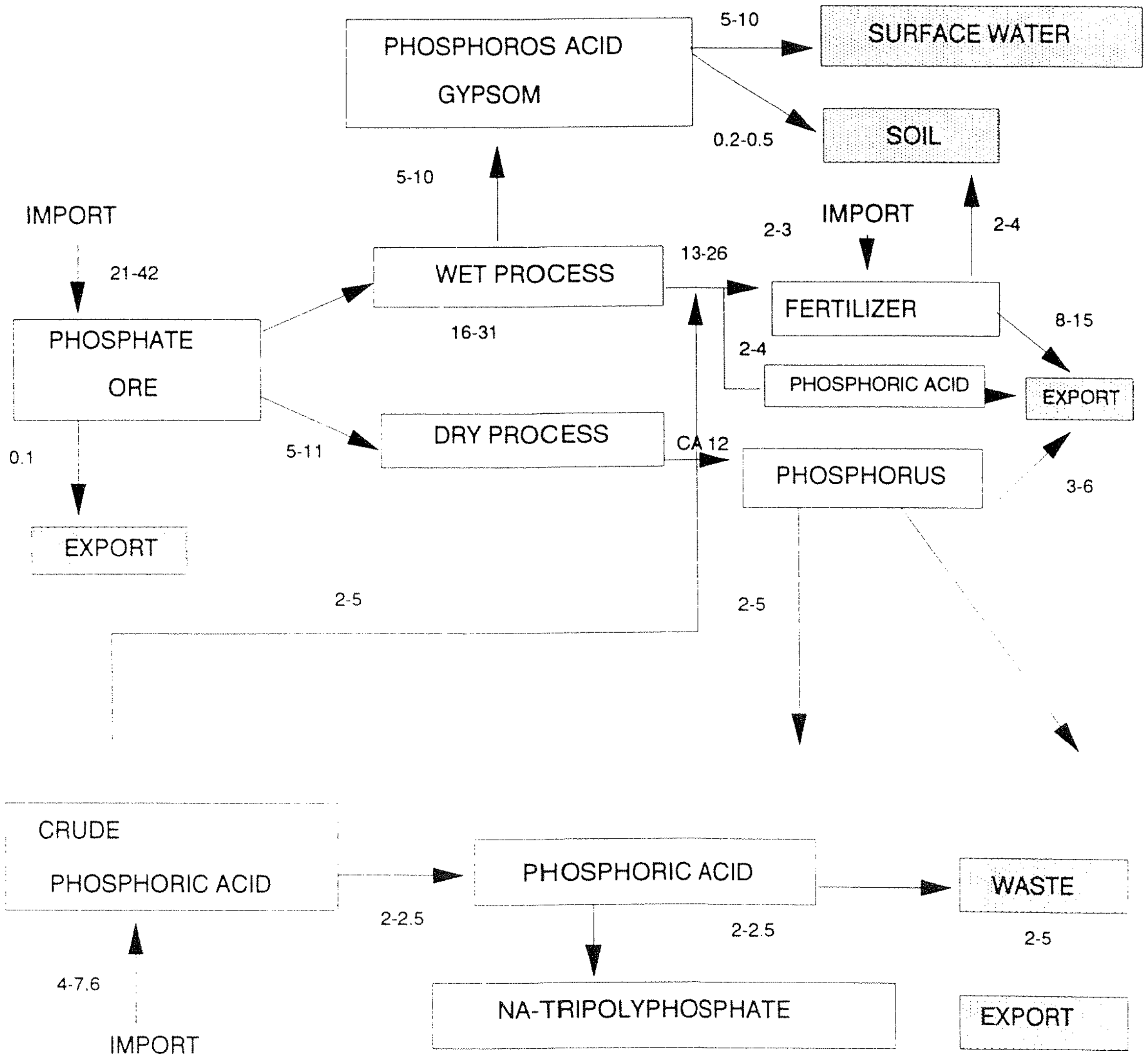


Figure 2.1. Arsenic flows related to phosphate-containing substances (tonnes of arsenic per year)

In the fertilizer industry phosphoric acid gypsum is produced as waste, which for the greater part is discharged on the surface water and for a small part stored on the soil. An estimated two thirds of the arsenic ends up in the phosphoric acid and one third in the gypsum (CBS, 1989b). The arsenic content of fertilizer is in that case 21-40 $mg.kg^{-1}$ P_2O_5 and the content of the gypsum 2.5-5.4 $mg.kg^{-1}$. The annual emission of arsenic to water is in that case 5-10 tonnes. The uncertainty about the extent of this emission is illustrated by the fact that it is 50-80% lower than the emission quantity as determined by the Emission Registration.

- <u>Chemical industry</u>

The major arsenic flows in the chemical industry result from the processing of phosphor ore/phosphoric acid and from titanium ore.

In the chemical industry phosphor ore is processed by means of a thermal procedure (see figure 2.1), during which phosphorus is produced and several residual products such as phosphorus slag, ferrophosphorus, arsenic sulphide and waste gypsum are formed. The arsenic content of phosphorus slag and ferrophosphorus is very low, the amount of arsenic in arsenic sulphide is 2-5 tonnes (Meijer, 1989). The arsenic sulphide was up to 1986 dumped as chemical waste abroad, since then the storage has taken place on the industrial sites. Almost all arsenic ends up in the phosphorus. This is for the greater part being exported and the remainder is converted into phosphoric acid. From this and from imported phosphoric acid sodium phosphate is produced, a component of detergents.

During the preparation of titanium white from almenite ($FeTiO_3$) a waste acid flow of appr. 300 tonnes arises. This acid, which is discharged on the New Waterway, contains small quantities of arsenic.

- <u>Non-ferro metal industry</u>

The major arsenic flows in the non-ferro metal industry are related to the production of zinc, lead, tin, wolfram and alloys. In the past the arsenic emissions to air were considerable (Dienst Milieuhygiene Gelderland, 1989). At present this situation has been brought under control and the major arsenic flow is found in solid waste. The main arsenic flow results from the production of zinc (CCRX, 1989). In this process jarosite is released, which is dumped as chemical waste. Zinc ore consists for appr. 85% of zinc

sulphide and for appr. 10% of iron sulphide (Dor, 19??). Furthermore, it contains various contaminants such as cadmium, lead, silver, cobalt, arsenic, copper and silicates. Various processes are available for the extraction of zinc and other metals such as cadmium and copper from zinc ore. The zinc extraction process currently applied in the Netherlands consists of a number of steps. The first step is the roasting of the crude ore at high temperatures during which sulphides are converted into oxides, which are soluble in acid systems. The SO_2 formed is converted into sulphuric acid. Subsequently the greater part of the remaining metals is dissolved during the neutral treatment with lye. After separation and following several purification steps the liquid is led to the electrolytic zinc extraction. In order to enable good elctrolysis to take place hardly any arsenic may be present in the flow (0.01-0.02 mg As per liter maximally). The underflow consisting of zinc ferrite, iron hydroxide, the insoluble zinc oxide and other non-soluble sulphides and sulphates forms one of the three inputs into the jarosite precipitation tanks. Following concentration, hot acid treatment with lye, and concentration once again, the final mixture is filtrated. The filtrate which is formed is jarosite.
On an annual basis the jarosite flow amounts to appr. 220 kilotonnes (130 kilotonnes dry matter). The arsenic content is 2000-2440 $mg.kg^{-1}$ (dry matter), the amount of arsenic in the jarosite being 260-317 tonnes. The production of jarosite on a global scale is appr. 1,800 kilotonnes per year. Since 1974 jarosite has been stored as waste, resulting in considerable depots. So far no effective processing methods have been developed.

The As flow as a result of the production of lead and tin varies highly annually because of the non-continuous operation of various processes and building-up of stocks of intermediate products. In 1987 this flow amounted to appr. 70 tonnes (CCRX, 1989). The flow mainly consists of metallurgical waste. In the production of lead and tin-containing products arsenic is sometimes added in small quantities. The amounts added depend on the required product (battery-lead, solder etc.). Part of the arsenic, together with the fly ash in the process gases of the smelters, enters the air. During remelting inorganic arsenic (III) oxides (As_2O_3 or As_4O_6) are released. Because of the low boiling point of As_4O_6 the greater part of the arsenic in the fly ash originates from this substance.

- Remaining industry

The emission of arsenic to air and surface water caused by the remaining industry is relatively small (table 2.2.). The emission from sewage plants (see 2.2.6.) has not been incorporated.

Table 2.2. *Emission of arsenic to air by remaining industry (tonnes per year)*

Sector	*Emission*		*Reference*
	Air	*Surface water*	
Chemical industry		*0.26-0.7*	*Emission Registration*
Wood preservation comp.		*0.005*	*Estimation RIVM*
Remaining industry	*0.038*	*0.016*	*Emission Registration*

2.2.3. Agriculture and animal husbandry

The As load on agricultural land has decreased considerably since 1984 because of the ban on arsenic-containing pesticides. The current As flow in agriculture and animal husbandry in the Netherlands amounts to 27-40 tonnes annually.

Arsenic enters the sector in the form of chemical fertilizer, imported base materials for concentrate and through deposition. The net flow to agricultural land is 22-24 tonnes annually. Foundation of the data by means of recent measuring data appeared not to be possible. The As flows in the form of chemical fertilizer, manure and base materials for animal feed are, therefore, determined based on the amounts which have been produced and/or imported and on the contents calculated or those measured in the past. The flow to the soil in the form of manure is based on a weighted average content of 0.8 $mg.kg^{-1}$ (Produktschap voor Veevoeder, 1987) in produced animal feed (15.4 Mtonnes in 1986-87) (Produktschap voor Veevoeder, 1987) of 0.03 $mg.kg^{-1}$ (Produktschap voor Veevoeder, 1985) in roughage (91 Mtonnes grass, 9.9 Mtonnes maize and 0.6 Mtonnes mangolds)(CBS, 1987; LEI, 1987). Assuming that appr. 15% accumulates in the animals fed the flow of arsenic to manure amounts to 13-15 tonnes.

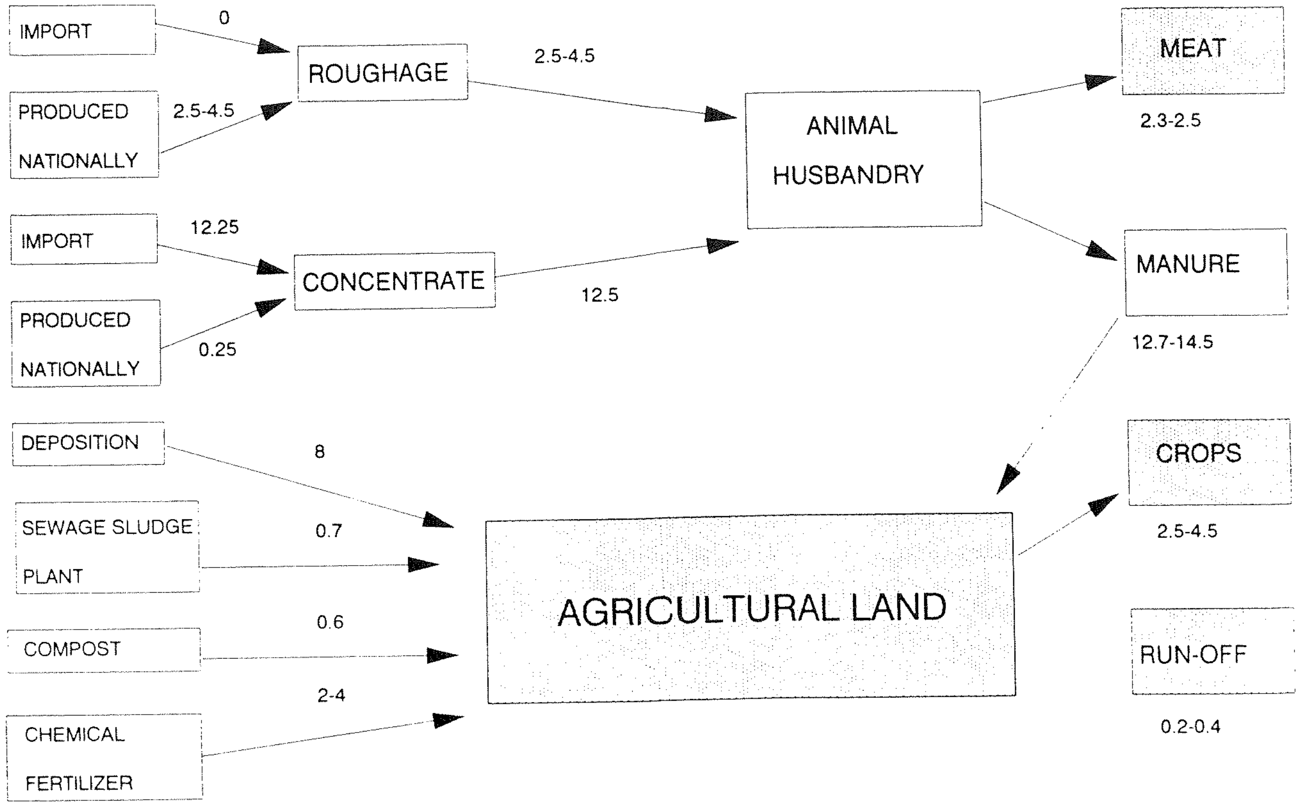


Figure 2.2. *Arsenic flows in the Dutch agriculture and animal husbandry (tonnes per year)(see 2.2.6. for deposition)*

2.2.4. Waste processing

- Urban waste, construction- and demolition waste and remaining waste

The total waste flow in the form of urban waste, construction- and demolition waste and remaining waste in 1986 amounted to 16.6 Mtonnes (Nagelhout et al., 1989). Urban waste consists of domestic waste and waste from small companies.

The arsenic contents of garbage bag waste, glass and paper have been reported by Van de Beek et al. (1989), the contents of the remaining kinds of waste have been estimated. The extent of the waste flows and their arsenic contents and the arsenic flows calculated from this are shown in table 2.3.

Table 2.3. *Amounts of urban waste, construction- and demolition waste and remaining waste and the related arsenic flows in 1988 (Nagelhout et al., 1989)*

Kind of waste	*Mtonnes*	*ppm As*	*tonnes As*
Domestic waste			
-garbage bag waste	*4.3*	*3.9-4.9*	*17-21*
-glass	*0.2*	*28-32*	*5.6-6.4*
-paper	*0.5*	*0.33-0.45*	*0.2-0.2*
-bulky refuse	*0.7*	*3.9-4.9*	*2.7-3.4*
Small companies/services waste	*1.8*	*4.5-5.4*	*8.1-9.7*
Urban waste (rounded)	*7.5*	*4.5-5.4*	*34-40*
Construction and demolition waste	*7.7*	*0.5*	*4*
*Remaining waste**	*1.4*	*3.9-4.9*	*5.5-6.9*
Total (rounded)	*16.6*	*2.6-3.1*	*43-51*

** market-, park-, sweep- and floating waste; dredgings; drain sludge*

In 1986 2.6 Mtonnes of these 16.6 Mtonnes was incinerated (Siemons, 1988), 0.1 Mtonne processed to compost, 10.4 Mtonnes dumped and 4.4 Mtonnes recycled. The arsenic present in the waste which is incinerated, for the greater part ends up in the slag or the fly ash of the incinerators. A small part is emitted to the air in the form of fly ash. The above-mentioned is shown in figure 2.3. This diagram shows there is a discrepancy between the input and the output of arsenic in the incinerators. The input is appr. 12 tonnes, the output appr. 16 tonnes. This is caused by the considerable scatter in the contents of the various flows.

- Remaining waste

In table 2.4. the extent of the annual solid waste flows of the remaining sectors is mentioned.

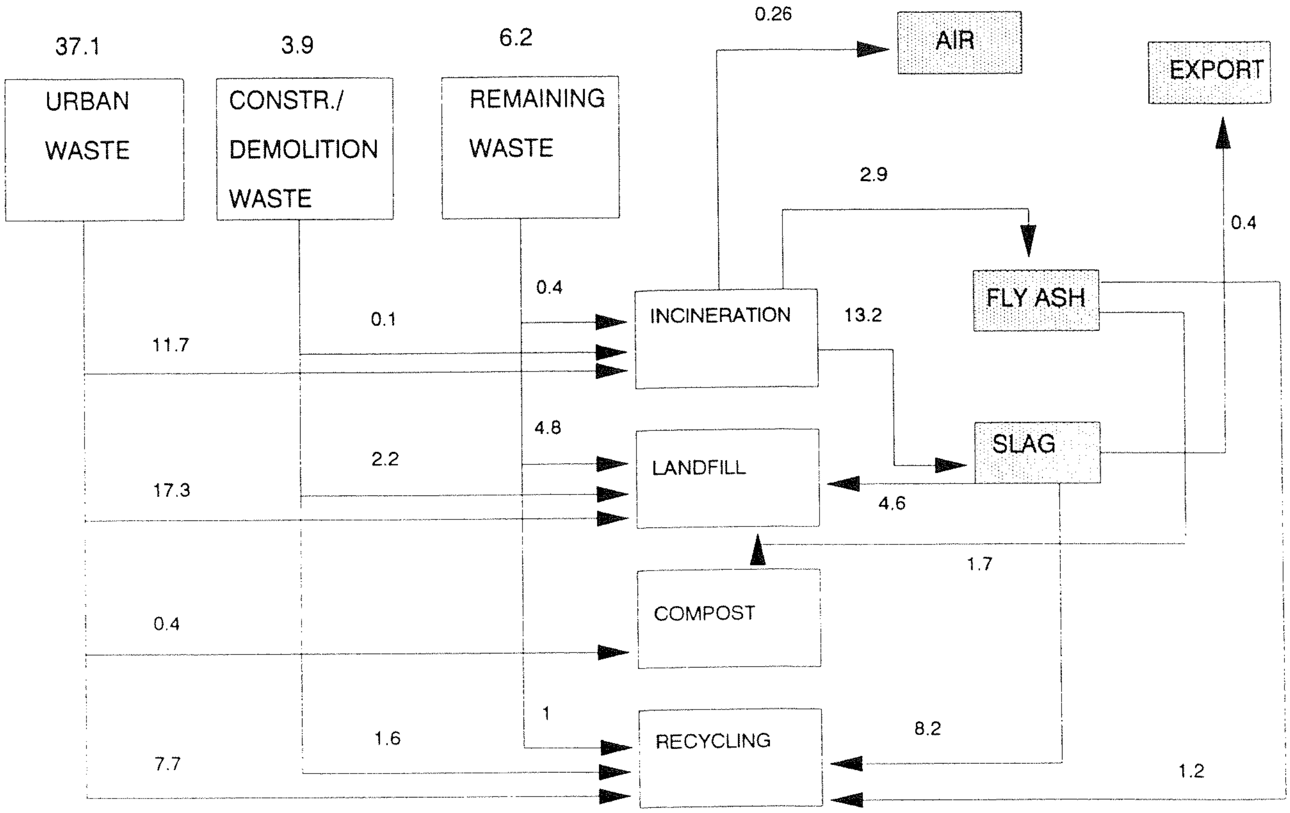


Figure 2.3. Arsenic flows (tonnes) as a result of the production and the incineration of urban waste, construction and demolition waste and remaining waste in 1986

Table 2.4. Arsenic flows (tonnes) as a result of the application and dumping of waste by the remaining industry in 1986 (1989)

Sector	*Dumping*	*Application*	*Export*	*Total*
Wood preservation companies	*n.c*	*n.c*	*n.c*	*0.25*
Dredgings	*18*			*18*
Shredder installations	*0.6*		*0.6*	*1.2*
Drinking water sludge	*4.6*	*0.2*		*4.8*
Slag incineration chemical waste	*0.52*			*0.52*
Total (rounded)	*24*	*0.2*	*0.6*	*25*

2.2.5. Power plants

Especially in the Netherlands arsenic is emitted into the environment by power plants in the form of waste originating from the combustion of coal. The arsenic for the greater part ends up in the solid wastes fly ash and slag and a small part is emitted to the air. The import of arsenic in the form of coal in 1987 amounted to appr. 22 tonnes. Most of it ends up in fly ash. In figure 2.4. the destination of arsenic is schematically shown.

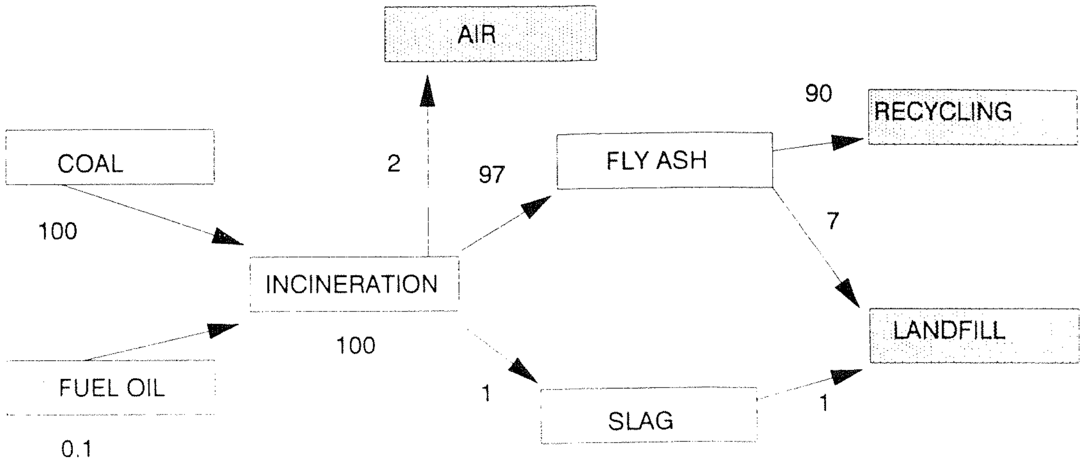


Figure 2.4. Arsenic flows in power plants in tonnes (1987)

The consumption of coal for the generation of electricity has decreased in the period 1960-1980 from appr. 5 million tonnes in 1960 to appr. 2.3 million tonnes in 1980 (CBS, 1975; Meij, 1988). From then on the use of coal has again increased to appr. 6 million tonnes in 1987 (Meij, 1988). By increasingly applying good dust collectors with a high efficiency (appr. 99.7%), the emission of fly ash to air in the period of 1980-1987 decreased by over a factor 8. Because of this the emission of arsenic to air, resulting from coal combustion, has also considerably decreased.

2.2.6. Diffuse sources

- Households and companies

Households and companies mainly emit arsenic to surface water and to sewage plants sludge. Based on an efficiency of 70% for the purification in sewage plants, the emission to surface water and to sludge by communal and private sewage treatment plants in 1985 amounts to 0.8 and 1.9 tonnes arsenic, respectively (CBS, 1989c). Furthermore 0.7 and 0.05 tonnes of untreated arsenic is discharged into surface water and into the soil (see figure 2.5.).

- Wood preservation

In the Netherlands arsenic is mainly applied in the preservation of wood. This process for the greater part takes place in some 35 companies. During the preservation process the emissions to the environment are to be neglected. Also during storage on the industrial premises the emission is slight (Houtinstituut TNO, 1987). The preserved wood is applied in the construction industry, shipbuilding, for packaging and bank protection. During the life of the products part of the arsenic leaches out. The greatest emission takes place to water and is estimated at appr. 1 tonne per year (Berbee, 1989). At an annual application of 40-45 tonnes of arsenic this means that the main part (93-97%) of the arsenic remains present in the products and finally ends up in the environment as waste. The way of removal determines which environmental compartment will be loaded and to what extent. During uncontrolled combustion appr. 50 (20-80)% of the arsenic enters the air, almost completely particle-bound (McMahon, et al., 1986). During controlled combustion the emission will be considerably lower because the dust is collected by means of electro-filters. It is not clear where exactly in the Netherlands the waste wood containing arsenic ends up, it is, therefore, not possible to make quantitative statements on the As flows associated with this.

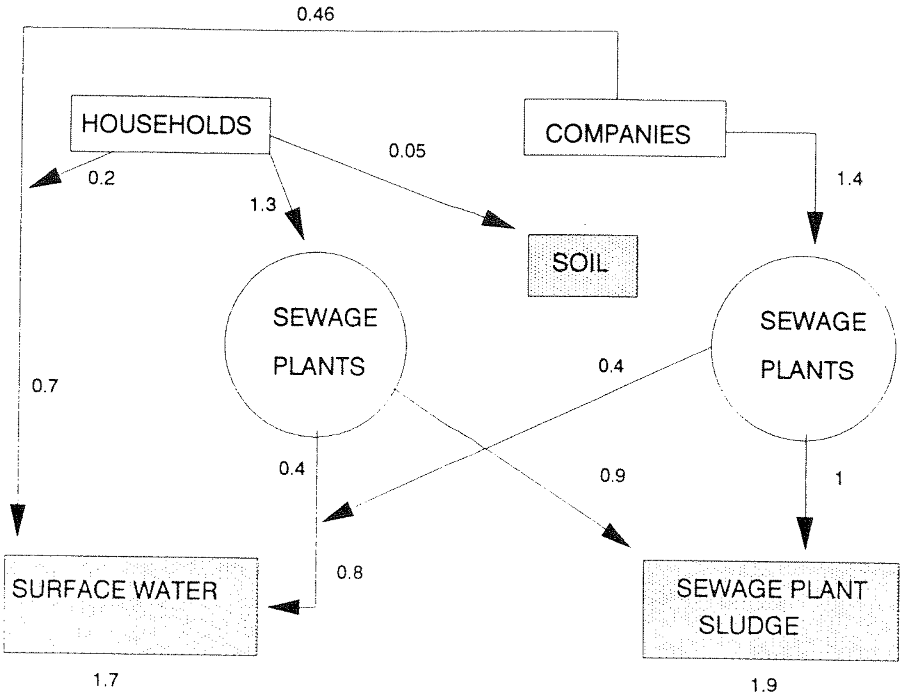


Figure 2.5. Arsenic flows (tonnes) as a result of waste water from households and small companies in 1984 (CBS, 1989c,d)

- Fish

Fish contains arsenic in the form of arsenobetaine. The arsenic contents of seafish range from 2 to 30 mg.kg^{-1} (CCRX, 1989). In the fish processing part of the arsenic enters the waste water which, in a number of purification boards in e.g. Friesland and the Northeast Polder, gives rise to elevated contents in the sewage plant sludge, interfering with the application of this sludge in agriculture. The extent of the As flows involved is not exactly known; it is estimated to be appr. 200 kg arsenic. It is not known whether the organically bound arsenic of fish offal can be converted into inorganic arsenic. Further research into this matter appears to be desirable. In figure 2.6. the As flows related to fish are mentioned.

- Combustion of fossil fuel

Besides in power plants fossil fuels are also used elsewhere for the generation of electricity. Fuels most relevant to arsenic are fuel-oil and coal. In table 2.5. the extent of the arsenic flows associated with this are shown. The emission to air is calculated based on an emission factor of 66 and 100 mg arsenic per tonne for coal and fuel-oil, respectively.

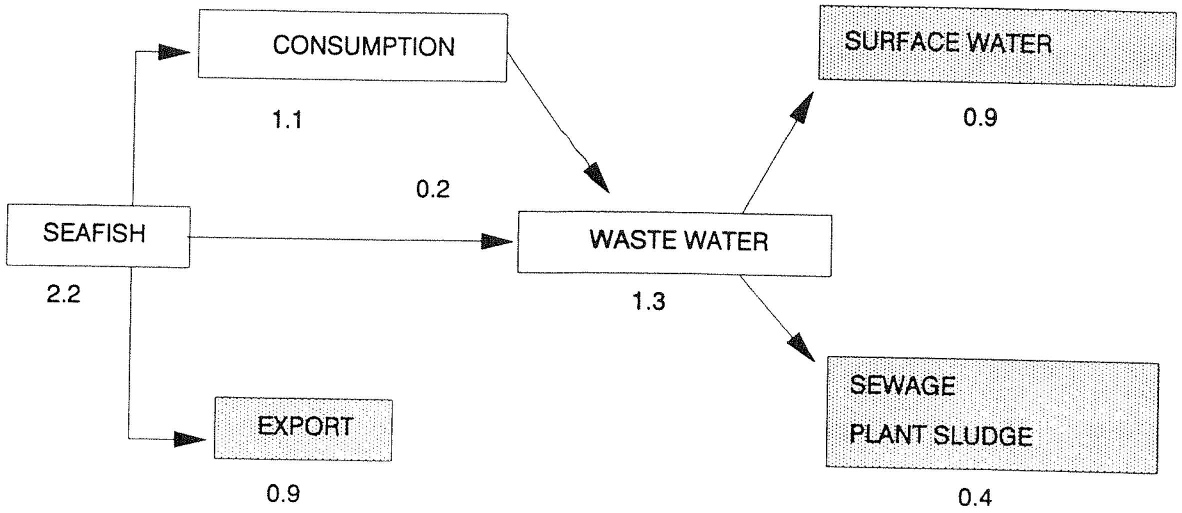


Figure 2.6. As flows (tonnes per year) related to catch, processing and consumption of seafish

Table 2.5. Arsenic flows resulting from combustion of fossil fuels in tonnes (1987)

Fuel	*Consumption*	*Emission to:*		
	(Mtonnes)	*Air*	*Soil*	
			dumped	*applied*
Fuel-oil	*1.61*	*0.16*		
Coal	*0.6*	*0.04*		
- as fly ash			*0.2*	*2.2*
- as slag				*0.02*

- Deposition

Based on calculations (see chapter 4, section 4.5.3.) the total deposition (wet and dry) is estimated at appr. 17 tonnes. Approximately 15 tonnes of this ends up on the soil, of which more than 8 tonnes on agricultural land.

- Remaining sources

The annual arsenic flows to the Dutch environment by remaining sources are given in table 2.6.

2.2.7. Sources abroad

In table 2.7. the arsenic emission to air in 1982 is shown (Pacyna and Munch, 1988). The greater part of the emission data is based on statistical data on the extent of certain activities and the related emission factors. According to this research the Dutch emission is 34 tonnes per year. This is considerably higher than the current emission and possibly also higher than the emission in 1982. This may mean that the calculated emissions for the other countries are too high as well.

Table 2.6. Arsenic flows by the remaining sources (tonnes per year)

Source	*Soil*	*Air*	*Reference*
Pellets of shot	*0.84-3.40*		*CCRX, 1989*
Fireplaces/multiburners/smoking		*0.03-0.08*	*Okken, 1983**
Leaching out preserved wood	*1*		*Berbee, 1989*
*Landfills ***	*<0.01*		*Beker, 1989*

* *Values shown in this table have been deduced from data extracted from the reference mentioned*

** *Departing from a situation where soil sealing at the dumping grounds is not applied. It concerns the emission to soil and surface water*

Table 2.7. Arsenic emissions to air in a number of European countries in 1982 (Pacyna and Munch, 1988)(see text)

Country	*Emission (tonnes)*
Switzerland	*4*
Denmark	*7*
Greece	*15*
Hungary	*16*
Norway	*41*
Belgium	*85*
East Germany	*95*
Italy	*96*
Finland	*106*
Great Britain	*117*
France	*170*
Sweden	*183*
Yugoslavia	*272*
West Germany	*351*
Poland	*597*
Russia (European)	*2098*
Remaining countries	*719*
Total (rounded)	*5000*

The input of arsenic through the Rhine and the Meuse over the past few years is given in table 2.8. In the Design Plan for the quality of the North Sea (1985) it is mentioned that the natural contribution to the arsenic flow of all rivers flowing down through the Netherlands is appr. 200 tonnes per year.

Table 2.8. Average annual arsenic input through the Rhine and the Meuse in the Netherlands (in tonnes)(RIWA, 1980-1986)

	1980	*1981*	*1982*	*1983*	*1984*	*1985*	*1986*
Rhine	*251*	*293*	*352*	*259*	*158*	*113*	*155*
Meuse	*132*	*97*	*52*	*31*	*25*	*12*	*18*
Remaining							*<10*

2.3. SUMMARY AND CONCLUSIONS

The arsenic load of soil, surface water and air has decreased in recent years. This decrease since 1980 amounts to appr. 70% for agricultural land where As-containing pesticides were applied and more than 50% for the state-owned waters as a result of decreased emissions abroad. The arsenic emissions to air in the Netherlands have decreased by appr. 80% through e.g. the application of dust collectors, as a result of which the load on a national scale is mainly determined by sources abroad.
The annual emission of arsenic from Dutch sources to air, water and soil amounts to appr. 440 tonnes per year (basic year 1987). Most arsenic ends up on the soil in the form of waste.

The annual emission to air is appr. 1.1 tonne arsenic. The most important sources being the non-ferro metal industry and power plants (table 2.9.).
The annual arsenic emission to surface water is 10 - 15 tonnes. The major source being the fertilizer industry (table 2.10.). It should be noted that a considerable part (5 - 10 tonnes) is discharged on the New Waterway and, therefore, affects the marine environment more than it does the fresh water environment.

Table 2.9. Emission of arsenic from Dutch sources to air (tonnes per year)

Kind of source	*Amount*	*Year*	*Reference*
Industry:			
Pig iron and steelindustry	*0.04*	*1982*	*Emission Registration, 1988*
Non-ferro metalindustry	*0.06**	*1986*	*Dienst Mil. hyg. Gld, 1989*
Remaining industry	*0.04*	*1984*	*Emission Registration, 1988*
Energy generation:			
Coal	*0.04*	*1987*	*Meijer, 1988; Meij, 1988 ***
Oil	*0.03*	*1987*	*Meijer, 1988; Meij, 1988 ***
SUBTOTAL	*0.33*		
Electricity power:			
Coal	*0.4*	*1987*	*Meijer, 1988; Meij, 1988 ***
Oil	*0.03*	*1987*	*Meijer, 1988; Meij, 1988 ***
	0.43		
Waste incinerators:			
Domestic waste	*0.26*	*1987*	*Meijer, 1988*
Chemical waste	*0.02*	*1987*	*CCRX, 1989*
SUBTOTAL	*0.28*		
Fireplaces/multiburners/ smoking	*0.03-0.08*		*Okken, 1983 ***
TOTAL (rounded)	*1.1*		

** This emission was considerably greater in the past*
*** The values incorporated in this table have been deduced from data extracted from reference(s) mentioned*

As a result of diffuse sources appr. 34 - 42 tonnes arsenic ends up on the soil annually; data are shown in table 2.11.
In table 2.12. an overview is given of the fate of arsenic-containing waste. Until now appr. 75 tonnes was exported (mainly by the non-ferro metal industry) and dumped abroad. As per 1 January 1990 licenses are no longer granted for this in the Netherlands.

Table 10. Emission of arsenic to surface water (tonnes)

Kind of source	*Amount*	*Year*	*References*
Industry:			
Fertilizer industry	*5-10*	*1987*	*CBS, 1989b*
Non-ferro metal industry	*0.41*	*1985-1987*	*Emission Registration, 1988*
Chemical industry	*0.26-0.7*	*1985-1987*	*Emission Registration, 1988*
Wood preservation companies	*0.005*	*1987*	*Estimation, RIVM*
Remaining industry	*0.016*	*1982-1987*	*Emission Registration, 1988*
SUBTOTAL	*5.7-11.1*		
Sewage plants:			
Public	*0.6*	*1985*	*CBS, 1989c*
Private	*0.2*	*1985*	*CBS, 1989c*
Sewage water run over	*0.1*	*1987*	*Meijer, 1988*
SUBTOTAL	*0.9*		
Deposition (wet + dry)	*2*	*1988*	*Chapter 3 + 4*
Domestic waste water	*0.2*	*1985*	*CBS, 1989c*
Leaching out impregnated wood	*1*	*1988*	*Berbee, 1989*
Landfills	*0.01*		
SUBTOTAL	*3.2*		
TOTAL (rounded)	*10-15*		

Table 2.11. Diffuse loading of the soil with arsenic (tonnes)

Kind of source	*Amount*	*Year*	*Reference*
To agricultural land:			
Purification sludge			
- communal	*0.55*	*1985*	*CBS, 1989c*
- private	*0.14*	*1985*	*Martens, 1989*
Compost	*0.5*	*1985*	*CBS, 1989c*
Chemical fertilizer	*1.9-3.6*	*?*	*CBS, 1989b*
Manure	*13-15*		*Estimation estimation*
Deposition	*8*	*1988*	*Chapter 3 + 4*
SUBTOTAL	*24-29*		
To remaining land:			
Purification sludge			
- communal	*1.4*	*1984*	*CBS, 1989c*
- private	*0.5*	*1985*	*CBS, 1989c*
Domestic waste water	*0.05*	*1986*	*CBS, 1988; Feenstra, 1986*
Deposition	*7*	*1988*	*Chapter 3 + 4*
Pellets of shot (hunting)	*0.84-3.4*	*1986*	*CCRX, 1989*
SUBTOTAL	*10-13*		
Leaching out materials/ waste products	*0.005*		*Estimation RIVM*
Leaching out landfills	*0.013*	*1986*	*Nagelhout, 1989*
TOTAL (rounded)	*34-42*		

Table 2.12. Fate of arsenic-containing waste in the Netherlands in tonnes As

Kind of source	Amount	Dumped	Applied	Year	References
Waste:					
Non-ferro metal	330-387	330-387		1987	CCRX,1989;Meijer,1988
Chemical industry	3	3		1987	Meijer,1988
Fertilizer industry	0.6	0.6		1986	Meijer,1988;Ereg,1988
Wood preservation	0.25	0.25		1986	CCRX,1989
Remaining industry:					
Shredder installations	1.2	1.2		1987	Meijer,1988
Dredgings	18	18		1987	Meijer.1988
Drinking water sludge	4.8	4.6	0.2	1987	Meijer,1988
Domestic waste	16.3	15	1.3	1986	RIVM,1988
Purification sludge					
- communal	1.7	0.55	1.11	1985	CBS,1989c
- private	0.7	0.42	0.29	1985	CBS,1989c
Fly ash coal combustion	22	2	20	1987	Meijer,1988; Meij,1988
Fly ash dom.waste incin.	2.9	1.6	1.2	1987	Meijer,1988
Fly ash chem. waste inc.	0.2	0.2		1987	Meijer 1988
Slag coal combustion	0.03		0.03	1986	RIVM,1988;Meijer,1988
Slag domestic waste inc.	13.2	5.0	8.2	1987	RIVM,1988;Meijer,1988
Slag chem. waste inc.	0.52	0.52		1987	RIVM,1988;Meijer,1988
TOTAL (rounded)	425-482	384-441	46		

3. SPECIATION AND DISTRIBUTION

3.1. BEHAVIOUR OF ARSENIC IN THE ENVIRONMENT

3.1.1. Inorganic arsenic compounds

The chemical behaviour of arsenic in soil and aquatic systems is complicated with oxidation-reduction, ligand exchange, precipitation and adsorption-desorption reactions. The most important inorganic compounds in natural systems are mentioned in table 3.1.

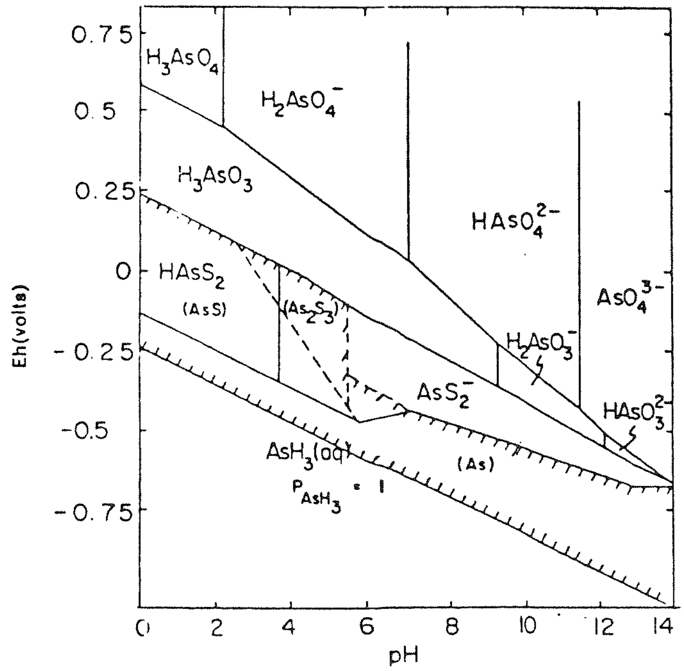
Table 3.1. Possible inorganic forms of arsenic in natural systems (excluding adsorption complexes)

Compound	*State*	*Valency arsenic*	*DGf (kJ/mol)*	*References*
H_3AsO_4	*dissolved*	+5	-766.1	*Wagman et al.,1968*
$H_2AsO_4^-$	*dissolved*	+5	-753.3	*Wagman et al.,1968*
$HAsO_4^{--}$	*dissolved*	+5	-713.4	*Wagman et al.,1968*
AsO_4^{---}	*dissolved*	+5	-648.5	*Wagman et al.,1968*
AsO^+	*dissolved*	+3	-163.8	*Wagman et al.,1968*
H_3AsO_3	*dissolved*	+3	-646.0	*Sergeyeva & Khodakovskiy,1969*
$H_2AsO_3^-$	*dissolved*	+3	-593.3	*Sergeyeva & Khodakovskiy,1969*
$HAsO_3^{--}$	*dissolved*	+3	-524.3	*Sergeyeva & Khodakovskiy,1969*
$HAsS_2$	*dissolved*	*covalent*	-48.6	*Sillen & Martell, 1964*
AsS_2	*dissolved*	*covalent*	-27.4	*Sillen & Martell, 1964*
AsH_3	*dissolved*	-3	99.6	*Sergeyeva & Khodakovskiy,1969*
As	*solid*	0	0.0	*Robie & Waldbaum, 1968*
AsS	*solid*	*covalent*	-70.3	*Robie & Waldbaum, 1968*
As_2S_3	*solid*	*covalent*	-168.4	*Robie & Waldbaum, 1968*
AsH_3	*gas*	-3	69.0	*Sergeyeva & Khodakovskiy,1969*

Oxides such as As_2O_3 and As_2O_5 have too high solubilities to be formed as precipitates in surface water. Also arsenate (AsO_4) precipitates have such high solubilities that they remain unsaturated. The arsenic sulphides are stable at a low redox potential and high sulphide concentrations. AsS is especially formed at low pH values, whereas As_2S_3 is especially stable at intermediary and high pH values (Ferguson and Gavis, 1972). In terrestrial soils where sulphide is usually absent, arsenic does not occur as sulphide.

In an oxidized environment the arsenate complexes are the thermodynamically stable forms; whereas in surface water and groundwater with no oxygen it is the arsenite (AsO_3) complexes. In environments with high sulphide concentrations (reduced marine sediments) the arsenic sulphide complexes are the most important dissolved compounds.

The chemical oxidation of arsenite into arsenate is a slow process (weeks to months, Johnson, 1972). This oxidation, however, is considerably accelerated by the catalytic action of manganese oxides in particular (Oscarson et al.,1981; Peterson and Carpenter, 1983). Especially in shallow fresh water and in soils where the amounts of manganese oxide are relatively high, this will cause the oxidation velocity to increase considerably, so that arsenic mainly occurs as arsenic (5+). Finally, in methanogenic systems, and at extremely low pH values, arsenic hydride (arsine) may become stable. A rough overview of the various forms of arsenic as a function of the pH and the redox are shown in figure 3.1.



<u>Figure 3.1</u>. Eh-pH diagram for arsenic (total As = 10 μmol/l and total S = 1 mmol/l (Ferguson and Gavis, 1972)

3.1.2. Adsorption of arsenic

In the aquatic environment as well as in the soil the distribution of arsenic over the solid and liquid phase is determined by adsorption processes. Adsorption can be described with a linear isotherm:

$S = Kd \times C$ in which
S = concentration in the solid substance ($mg.kg^{-1}$)
Kd= linear adsorption coefficient ($m^3.kg^{-1}$)
C = concentration in solution ($mg.m^{-3}$)

The equation is a simplification of the Langmuir isotherm (Bolt and Bruggenwert, 1978). The value of Kd depends on the properties of the adsorbent, the pH in the solution, the speciation of the adsorbed substance in the solution and on any possible competition with other substances.
The adsorption behaviour of arsenate may well be compared to that ofphosphate: strong adsorption on iron(III)hydroxide (Pierce and Moore, 1982) and aluminium hydroxide in particular, and adsorption on free aluminium groups of clay minerals (Jacobs et al., 1970; Livesey and Huang, 1981; Xu et al., 1988; Goldberg and Glaubig, 1988).
The adsorption of arsenate is highly dependent on the pH and reaches its maximum between pH=5 and pH=7. At high phosphate concentrations (3 g $P.m^{-3}$) competition for the available adsorption places occurs, resulting in a significant decrease of the adsorption of arsenic. Nitrate, chloride and sulphate show no competition effects (Livesey and Huang, 1981). Goldberg and Glaubig (1988) found above pH=9 again an increase of the bonding to the solid substances, which is clearly influenced by the presence of calcite. The calcite being removed the adsorption decreases also above pH=9; in the presence of calcite the adsorption increases above this pH value (which should be considered as exceptionally high for the Netherlands). The linear adsorption coefficients for total arsenate for the compounds iron(III)-hydroxide, kaolinite and montmorillonite (clay minerals) and aluminium hydroxide are shown in table 3.2.

Table 3.2. Linear adsorption coefficients for arsenate

Compound	pH	Kd ($m^3.kg^{-1}$)	References
$Fe(OH)_3$ (amorphous)	5	2780	Pierce and Moore, 1982
	6	2750	
	7	520	
	8	315	
	9	157	
Kaolinite	6	10	Xu et al., 1988
	7	0.7	
	8	0.05	
Kaolinite	5.5	0.02	Frost and Griffin, 1977
Montmorillonite	5.5	0.025	Frost and Griffin, 1977
$Al(OH)_3$	6	350	Calculated from data
	7	30	Xu et al., 1988
	8	2.5	
Several soils	6-7.5	0.05-1.0	Livesey and Huang, 1981

Arsenite shows strong adsorption on iron(III)hydroxide (Pierce and Moore, 1980). Furthermore, adsorption on kaolinite and montmorillonite is reported in the literature (Frost and Griffin, 1977). The available data concerning the linear adsorption coefficients are given in table 3.3.

Table 3.3. Linear adsorption coefficients for arsenite

Compound	pH	Kd ($m^3.kg^{-1}$)	References
$Fe(OH)_3$ (amorphous)	5	705	Pierce and Moore,1980
	6	1050	
	7	1190	
	8	990	
	9	563	
Kaolinite	5	0.002	Frost and Griffin,1977
Montmorillonite	5	0.003	Frost and Griffin,1977
Organic carbon		8.3	Calculated from data Kerdijk et al.,1984

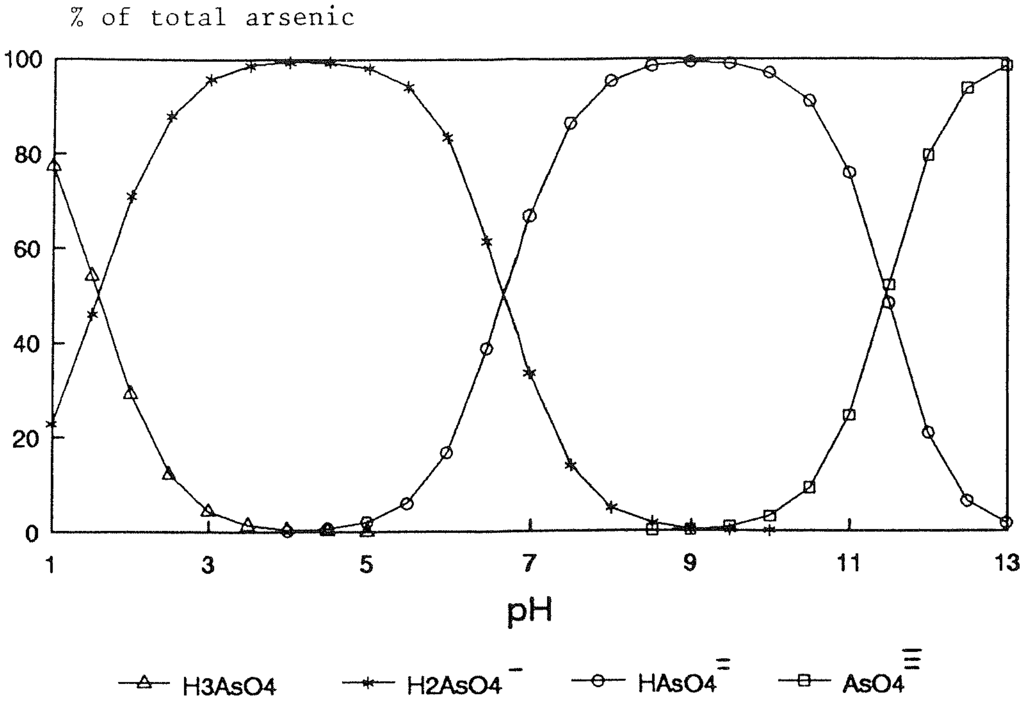


Figure 3.2. *Speciation of arsenic (5+) as a function of the pH in the absence of adsorbing substances*

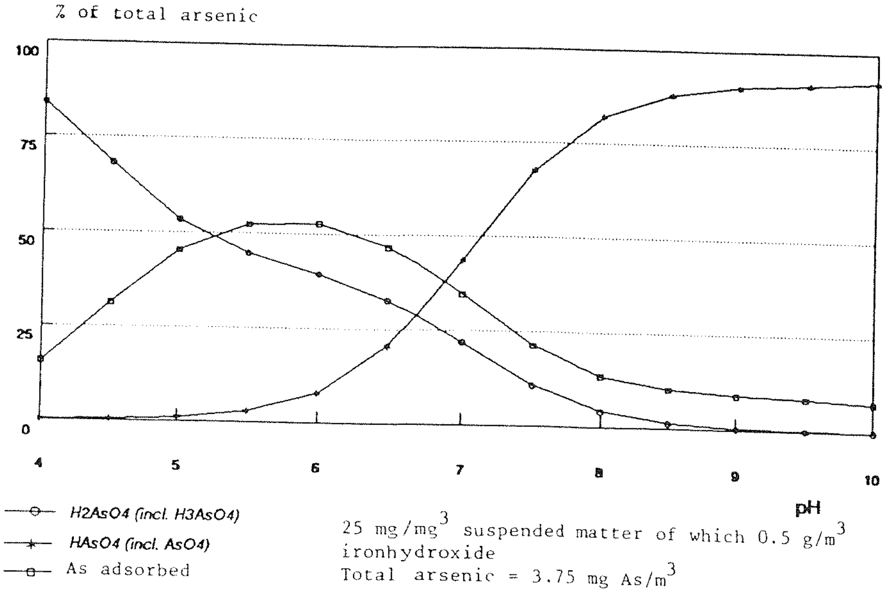


Figure 3.3. *Speciation of arsenic (5+) as a funtion of the pH in the presence of adsorbing substances*

3.1.3. Calculated speciation for several characteristic environments

In figure 3.2. the bonding forms of As (5+) have been put against the pH in the absence of adsorbing substances and in figure 3.3. in the presence of adsorbing substances. The maximum adsorption to the solid phase occurs at a pH value of appr. 6 and strongly decreases at increasing pH values to arrive at a constant level of appr. 10% at pH > 8.

3.1.4. Organo-arsenic compounds

The biological cycle for arsenic in the aquatic environment is shown in figure 3.4. It is not known to what extent organic arsenic is transformed into inorganic arsenic in the aquatic environment.

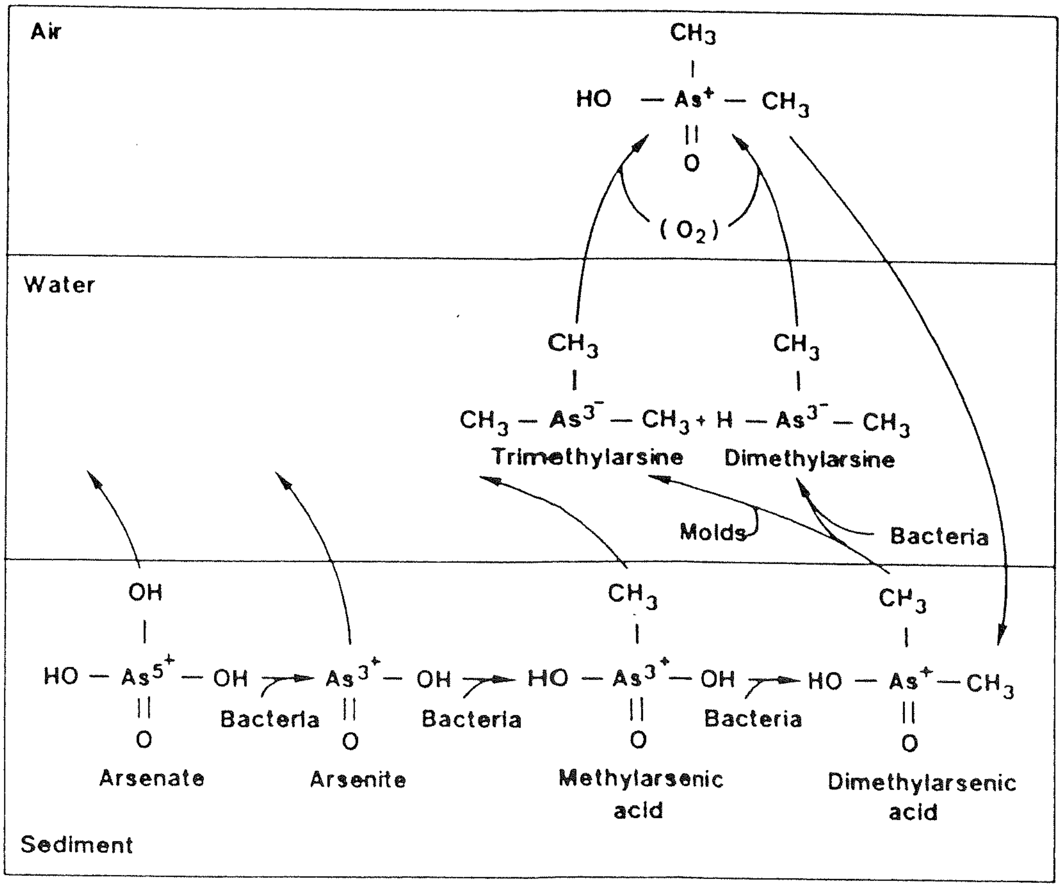


Figure 3.4. Biological cycle for arsenic in the aquatic environment (Wood, 1974)

The cycle is complicated because of arsenic being subject to both oxidation/reduction and methylation. Under reducing conditions toxic dimethyl and trimethyl arsine are formed by bacteria. These arsines are, however, rapidly oxidized into less toxic products in oxygenated environments. In pore water of marine sediments, however, no methyl arsine is found. In seawater methylated arsenic compounds are found, which have probably been produced by phytoplankton (Andreae, 1978). Quantitatively the organic arsenic compounds are not important, as is shown by a.o. Byrd (1988) and Brannon (1984).

In fish the compound arsenobetaine (o-fosfatidylmethyl arsoniumlactate) is found (Luten et al., 1983). This compound is very stable and highly water soluble. In seafish arsenobetaine constitutes more than 70% of the total As content.

3.2. BEHAVIOUR IN THE SOIL

In the top soil arsenic generally occurs as arsenate which is strongly bound to iron. The adsorption capacity for arsenate generally decreases in the following order: arsenic-containing soils > clayey soils > calciferous peaty soils > acid woodland soils (Braunerden) > podzol soils > sandy soils.

The behaviour of arsenic in the soil is highly connected with thatof iron because of the strong adsorption of arsenic on iron and through the fact that both elements convert into other forms, under reduced conditions. Under reduced conditions iron(III)hydroxide is decomposed and Fe(II) formed, resulting in a decrease of adsorption sites causing desorption of arsenate to occur. Furthermore, arsenate is converted into arsenate. The two processes cause elevated iron and arsenic contents to occur in reduced groundwater. In mudflats where groundwater containing arsenic and iron comes to the surface the iron will precipitate through the elevated oxygen content. Initially the arsenite will adsorb onto this and subsequently be transformed to arsenate. As a result of these processes in a number of areas in the south and the east of the Netherlands in soils with elevated iron contents also elevated arsenic contents are found (see figure 3.5).

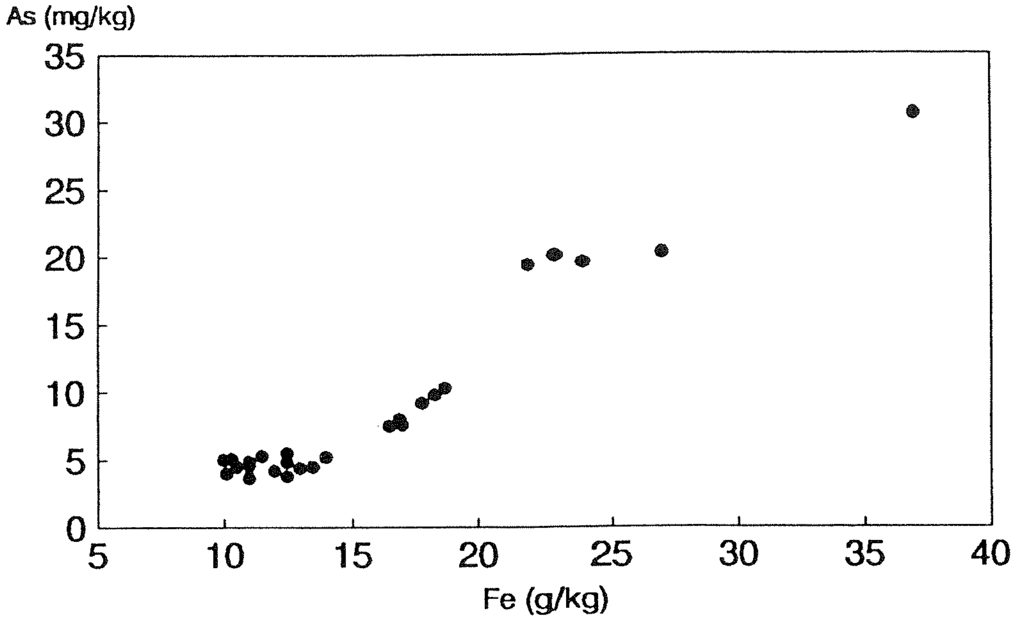


Figure 3.5. Relation between arsenic and iron in the top soil

The behaviour of arsenate in the soil bears great resemblance to that of phosphate. The pH can indirectly influence the As availability by interaction with the anion adsorption capacity, the phosphate balance and the redox potential (Benson, 1953; Frost and Griffin, 1977). For instance in alkaline soils a lower redox potential is required for the reduction of arsenate than in acid soils (see also figure 3.1). The pH affects the adsorption of arsenate considerably (Frost and Griffin, 1977; Pierce and Moore, 1982). The charge of the soil particles also determines the extent of adsorption. This may be illustrated with the pH_{pzc}-value: the pH value at which the surface charge changes from positive into negative. Electropositive minerals will adsorb considerably more anions (and consequently also more arsenate) than the electronegative ones.

Iron hydroxide is electropositive to a pH value of about 8 (depending on the ion power). The positive charge of iron hydroxides over the pH range important to the soil, therefore, renders these compounds an optimal medium for the immobilization of electronegative ions such as arsenate. At even higher pH values also carbonate (calcite) plays a role in the immobilization, probably through coprecipitation of calcium arsenate.

The strong soil chemical dynamics of the element arsenic and the varied origin demonstrate that it is not an easy task to determine criteria for an anthropogenic load. To arrive at an upper limit for the natural background values Lexmond and Edelman (1987) have deduced a formula for soils with an organic matter content lower than 25%:

$[As] = 8 + 0.4 \times L \quad mg.kg^{-1}$ L = weight percentage lutum

and for soils with an organic matter content higher than 25%:

$[As] = 25 + 0.4 \times L \quad mg.kg^{-1}$

Further differentiation with regard to organic matter is used in the Environmental Programme 1988-1991:

$[As] = 15 + 0.4(L+H) \quad mg.kg^{-1}$ H = weight percentage organic matter

L = weight percentage lutum

3.3. BEHAVIOUR IN SURFACE WATER

The behaviour of arsenic in surface water strongly depends on the pH, the redox and the amount of suspended matter. The pH greatly affects the adsorption (figure 3.3) and a considerable pH increase, as occurring in eutrophic lakes such as Lake IJssel, may result in the increase of the dissolved concentration of arsenic. This effect may also result in a net export of arsenic from Lake IJssel to the Wadden Sea.

At high phosphate concentrations competition for the available adsorption sites takes place, causing the adsorption of particularly arsenate to decrease. During precipitation of calcite arsenate may be removed from the solution by coprecipitation. In the marine environment (at equal total concentrations), through ion power effects and the higher pH values (8.2 - 8.3), more arsenic is present in a dissolved form than in freshwater.

The dissolved concentration of arsenic being thus strongly affected by either the presence or the absence of iron(III)hydroxide, the concentration in reduced environments, where iron(III)hydroxide is not stable, is often several times higher than in oxidized environments. Besides arsenate is converted into arsenite which is more readily soluble. The elevated arsenic concentrations in pore water in the deeper sediment layers (figure 3.6) may form a source of arsenic for groundwater as result of perculation.

The behaviour of arsenic during the substance exchange between soil and surface water is practically identical to that of phosphate. In the presence of a sufficiently thick oxidized zone on the sediment, the flux from it is low. The arsenic flux from the sediment increases considerably with a decreasing thickness of the oxidized zone. During anaerobia on the soil sudden explosive fluxes may occur, because the blanketing effect of the iron(III)hydroxide in the oxidized zone of the sediment disappears.

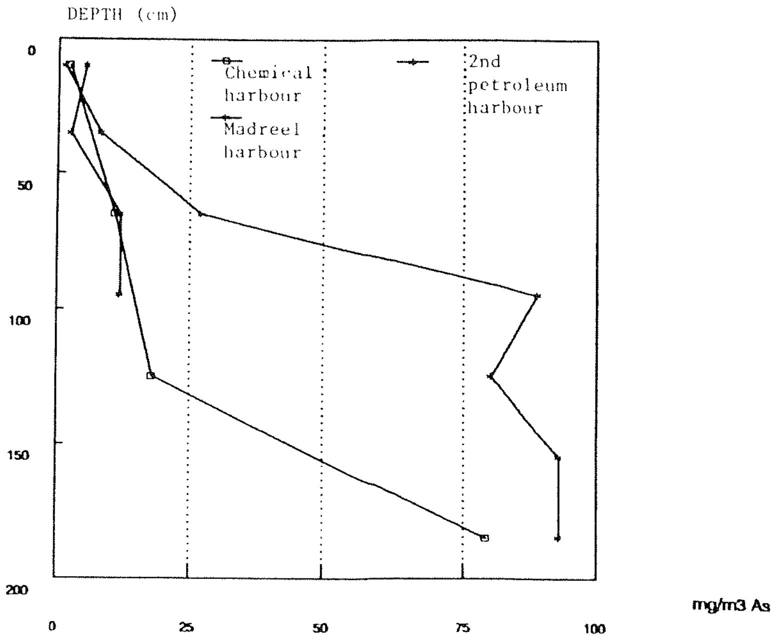


Figure 3.6. Arsenic in pore water of several Rotterdam harbours; elevated As-concentrations in the deeper sediment layers which may form a source of arsenic for groundwater by perculation

Little is known on the behaviour of arsenic in estuaries. In the Scheldt estuary dissolved arsenic is removed from the solution in seaward direction. This removal is probably related to the simultaneous removal of iron.

3.4. BEHAVIOUR IN AIR

In air arsenic mainly occurs aerosol bound (Bennett, 1981; Thijsse and Huygen, 1985); the contribution of arsenic in the gas phase amounts to only a few percent of the total atmospheric concentration. For the transport and deposition properties of As-containing aerosol the particle size dispersal plays an important role. The mass median diameter varies between appr. 0.3 and 3 µm and amounts to 1.1 µm on average (Milford and Davidson, 1985). Measurements of arsenic in aerosol in the Netherlands (Van Jaarsveld and Onderdelinden, 1986) show that in rural areas arsenic predominantly belongs to the finest aerosol fraction (mass median appr. 0.4 µm and 85% below 1 µm). In urban and industrial areas (particularly near metallurgical installations such as copper smelters) the particle size of arsenic is greater, the mass median increasing to the 3 µm mentioned (Fukuzaki et al., 1986). Furthermore, the arsenic particles in airborne soil, c.q. street dust are coarse (Van der Meulen, 1987).

The effective dry deposition rate of the average arsenic-containing aerosol can be estimated at 0.1 - 0.2 $cm.s^{-1}$ (Van Jaarsveld et al., 1986), in the immediate vicinity of a source the initial value amounts to 0.2 $cm.s^{-1}$. During transport of the atmospheric aerosol the deposition rate decreases by preferential removal of the larger particles as a result of dry and wet deposition. Thus a representative value of appr. 0.1 $cm.s^{-1}$ for the dry deposition rate of arsenic is found for outdoor air. The removal through dry deposition takes place at an average rate of 0.5% per hour. Under the conditions prevalent in the Netherlands wet deposition takes place at an average rate of 1.2 - 1.5% per hour.

Thus for the lifetime of the atmospheric arsenic aerosol by means of the above-mentioned data a value of appr. 2.5 days is found. During this residence time, more or less independent of the height of the source, the arsenic aerosol may be transported over 1000 km.

For (extremely) coarse aerosol, originating from airborne dust, the deposition rate is considerably greater (to a factor 10), making the transport distance appr. 2 to 3 orders of magnitude smaller (Van der Meulen et al., 1984).

3.5. BEHAVIOUR IN BIOTA

In aquatic organisms a slight to moderate bioaccumulation of inorganic arsenic occurs. A number of studies (see appendix) show that biomagnification (accumulation via the food-chain) of inorganic arsenic does not occur. For instance in organisms of lower trophic levels higher bioconcentration factors are found than in organisms of higher trophic levels (for algae, invertebrates and fishes a factor of 700, 400 and 150, respectively). On the other hand the organic arsenic is accumulated in food chains, the primary producers converting the inorganic arsenic taken up into organic arsenic. It is assumed that in algae the uptake particularly occurs through replacement of phosphate with arsenate in view of the great similarities in behaviour of these compounds.

The uptake of arsenic from the sediment by biota is slight.

With regard to the soil for most crops no relation has been found between the As content in the crop and the corresponding soil (Wiersma et al., 1985).

3.6. SUMMARY AND CONCLUSIONS

The behaviour of arsenic in the soil and in aquatic systems is dependent on various processes the most important ones being oxidation-reduction, precipitation, adsorption-desorption, ligand formation and biomethylation. Regarding the effects the trivalent arsenic (arsenite) is the most important. The distribution As(III)/As(V) mainly depends on the redox potential and the pH. The redox reactions involved are catalysed by manganese oxides.

Arsenate in soil and groundwater is to a high extent adsorbed onto iron hydroxides. Under reducing conditions, however, iron(III)hydroxide may convert into divalent dissolved iron, the arsenic also dissolving as arsenite. The arsenic content in the soil is related to the organic matter content and the lutum fraction of the soil. In oxygenated water arsenic is mainly found as arsenate; in anoxic water as arsenite. In reduced marine sediments arsenic sulphide complexes occur. Under methanogenic conditions and at low pH values arsenic hydride (arsine) may arise.

The adsorption behaviour of arsenic is highly comparable to that of phosphate and is influenced by the pH. Particularly in stagnant eutrophic lakes at increased pH values the adsorption of arsenate on suspended material may decrease by competition for adsorption sites with the phosphate present.

In air arsenic is mainly found aerosol bound. The mass median diameter of arsenic-containing aerosol ranges from appr. 0.3 to 3.0 μm. Removal through dry deposition amounts to 0.5% per hour, that through wet deposition under Dutch conditions 1.2 - 1.5% per hour. The average life of arsenic aerosol in air is, therefore, appr. 2.5 days, corresponding with a transport over a distance of appr. 1000 km.

Inorganic arsenic is little bioaccumulative and does not accumulate in food chains. Bioaccumulation and biomagnification, however, do occur in organic arsenic, which is produced by algae.

4. CONCENTRATIONS, FLUXES AND EXPOSURE LEVELS

4.1. SAMPLING AND MEASURING METHODS

In the procedures for the determination of arsenic in environmental samples 4 steps may be distinguished: (a) sampling, storage and preservation; (b) pretreatment of the samples; (c) rendering soluble; (d) analysis. In addition a distinction can be made between procedures aimed at the total determination of arsenic and those aimed at the determination of the speciation of arsenic.

4.1.1. Sampling, storage, preservation and rendering soluble

- Soil and groundwater

Sampling of soil may be carried out with a spade or auger (OKB, 1988). Storage should preferably take place in polyethene bottles or jars. Prior to taking subsamples for analysis the sample need to be homogenized. This may be done prior or following drying. Drying may take place at 40 ^{o}C or by means of freeze drying. A much applied method of rendering soluble for the determination of total arsenic in soil is that with aqua regia (NEN 6465, 1981). Different mixtures of acids may also be applied. Rendering soluble may also be carried out by means of a teflon bomb.
During sampling groundwater need to be filtered anaerobically through a filter of 0.45 μm (OKB, 1988) and be collected in a polythene bottle. To preserve the sample for total arsenic it need to be acidified to a pH value of 2. Depending on the analysis method (see relevant section) the sample should be rendered soluble with aqua regia (NVN 6432, 1988). To determine the distribution of arsenic over various fractions in soil it can be extracted with a range of extracting agents of increasing concentrations (IB, pers. comm.). The biological availability of arsenic may in principle be determined in this way.

- Surface water

Sampling is carried out by taking samples in polyethene bottles. For the determination of total arsenic the sample is rendered soluble with aqua regia (NVN 6432, 1988). For the determination of the dissolved fraction the sample is filtered over a 0.45 µm filter, subsequently the filtrate is acidified with HNO_3 to a pH value of 2. If the speciation pattern is to be determined, acidification is omitted. In sampling sediments the mudsampler is less likely to disturb the particle size composition (Siebers and Donze, 1984). For speciation studies in sediments reference is made to soil and groundwater.

- Air

Aerosol bound arsenic can be sampled with a "High Volume Sampler" according to NEN 2785, applied to arsenic. The arsenic collected on the filter may be liberated by rendering soluble of the sample with aqua regia (NEN 6465, 1981). Rendering soluble can also be carried out in a teflon bomb (this is done in the Air Measuring Network).

- Biota

The sampling depends on the nature of the material and the intended purpose of the analysis. If necessary subsamples should be taken, which are reduced again and homogenized. The pretreatment procedure may also contain a drying step, preferably by means of freeze drying. Biota samples are frequently rendered soluble by heating a mixture of nitric acid and sulphuric acid, followed by a treatment with perchloric acid and hydrogen peroxide (Vaessen et al., 1986). Rendering soluble can also be carried out in a teflon bomb according to NEN 6465, subsequently the sample can be processed according to NVN 6432.

4.1.2. Measuring methods

- Determination total arsenic content

A technique which is frequently used for the determination of total arsenic in liquid samples is atomic absorption spectrometry, by means of flames (flame-AAS), graphite ovens (GO-AAS, NEN 6457, 1981) or the hydride generation technique (hydride-AAS, NVN 6432, 1988). The detection limits of these techniques are 20 $\mu g.l^{-1}$, 1 $\mu g.l^{-1}$ and 0.2 $\mu g.l^{-1}$, respectively. With voltammetry following reduction to As(III) total arsenic can be determined up to a level of 0.2 $\mu g.l^{-1}$ (Cleven and Wolfs, 1988). With spectrophotometry arsenic in solution can de determined, such as destruates of biota, with a detection limit of 0.15 μg absolute, which is 0.03 $\mu g.l^{-1}$ for a 5 g sample (Vaessen et al., 1986).

For air dust following rendering soluble in the teflon bomb and analysis by means of GO-AAS which is common practice in the National Air Measuring Network, the detection limit is appr. 0.5 $ng.m^{-3}$. In solid samples arsenic may be determined with the neutron activation analysis with the detection limit ranging from 0.1 to 0.5 $\mu g.g^{-1}$, depending on the matrix (De Bruin et al., 1982).

- <u>Speciation arsenic</u>

For the determination of the speciation pattern of arsenic in aqueous samples, the distinction between organic and inorganic arsenic in particular, the following procedures are available:

- selective hydride generation followed by AAS-detection (Anderson et al., 1986);
- hydride generation, collecting of the hydrides at liquid nitrogen temperature, followed by selective evaporation of the various arsenic (hydride) species and AAS detection (Howard and Arbab-Zavar, 1981);
- idem, however, in this procedure the hydrides are evaporated simultaneously, separated with GC and detected with AAS, FID detection or MS (Ebdon et al., 1988);
- separation of the various arsenic species by means of ion exchange chromatography, followed by hydride generation and AAS detection (Pacey and Ford, 1981, Tye et al., 1985);
- voltammetric techniques (Henry et al., 1979).

Of these procedures the second and the third methods have the lowest detection limits.

For the determination of the speciation in biota these can be extracted with NaOH under reflux (Maher, 1981). Subsequently the extract is acidified and processed according to one of the procedures mentioned above.

4.2. BACKGROUND CONCENTRATIONS

The arsenic concentration in soil may vary greatly depending on the type of soil and the hydrology. The average concentration is 11.3 $mg.kg^{-1}$ (Ure and Berrow, 1982). In areas with soils rich in iron natural arsenic contents of op to 600 $mg.kg^{-1}$ dry matter are sometimes found. In the south and the east of the Netherlands high natural concentrations of up to 390 $mg.kg^{-1}$ dry matter occur in iron ore layers lying on the surface (concentrations are the average of six observations; Pruissen and Zuurdeeg, 1988; Oranjewoud, 1987).

The natural background concentration in surface water amounts to a appr. 1-2 $\mu g.l^{-1}$ (WL, 1987).

The natural background concentration in air in situations where air contains little dust (Antarctica) amounts to 0.02 - 0.3 $ng.m^{-3}$; in situations where air contains more dust (such as in the Netherlands) this level is an estimated 1 - 2 $ng.m^{-3}$.

4.3. OCCURRENCE IN SOIL AND GROUNDWATER

4.3.1. Concentrations in the soil

Recently the Measuring Programme Soil Quality was initiated. To this end, at 40 locations samples were taken from the topsoil at two (different) depths below groundlevel (0 - 10 cm and 10 - 30 cm -gl). The locations covered 10 different combinations of soil usage/type of soil, each combination being sampled at four locations in the Netherlands. At each sampling location four replication samples were analyzed for each depth. The samples were analyzed for arsenic with a detection limit of 0.15 $mg.kg^{-1}$. Table 4.1. shows the results after statistical calculations (De Kwaadsteniet, 1989).

Previously studies were carried out on the As contents in soil from nature reserves and agricultural and horticultural land.

The arsenic contents in soil from nature reserves in the 0 - 10 cm layer varies from 1.4 to 33 $mg.kg^{-1}$ d.m. (table 4.2.).

Table 4.1. 95% Confidence intervals for the average arsenic content (mg/kg) for 10 different combinations of soil usage/soil composition at different depths (detection limit: 0.15 mg/kg)

Soil usage/-composition	Depth (- ground level) 0 - 10 cm	 10 - 30 cm *
Grassland/feld podzol poor in loam	0.5- 1.9	0.5- 1.8
Grassland/plaggept (man-made humus) soil	1.0- 6.6	1.5- 6.1
Grassland/ peaty soil	1.9-47	1.7-50
Agricultural land/calcareous heavy sandy clay-light clay soil	12 -22	10 -18
Agricultural land/feld podzol poor in loam	0.8- 2.2	0.5- 1.5
Agricultural land/plaggept (man-made humus) soil	0.9- 6	0.6- 5
Orchard/calcareous heavy sandy clay-light clay soil	5.9-20	6.0-21
Orchard/plaggept (man-made humus) soil	1.6- 4.4	1.7- 4.4
Woodland/feld podzol poor in loam	0.5- 1.4	0.6- 0.8
Woodland/typic psannaquent poor in loam	1.2- 3.3	0.6- 2.1

* for agricultural land: depth 30 - 50 cm

Table 4.2. Average As content ranges in the 0 - 10 cm layer in soil from nature reserves, in mg/kg d.m. (Edelman, 1983)

Type of soil	As content
Sand	1.4 - 8.6
Sandy clay/loam	5.1 - 18.0
Clay	12.0 - 21.0
Peaty clay	11.0 - 33.0
Peat	4.7 - 24.6

Table 4.3. shows the average As contents and the distribution in the 0 - 20 cm layer in Dutch agricultural land (Van Driel and Smilde, 1981). In river clay lands relatively high arsenic contents were found, particularly in the Rhine, in which in comparison with the Meuse, especially from the fifties to the beginning of the seventies considerable amounts of arsenic rich silt were deposited on the river forelands (see also section on river forelands mentioned below).

As contents in a number of greenhouse- agricultural- and horticultural lands are given in table 4.4.

Table 4.3. Arsenic contents in Dutch agricultural lands in mg/kg d.m. in the 0 - 20 cm layer (Van Driel and Smilde, 1981)

	As contents	
	Average	*Range*
Marine clay	*13*	*3.1 - 29*
River clay (Rhine influence)	*67*	*7.3 - 205*
River clay (Meuse influence)	*30*	*4.1 - 91*
Loess	*8.3*	*6.1 - 12*
Reclaimed peatland	*4.9*	*0.2 - 11*
Peaty soil	*12*	*1.7 - 21*
Sandy soil	*7.8*	*1.7 - 24*

Table 4.4. Arsenic contents in Dutch soils (0 - 20 cm layer) in mg/kg d.m. (Wiersma et al., 1986)

	Number of samples	*Range min-max*	*Average*	*90% below*
Agricultural and horticultural lands	*704*	*0.1-110*	*12*	*22*
Greenhouse lands	*155*	*1 - 35*	*12*	*20*

Until recently sodium arsenite (the use of which was legally permitted in 1953) was applied on a large scale in the Netherlands as a defoliant in the cultivation of potatoes. Since 1960, because of the damage caused to following crops on sandy soils (e.g. beans, peas), it was only used on clay and sandy clay soils. Because it was feared that at long-term application phytotoxic effects would also occur in clay and sandy clay soils its use was prohibited for all soils in 1985. The annual use of sodium arsenite in agriculture is illustrated in table 4.5.

Table 4.5. Annual use of sodium arsenite in agriculture for the years 1974 - 1980 (Dolman, 1981)

Year	*Use (in tonnes)*
1974	*432.5*
1975	*403.5*
1976	*380*
1980	*60*

Assuming an application of arsenite once every four years, and a use of 10 kg As per ha per application, an increase of the As content of 20 - 40 $mg.kg^{-1}$ in the soil (0 - 20 cm) can be calculated for the period 1953 through 1984. Comparing this with the natural situation the additional arsenic is presumably present in a mobile form.

For this situation model calculations with the chemical model CHARON showed an elevation of the dissolved arsenic content in the soil liquid in the 0 - 20 cm layer of 3 $\mu g.l^{-1}$ in 1950 (before arsenic was being applied) to 65 $\mu g.l^{-1}$ in 1985 (after the ban on the use of arsenic), with an increase of the interchangeable arsenic in solid matter of 3 - 40 $mg.kg^{-1}$. This model calculation is only indicative, calibration as well as verification of the model were not carried out. Boesten (1982) also predicted a significant elevation of the As concentration in the soil liquid in the top layer.

According to this model calculation the concentration in soil liquid will also after 1985 continue to increase, if more phosphor fertilizer is applied than is taken up by plants.

Despite studies by Loch (1982) and Boesten (1982) in particular little is still known on the behaviour of arsenic in these soils. Attention was only given qualitatively on the effects of fertilizer on the mobility and the washing away behaviour (competition of P with As for available adsorption sites) (see chapter 3).

- <u>River forelands</u>

With regard to the river clay the data in table 4.3. are slightly dated and rather high. From time ranges for recently deposited silt on the river forelands of the Rhine, it may be deduced that the concentrations between 1958 and 1972 have decreased (table 4.6.), while the arsenic content of the Rhine water in the period 1972 - 1987 has further decreased (see 4.4.).

The As concentrations in the river forelands of the river Meuse are, compared with the values observed in agricultural land, not or hardly elevated. The calcareous soils appear to have the highest concentrations with an average of appr. 20 $mg.kg^{-1}$ (min-max : 7.8-34.2). With increasing flooding frequency the concentrations are generally higher (Van Driel et al., 1987).

Table 4.6. *Arsenic contents at 50% < 16 micron in river forelands at various locations along the Rhine and the Waal (Zschuppe et al., 1988)*

Rhine silt	*1958* *n = 198*	*1970* *n = 136*	*1972* *n = 94*
German-Dutch border	*82*	*62*	*28*
Spijk		*39*	*28*
Pannerdense Kop	*77*		*32*
Doornik/Bemmel	*78*	*40*	
Lent	*123*	*65*	
Oosterhout		*38*	
Winssen		*33*	
Deest		*46*	
IJzendoorn		*39*	
Tiel		*38*	
Varik		*56*	*26*
Hellouw		*45*	
Dalem		*62*	
All locations (av.)	*90*	*47*	*29*

4.3.2. Concentrations in groundwater

The average As concentrations in groundwater amounts to appr. 8 $\mu g.l^{-1}$ (total reduced; oxidized groundwater contains appr. 2 $\mu g.l^{-1}$, RIVM, 1985). In the Netherlands higher concentrations are, however, not uncommon, especially because some layers of soil contain much natural arsenic (see 4.1.). The soil usage can also influence the As concentration in groundwater. For instance in groundwater under agricultural lands a higher As concentration is found than in nature reserves (table 4.7.). Furthermore, the type of soil influences the As concentration in groundwater (table 4.8.).

Since clay soils contain the highest As contents, much arsenic will dissolve under reducing conditions, resulting in higher As concentrations in the groundwater of clay soils than in that of other types of soil. Generally 10 $\mu g.l^{-1}$ is not exceeded in the Netherlands.

Table 4.7. Average As concentrations (μg/l) in fresh groundwater at various depths broken down into type of soil and use (RIVM, 1985)

Type of soil	*Sand*			*Clay*			*Peat*		
Number of meters under groundlevel	*10*	*15*	*15*	*10*	*15*	*25*	*10*	*15*	*25*
Farmland	*5.0*	*2.7*	*2.2*	*2.7*	*8.3*	*2.8*	*2.7*	*3.4*	*3.1*
Grassland	*4.4*	*4.3*	*3.0*	*8.5*	*5.3*	*5.6*	*2.0*	*2.1*	*1.8*
Nature reserve	*2.3*	*3.2*	*2.3*	*1.8*	*3.8*	*2.8*	*3.1*	*6.1*	*3.7*
Residential areas	*3.3*	*11*		*4.1*	*2.4*	*1.6*	*1.4*		
Dunes	*1.8*	*1.8*	*1.6*						

Table 4.8. Arsenic contents in μg/l in fresh groundwater with respect to the type of soil (RIVM, 1985)

Meters under groundlevel	*Sand*	*River clay*	*Marine clay*	*High moor peat*	*Peat moor*	*Loam*
10	*3.8*	*9.1*	*2.3*	*2.3*	*2.3*	*3.0*
25	*2.9*	*5.1*	*1.9*	*2.7*	*2.1*	*3.2*

In North Brabant, however, at some places elevated contents are found at greater depths (40 to 300 m). The contents very seldom exceed 50 $\mu g.l^{-1}$, however, they are usually above 10 $\mu g.l^{-1}$. The shallow groundwater at these sites contained lower concentrations, therefore, the "As contamination" of the deep groundwater could not be caused by local pollution of the soil. Studies by Flink (1985) showed that this elevation is caused by natural sources and that the arsenic originates from glauconite layers in North Belgium. At these depths arsenic occurs as arsenite which adsorbs less on clay minerals and iron(hydr)oxides than arsenate does.

The RIVM has recently reported extensively on the quality of groundwater in the Netherlands (Van Duijvenbooden et al., 1989). With regard to arsenic the findings are consistent with the above-mentioned.

4.4. OCCURRENCE IN SURFACE WATER AND SEDIMENT

4.4.1. Concentrations in surface water

- Freshwater environment

In the freshwater environment a distinction can be made between the state-owned waters and the provincial and municipal waters. Besides within the freshwater environment a distinction can be made between the surface water, dissolved as well as particular, and the sediment, for solid material as well as for pore water.

State-owned waters

A review of the concentrations in the Rhine and the Meuse basins is shown in tables 4.9 and 4.10. As is the case with the Rhine the content of arsenic in the Meuse water at Eijsden has decreased considerably since 1980. Here also the concentration appeared to have stabilized at 2 $\mu g.l^{-1}$ since 1984.

Table 4.9. Concentration total (T-As) and dissolved (D-As) arsenic at 4 locations in the Netherlands (µg/l)

Year	*Lobith*		*Hagestein*		*Vuren*		*Kampen*	
	T-As	*D-As*	*T-As*	*D-As*	*T-As*	*D-As*	*T-As*	*D-As*
1974	*5.2*	*3.5*	*3.0*		*3.0*		*5.0*	
1975	*4.5*	*2.7*	*6.8*	*4.8*	*5.5*	*4.0*	*5.3*	*4.0*
1976	*6.4*	*3.2*	*3.9*	*1.8*	*2.6*	*2.5*	*3.1*	*1.8*
1977	*4.5*	*2.8*	*7.0*	*5.5*	*6.5*	*5.0*	*5.5*	*4.0*
1978	*4.3*	*2.2*	*5.1*	*2.4*	*4.9*	*2.3*	*4.5*	*3.1*
1979	*4.0*	*2.2*	*5.3*	*2.4*	*4.1*	*2.1*	*4.6*	*1.5*
1980	*3.1*	*1.8*	*3.3*	*2.3*	*3.8*	*2.4*	*3.3*	*2.5*
1981	*3.6*	*2.1*	*4.3*	*2.1*	*4.9*	*2.3*	*3.2*	*2.6*
1982	*4.0*	*2.4*	*4.1*	*2.7*	*4.5*	*2.5*	*4.0*	*2.7*
1983	*3.6*	*1.8*	*4.0*		*3.5*		*3.0*	
1984	*2.0*	*1.4*	*2.1*		*2.3*		*1.8*	
1985	*1.8*	*1.3*	*2.1*	*1.7*	*1.9*	*1.4*	*1.7*	*1.4*
1986	*2.4*	*1.5*	*2.1*	*1.5*	*2.0*	*1.4*	*2.0*	*1.4*

Table 4.10. As concentration (total and dissolved in µg/l) at 3 locations in the Meuse basin in the Netherlands

Year	*Eijsden*		*Keizersveer*		*Lith*	
	T-As	*D-As*	*T-As*	*D-As*	*T-As*	*D-As*
1976	*20.4*	*18.0*			*7.4*	*5.3*
1977	*9.6*	*7.6*	*8.0*	*6.0*	*5.9*	*3.9*
1978	*15.8*	*12.9*			*5.7*	*4.5*
1979	*13.0*	*9.6*	*10.0*	*7.0*	*6.2*	*4.8*
1980	*14.2*	*10.7*			*9.3*	*6.9*
1981	*8.6*	*5.4*			*4.9*	*3.2*
1982	*5.8*	*4.5*				
1983	*3.2*	*1.8*				
1984	*2.4*	*1.6*	*2.0*	*2.0*		
1985	*1.9*	*1.7*	*1.6*	*1.4*	*2.0*	*1.8*
1986	*2.0*	*1.5*	*1.8*	*1.4*	*1.7*	*1.3*

Lake IJssel-Lake Ketel and the Northern Delta basin are sedimentation basins of the Rhine, the Rhine and the Neuse, respectively. Through the sedimentation of particular bound arsenic the total contents of arsenic in surface water decreases (table 4.11.).
As with the big rivers the arsenic concentrations in the lake systems appear to decrease up to and including 1983 to almost stabilize over the period 1984 - 1986. Also in Lake Veluwe and Lake Eem the arsenic concentrations decreased considerably, between 1983 and 1984 in particular. Arsenic balances for Lake IJssel and the Haringvliet show that in 1982 accumulation of arsenic still occured in the basins, whereas in 1986 a net export of arsenic from these basins into the North Sea and the Wadden Sea was observed. This net export is the result of remobilization of arsenic from the sediment (Salomons and Forstner, 1984).

In table 4.12. the As concentrations are given of the Dutch IJssel, the North Sea Canal and the Canal from Ghent to Terneuzen as mentioned in the quarterly reports published by the Department of Public Works on the quality of surface water in the Netherlands for the years 1984 through 1986.

Table 4.11. As contents (total and dissolved in µg/l) in the surface water of Lake IJssel, Border Lakes and the Northern Delta basin

Location	1982		1983		1984		1985		1986	
	T-As	D-As	T-As	D-As	T-As	D-As	T-As	D-As	T-As	D-As
	New Merwede-Hollands Diep-Haringvliet									
NM15	4.0		2.8		2.3		1.6	1.2	1.5	1.3
H9	4.0		2.7		3.2		2.1	1.9	1.6	1.5
H12	4.1	3.7	2.8	2.4	2.2	2.0	2.4	2.2	2.0	1.8
	Lake Ketel-Lake IJssel									
Ket.	2.8	2.8	2.8	2.2	1.8	1.7	2.0	1.6	1.7	1.4
Y23	3.4	2.9	3.0	2.0	1.8	1.8	2.2	1.7	2.1	1.6
Y1	3.1		3.0		2.0		2.2	1.8	2.2	1.9
Y2	3.0		2.8		1.8		2.1	1.9	1.9	1.7
	Lake Veluwe-Lake Eem									
V8	2.3		2.6		1.0		1.0	0.7	0.9	0.7
Y125	4.0		4.2		2.3		2.2	1.9	2.5	2.4

Table 4.12. As concentrations (total and dissolved in µg/l) in several remaining state-owned waters

Location	1984		1985		1986	
	T-As	D-As	T-As	D-As	T-As	D-As
Dutch IJssel	2.7		2.3	1.7	2.7	2.3
North Sea Canal	2.6		3.3	2.9	3.2	2.9
Ghent-Terneuzen Canal	5.0	4.6	5.1	4.3	4.6	3.8

Non-state-owned waters

In table 4.13. the As concentrations are shown as measured in the past few years in various frameworks and by various agencies.

Table 4.13. *As concentrations (total in µg/l, average value) in non-state-owned waters (CCRX, 1988)*

Location	*1983*	*1984*	*1985*	*1986*	*1987*
Bodegraven-North	*7.0*	*2.0*			
Middle-Delfland	*8.5*	*5.0*			
Central Land Reclamation			*5.0*		
Polder Board Delfland			*5.1*		
Regge and Dinkel			*1.8-2.8*		
Polder Board West Brabant				*2-3*	*2-3*

- Marine water environment

The total and dissolved arsenic in the Dutch part of the North Sea is shown in table 4.14. The highest contents are found in the West Scheldt. The total As content in the West Scheldt decreases in westerly direction. In the Scheldt estuary (on Belgian territory) arsenic is subject to adsorption processes in the water column, resulting in a considerable decrease of the dissolved concentrations (Salomons and Forstner, 1984).

Table 4.14. *As concentrations (total and dissolved in µg/l) in the North Sea and related waters in 1986 (CCRX, 1988)*

Location	*Total arsenic*	*Dissolved arsenic*
North Sea 70 km off the coast	*1.6-1.7*	*up to 1.5*
North Sea 20-30 km off the coast	*1.9-2.6*	*1.3-2.1*
North Sea 0-10 km off the coast	*2.1-9.5*	*1.4-2.5*
Eems-Dollard	*3.0-4.8*	*1.0-1.3*
East Scheldt	*2.9*	*1.9*
West Scheldt	*7.0-10.6*	*2.3-5*

4.4.2. Concentrations in sediment

The concentrations of arsenic in sediments are dependent on for instance their compositions and origins. To be able to compare these sediments with each other adjustment for the composition is necessary. An adjustment which was previously commonly used is the conversion of the concentration measured into a standardized concentration at 50% particles < 16 µm. To this end a number of samples taken from one location is analysed for the percentage < 16 µm and the arsenic content. Through the points obtained a regression line is calculated and subsequently the contents at 50% < 16 μm are used for a comparison with other locations. A different adjustment method used is to separate the fraction < 63 μm and to determine its As content (Forstner and Salomons, 1980). The contents in this fraction roughly correspond with those at 75% < 16 μm of the regression methods (Kerdijk, 1989).

- <u>Freshwater environments</u>

Lake Ketel, the Hollands Diep and the Rotterdam Harbours are sedimentation areas with polluted river silt. The results of an inventory of a number of state-owned waters is given in table 4.15.

<u>Table 4.15</u>. As content (in mg/kg dry matter) in sediments in state-owned waters (1983-1984) (Kerdijk, 1985)

Location	*Absolute contents 10, 50 and 90 percentiles*			*Adjusted contents * 10, 50 and 90 percentiles*		
	10	*50*	*90*	*10*	*50*	*90*
Rhine and Rhine branches	*5*	*20*	*43*	*16*	*25*	*78*
Meuse and Meuse branches	*4*	*16*	*32*	*17*	*27*	*35*
Dutch IJssel	*5*	*18*	*32*		*20*	*25*
Rotterdam harbours	*14*	*22*	*37*	*16*	*23*	*40*
New Meuse	*3*	*10*	*18*	*10*	*15*	*23*
Ghent Terneuzen Canal				*41*	*58*	*109*
Amer					*34*	
New Merwede					*36*	
Hollands Diep					*28*	
Haringvliet					*27*	

** adjusted contents are contents at 50% < 16 μm*

For a number of sediments in the Northern Delta basin the course in time has been described by Kerdijk (1985). In figure 4.1. this is shown graphically.

From Lake Ketel and Lake IJssel no recent measurements are available with regard to the sediment quality. An inventory study, carried out in 1974, showed that no great regional variations in the As contents occurred in Lake IJssel (WL, 1976). The contents at 50% < 16 μm amounted to an average of 33 $mg.kg^{-1}$ and were a little over 40 $mg.kg^{-1}$ for Lake Ketel.

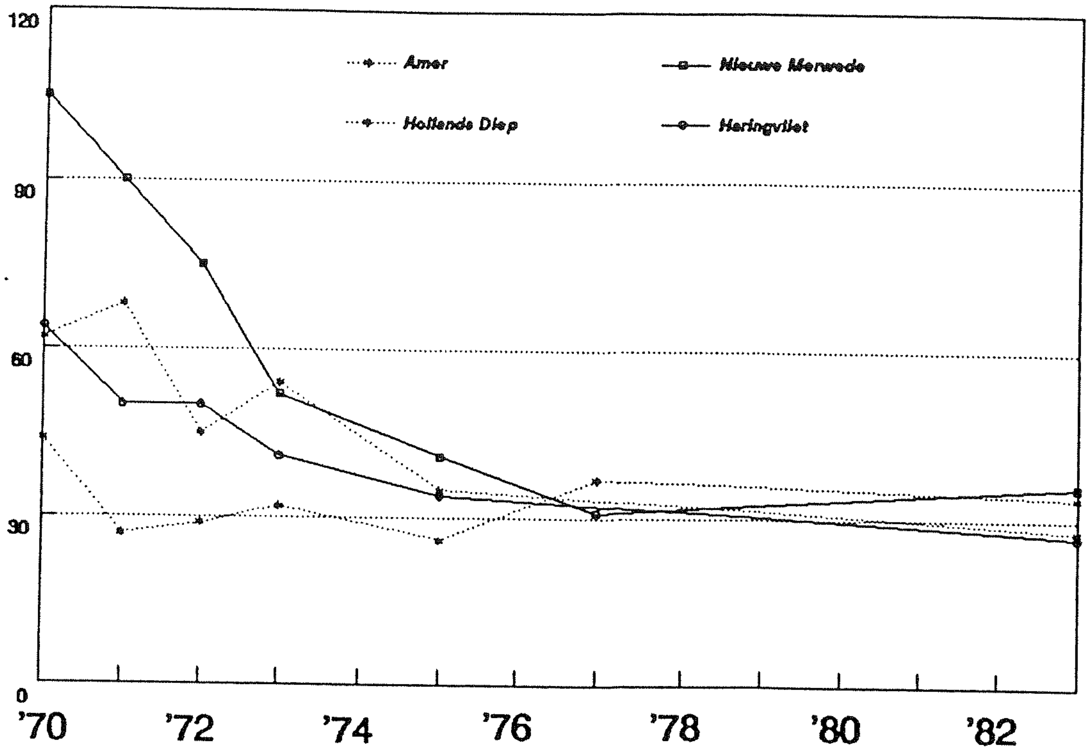


Figure 4.1. The course of the As content (mg/kg d.m. at 50% < 16 μm) in the Northern Delta basin in the period 1970-1983

For the provincial and municipal sediments relatively few data are available. An indication is obtained based on the available (non-standardized) data which are mentioned in table 4.16. These values correspond with the results of extensive studies on the quality of the sediment of 15 different waters in North Brabant, which are shown in the same table.

Table 4.16. As content (in mg/kg d.m. not adjusted) in sediments in provincial waters in the period 1983-1986 (PZH, 1986, 1987; PS, 1987)

Location	Average	Standard deviation
Groningen	9	6
Bodegraven-North	17	6
Middle Delfland	10	6.5
Central Land Reclamation	11	4
Bulbgrowing area	7	4

North Brabant	Contents:	<1	1-3	3-10	11-29	30-58
(n=269)		13%	19%	31%	30%	7%

For the Amsterdam canals arsenic contents of 2.2 to 67 $mg.kg^{-1}$ dry matter were found in the sediment (Heida, 1985).

- Marine water environment

In figure 4.2. the results are given of studies carried out in 1980-1981 on the As content of the sediment of the Wadden Sea (Kerdijk, 1985). A distinct geographical tendency is not observed, however, in the east the concentration of arsenic appears to decrease slightly.

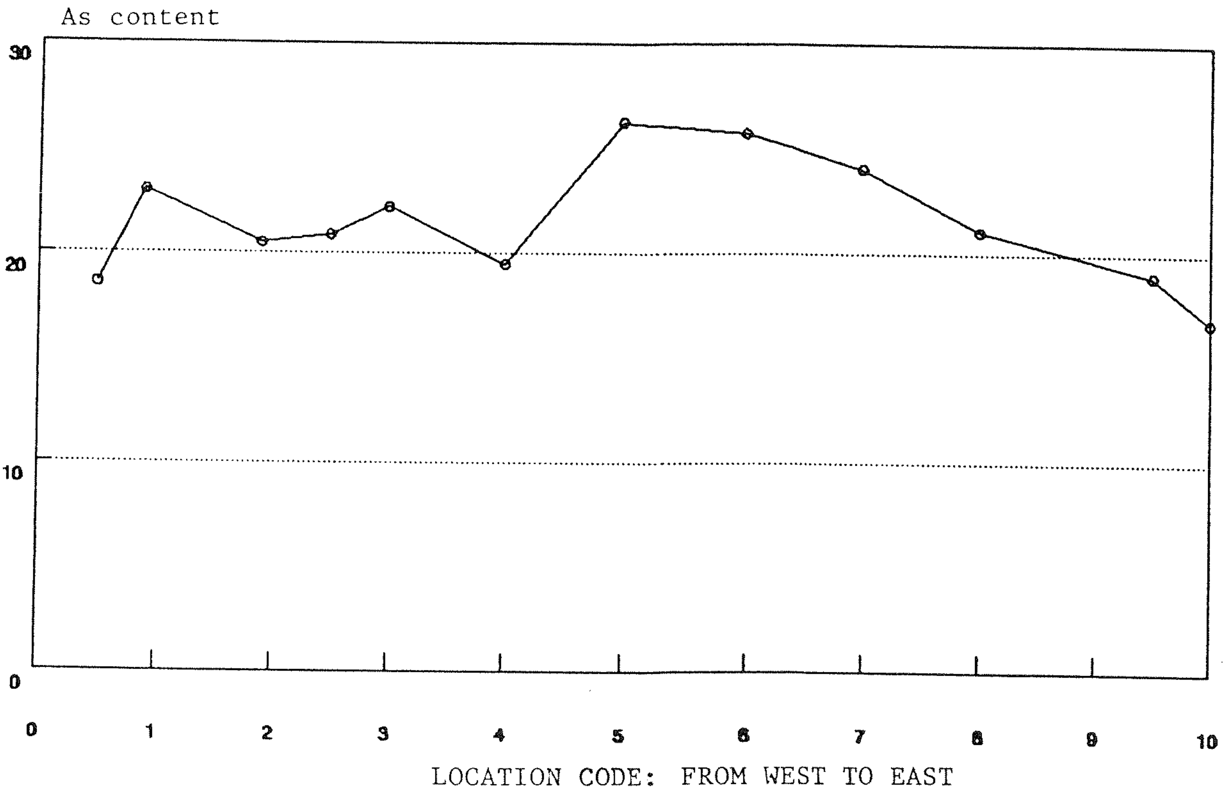


Figure 4.2. As content (in mg/kg d.m. at 50% < 16 µm) in sediment of the Wadden Sea in the period 1980-1981, from west to east

In figure 4.3. the development of the As content in the West Scheldt estuary is shown for 1979 and 1984. The As content decreases considerably the closer the location is situated to the North Sea. This decrease in the Scheldt estuary is mainly the result of the decreasing contribution of the polluted Scheldt silt to the sediments.

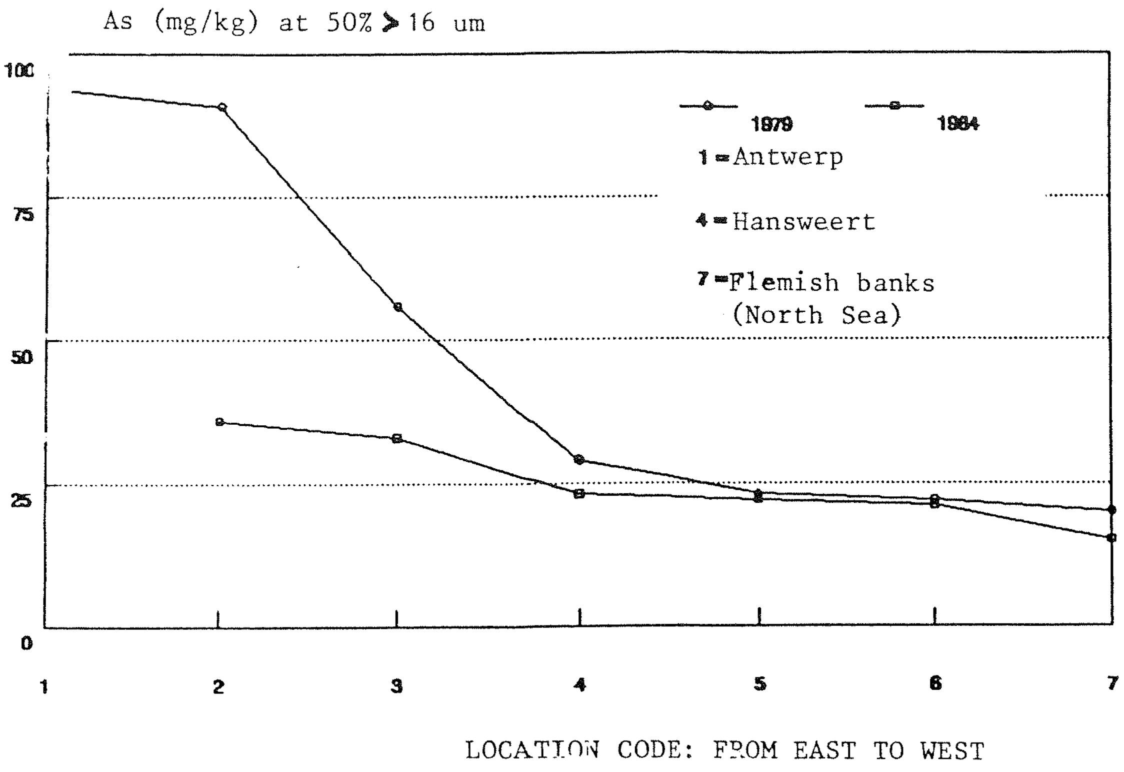


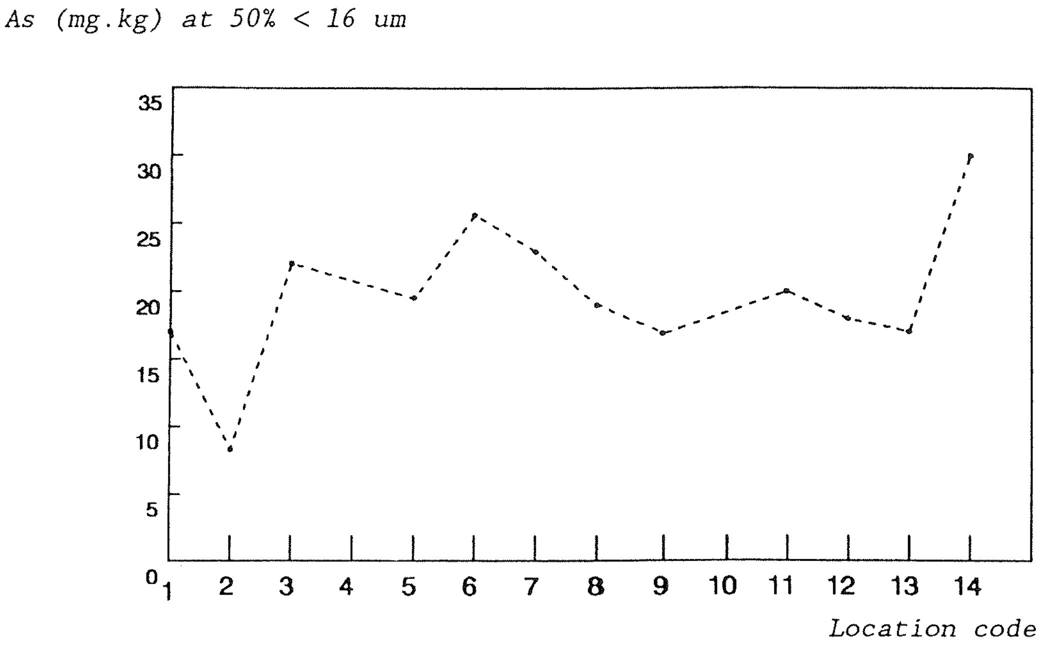
Figure 4.3. *As content (in mg/kg d.m. at 50% < 16 μm) in sediment of the West Scheldt in the period 1979 and 1984, from east to west*

For the North Sea sediments Kerdijk (1983, 1984) gives a 50-percentile value of 22 mg.kg^{-1} As (adjusted for 50% < 16 μm), as well as 10 and 90 percentile values of 14 and 35 mg.kg^{-1}.

Considering the Dutch coast as part of the North European coast (figure 4.4.), the arsenic contents in the Netherlands may be compared with those off the Belgian and German coast (Salomons, 1987). There is no distinct tendency in the arsenic contents of the marine sediments along the coast of Belgium-the Netherlands-Germany, indicating the absence of great local sources.

4.4.3. Concentrations in pore water

Arsenic dissolved in pore water of sediments is infrequently measured. The only available data are from the WL, where in 1977 and 1978 pore water was studied at various locations in the Rhine and the Meuse basins (table 4.17.). Besides data are available on the sediments in the Rotterdam harbours (see also figure 3.6).



1 =Seine
2 =Somme
3 =Flemish banks
4 =East Scheldt
5 =Balgzand
6 =Friesland
7 =Groningen
8 =Dollard
9 =Kanalpolder
10 =Leybocht
11 =East Friesland
12 =Jade Bay
13 =Weser
14 =Elbe

Figure 4.4. As content (in mg/kg at 50% < 16 µm) along the North Sea coast between the Seine and the Elbe

Table 4.17. Concentration arsenic (in µg/l) in pore water of sediments of the Rhine-Meuse basin (ZMAS, 1984) and in pore water of dredgings from the Rotterdam harbour (Kerdijk et al., 1984)

Location	*As concentration in pore water*	*As concentration in surface water*
Meuse, 1978	*4.7*	*2.1*
New Merwede, 1978	*2.8*	*1.1*
Haringvliet, 1978	*11*	*1.1*
Lake Ketel, 1977	*12*	*0.8*
Lake IJssel, 1977	*13*	*0.9*
Rotterdam-East	*8*	*1.5*
Rotterdam-West	*45*	*0.8*

The concentrations in the pore water are usually an order of magnitude higher than those in surface water. This increase is due to the reduced conditions.

4.5. OCCURRENCE IN AIR

4.5.1. Concentrations in the working environment and in indoor air

Data with regard to the concentrations at the workplace are not available for the Netherlands. Data from abroad are usually dated, the concentrations mentioned varying considerably (also within one branch of industry).
Indoors the concentrations of aerosol components are appr. 30-50% of those in outdoor air (EPA/APCA, 1988) assuming that no (fine) arsenic (dust) sources are present indoors. The contents in household dust may be compared with those in street dust in urban areas (Harrison, 1979). These contents range from 10 $mg.kg^{-1}$ in small towns to appr. 25 $mg.kg^{-1}$ in large ones (Ferguson et al., 1984; 1986). It should be noted, however, that the resuspended arsenic may be considered as belonging to the (extremely) coarse aerosol fraction with extemely short residence times in air.
As a result of the decreased use of arsenic-containing pesticides the As content in tobacco has decreased considerably, mostly to 1 - 10 $mg.kg^{-1}$ (WHO, 1981). Such a level would cause an emission of 15 - 150 kg As per year in Dutch residences. It is estimated that the arsenic in residences as a result of smoking will increase by several tens of $ng.m^{-3}$ at the most (Lebret, 1989).

4.5.2. Concentrations in outdoor air

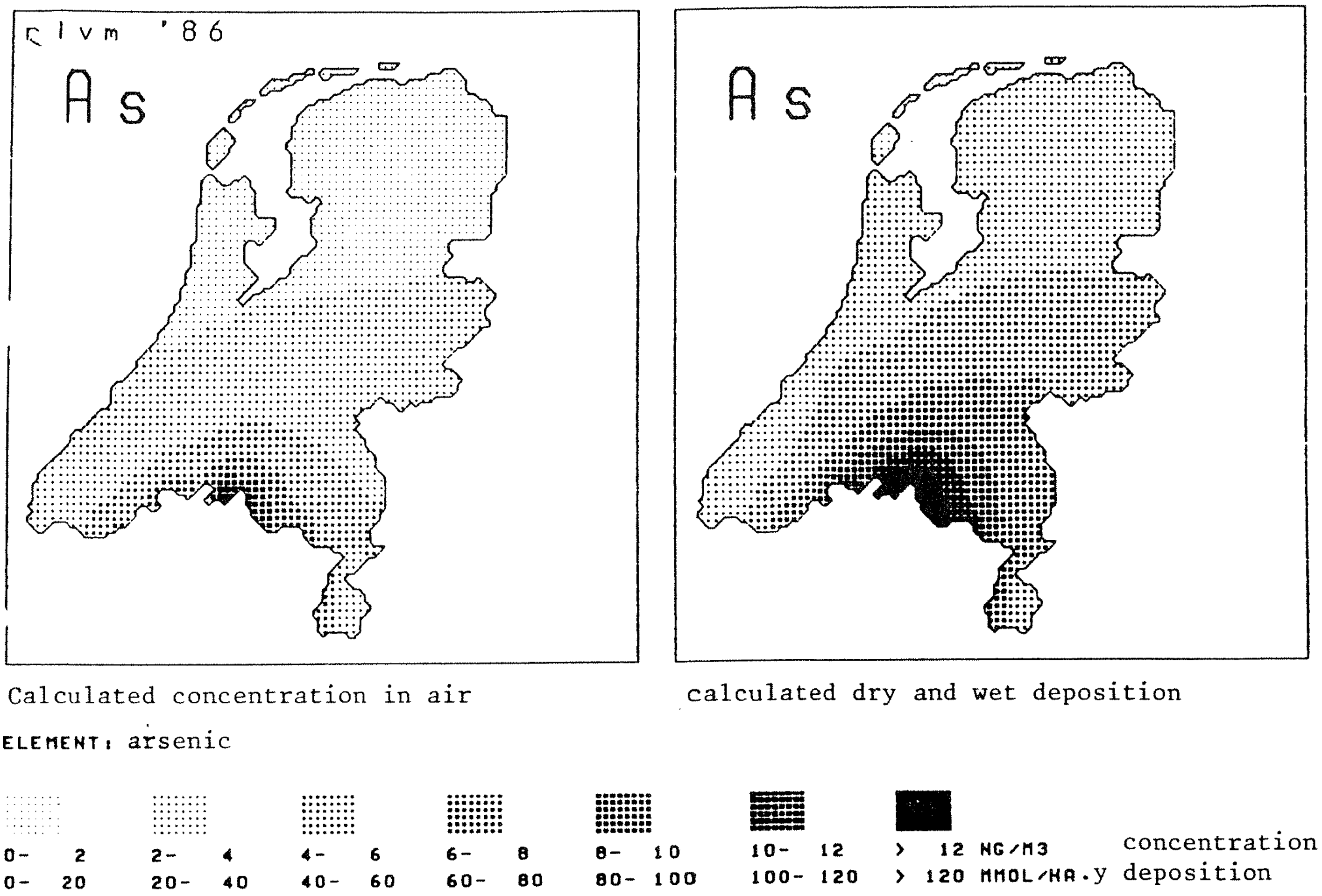
In a review of concentration levels in the atmosphere, measured at various places in the world, Bennett (1981) mentions 0.2 - 40 $ng.m^{-3}$ (average appr. 6 $ng.m^{-3}$) for inhabited areas and 10 - 750 $ng.m^{-3}$ (average appr. 20 $ng.m^{-3}$) for urban c.q. industrial areas. In Belgium and Germany the annual average in large towns amounts to 5 - 20 $ng.m^{-3}$ (IHE, 1988; Bundesamt, 1987). In the vicinity of non-ferro metallurgical installations the annual average in the past amounted to several hundreds of $ng.m^{-3}$; in the second half of the eighties, however, a distinct decrease in the arsenic levels occurred to the current concentrations of several tens of $ng.m^{-3}$ at the most.
Compared with situations abroad the values measured in the Netherlands, at least in urban-industrial areas, are not high: that is appr. 4 - 16 $ng.m^{-3}$

(Thijsse and Huygen, 1985). The national levels in the Netherlands (2 - 7 ng.m^{-3}) do not or hardly differ from those in Germany and Belgium. In table 4.18. an overview is presented of the annual average arsenic concentrations in outdoor air at various places in the Netherlands determined in various measuring series (CCRX, 1988).

<u>Table 4.18</u>. As concentrations (in ng.m^{-3}) in outdoor air in the Netherlands in the period 1980 - 1985

Location	*Average concentration*	*Year*
Arnhem (Callandweg)	*16*	*1980-81*
Nijmegen	*8*	*1980-81*
Arnhem (Stevinweg)	*4*	*1981-82*
Nijmegen (Pelseland)	*12*	*1981-82*
Nijmegen (Waalhaven)	*8*	*1981-82*
Witteveen	*0.3-2.3*	*1982-83*
Rekken	*0.3-3.2*	*1982-83*
Bilthoven	*0.4-3.2*	*1982-83*
Vlaardingen	*0.4-3.0*	*1982-83*
Biest-Houtakker	*1.1-6.9*	*1982-83*
Bodegraven-North	*5.1-7.3*	*1984-85*
Middle-Delfland	*4.9-7.4*	*1984-85*
Zoetermeer	*1.8-2.1*	*1985*
Maasland	*2.9-4.1*	*1985*

By extrapolation of these measuring results by means of meteorological data and dispersal models the spacial annual average concentration pattern over the Netherlands is obtained (figure 4.5.). According to this calculation the large-scale annual average arsenic concentration amounts to appr. 3 ng.m^{-3} (Van Jaarsveld and Onderdelinden, 1986). There is a distinct decrease in levels from south to north. The high concentration levels in North Brabant may be explained by the substantial contribution from source areas abroad, particularly the non-ferro metallurgical industry in the north of Belgium. In general the Dutch arsenic concentrations in air are greatly determined by sources abroad (up to 85%), especially Belgium (55%) and Germany (25%).



Calculated concentration in air

calculated dry and wet deposition

Figure 4.5. *Large-scale annual average As concentrations (in ng.m^{-3}) and deposition (wet and dry) in the Netherlands in the period 1982-1983*

4.5.3. Deposition

Direct measurements of dry deposition are not available. Therefore the dry deposition is estimated from the deposition rate of the As-containing aerosol and the concentration of the particulate pollution. For arsenic the average dry deposition rate is appr. 0.1 cm.s^{-1}. Assuming the above-mentioned large-scale annual average concentration pattern over the Netherlands the total dry arsenic deposition on the Dutch soil can be estimated at 0.7 - 2.2 g.ha^{-1}.$year^{-1}$, that is 3 - 8 tonnes annually for the entire Netherlands (RIVM, 1989). For 1988, based on air concentrations of 3 ng.m^{-3} on average a dry deposition of 4 tonnes could be calculated for the Netherlands.

The wet deposition of heavy metals is directly determined: the annual average As concentration in rainwater varies from appr. 0.75 to 1.50 $\mu g.l^{-1}$ in the period 1978 - 1982. Since 1983 arsenic in precipitation has only been determined in De Bilt. The average concentration is appr. 0.60 $\mu g.l^{-1}$. During rainfall, through wash out the arsenic contents in rainwater in areas with higher arsenic concentrations will be elevated compared to those in areas with lower levels. Therefore, the greatest wet deposition is observed in the south of the Netherlands (North Brabant), where the outdoor air concentrations are highest.

The average wet deposition over the Netherlands in the eighties varies from 17 to 29 tonnes per year. Through the former use of "open" rain collectors these figures might be an overestimation of the wet deposition. In 1988, at three locations in the Netherlands, only the wet deposition was measured, amounting to an average of 13 tonnes of arsenic.

In figure 4.6. the large-scale annual average total (=wet + dry) deposition pattern over the Netherlands is given as well. For 1988 the total arsenic deposition is estimated at 17 tonnes maximally of which 4 tonnes dry and 13 tonnes wet (RIVM, 1989).

4.6. OCCURRENCE IN FOOD AND DRINKING WATER

The occurrence of arsenic in food and drinking water has been discussed extensively in the Draft Assessment Report Arsenic in the Dutch Environment (CCRX, 1988).

Since 1970 systematic studies have been carried out on the arsenic content in meat and offal (Vaessen, 1986; Vos et al., 1987a,b; Vos et al., 1988; Van Delft en Vos, 1988), in which contents of up to 0.6 $mg.kg^{-1}$ fresh product were observed and no distinct decreasing or increasing tendency over time was observed.

Since 1971 the arsenic contents in fish have been frequently determined (Vos et al., 1986; LAC, 1986; Luten, 1978; RWS/DGW, 1987). The highest contents were found in plaice, an average of appr. 20 $mg.kg^{-1}$ fresh product. The contents in freshwater fish are lower than in sea fish. The arsenic contents for mussels and shrimps are between those for sea fish and freshwater fish. In sea fish the arsenic contents are generally considerably higher than in slaughter animals and crops. The contents of inorganic arsenic in plaice, sole, herring, cod and mussels is below 0.1 mg.kg^{-1} (Luten, 1978).

In the Netherlands consumption of seaweed is increasing. The arsenic contents in seaweed are high, an average of appr. 40 mg.kg^{-1} dried product (Luten, 1983).

In 1982 and 1985, 259 and 253 petrol stations, respectively, were studied on the As content in drinking water (Fonds et al., 1987). The contents were below 1 μg.l^{-1} in 93% and 81% of the petrol stations, respectively. The monitoring limit was exceeded twice in 1982 and three times in 1985, however, the contents were never above 10 μg.l^{-1}.

For the determination of the daily intake arsenic contents were determined in 24-hour feeds (table 4.19.). Vaessen and Van Ooik carried out studies on a population group consisting of men and women between the age of 18 and 74 with an average consumption of 2.23 kg per day, whereas in the studies by Van Dokkum et al. it concerned 16-18 year-old boys with an average daily consumption of 3.63 kg.

Table 4.19. Arsenic contents in 24-hour feeds (μg) in a number of countries

Country	*Period*	*Range*	*Av.*	*Reference*
The Netherlands	*1976-1978*	*11-1001*	*<22 **	*Vaessen et al., 1979, 1980*
	1976-1978		*15*	*CCRX, 1988*
	1984-1985	*5- 111*	*15*	*Vaessen and Van Ooik, 1988*
	1984-1986	*20- 88*	*46*	*Van Dokkum et al., 1986*
		*8-459 ***		*CCRX, 1988*
England	*1974-1978*	*67-250*	*89*	*FACC, 1982*
Canada	*1981*	*3-100*	*17*	*Dabeka et al., 1987*
USA	*1981-1982*		*46*	*Gartrell et al., 1986*
Belgium	*1981*	*1-720*	*45*	*Buchet et al., 1983*

** = median*
*** = calculated values*

Exploratory studies on 24-hour feeds indicated that probably 25% of the total arsenic is present in inorganic form (Vaessen and Van Ooik, 1988).

4.7. ARSENIC BALANCE FOR THE NETHERLANDS

The balance shown in figure 4.6. is drawn up based on the dust flows mentioned before, supplemented with estimates. It has been made balanced, the separate balance terms having been adjusted. The balance concerns the Dutch environment, highly schematized, the air over the Netherlands being considered a mixed system and only one water and two soil compartments being distinguished.

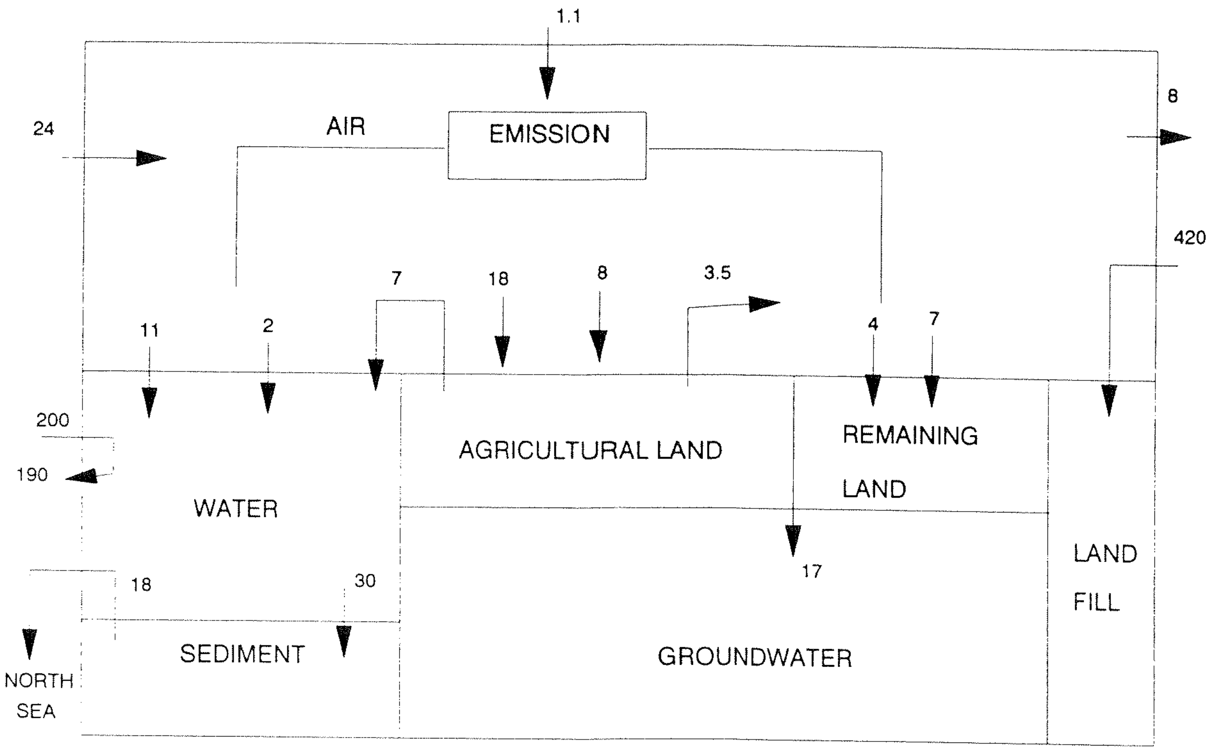


Figure 4.6. Arsenic fluxes in tonnes per year in the Dutch environment (excl. biological cycle; see figure 3.4.)

* Air :It is estimated that in total appr. 17 tonnes per year is deposited on the Netherlands: 15 tonnes to the soil and 2 - 3 tonnes to surface water. From the average As concentration (3 ng.m^{-3}) in the air and the residence time over the Netherlands an export of maximally 8 tonnes per year is calculated. The corresponding import term is 24 tonnes per year at an emission in the Netherlands of 1.1 tonne annually.

* Water :The import via the Rhine and Meuse water is estimated at 200 tonnes per year, based on a flow of 2500 $m^3.s^{-1}$ and an average concentration of 2.4 $\mu g.l^{-1}$. The export is estimated to amount to appr. 190 tonnes per year (2750 m^3 x 2.0 $\mu g.l^{-1}$). The emission amounts to appr. 11 tonnes, the contribution of the deposition being 3 tonnes per year. Besides the surface water is loaded with appr. 7 tonnes per year by washout, based on an As concentration in the washout water of 2.0 $\mu g.l^{-1}$ and 10 cm of washout rainwater per year. Roughly 30 tonnes is deposited of which 18 tonnes is dredged annually and dumped at sea, the remaining part (12 tonnes) accumulates in the sediment.

* Soil :The deposition amounts to 15 tonnes in total, immission being 18 tonnes, washout 7 tonnes and crop extraction 3.5 tonnes. Assuming an arsenic content in the soil of 12.5 $mg.kg^{-1}$ and a Kd-value of 25.10^3 $l.kg^{-1}$ (at a pH value of 7.35 and the presence of 2% Fe in the soil) at a concentration of 2.0 $\mu g.l^{-1}$ and a transport rate through the soil of 25 cm per year the washout to groundwater amounts to appr. 17 tonnes. The accumulation amounts to appr. 10 tonnes per year (corresponding with appr. 1.7 $\mu g.kg^{-1}$ per year.

It should be emphasized that the contribution of anthropogenic sources to the As fluxes through the environment will be relatively small.

4.8. SUMMARY AND CONCLUSIONS

Measuring methods for the determination of arsenic contents in various matrices are sufficiently available, as well as methods for the determination of the chemical quality (speciation of arsenic).

In table 4.20. an overview is given of ranges of average arsenic levels in soil, water, air and food in the Netherlands. Application of arsenic-containing pesticides in the past resulted in arsenic contents in agricultural areas which are slightly elevated in comparison with those in nature reserves.

The concentrations in surface water and sediments have, since 1980, substantially decreased.

Table 4.20. As contents (ranges, total contents) in soil, water, air and food in the Netherlands

Soil	Agricultural lands (top layer 0-20 cm)	Marine clay	3 - 30	mg.kg^{-1}
		River clay - Rhine	7 - 205	mg.kg^{-1}
		- Meuse	4 - 90	mg.kg^{-1}
		Loess	6 - 12	mg.kg^{-1}
		Reclaimed peatland	0.2 - 11	mg.kg^{-1}
		Peaty soil	2 - 21	mg.kg^{-1}
		Sandy soil	2 - 25	mg.kg^{-1}
	Nature reserves (top-layer 0-10 cm)	Sandy soil	1 - 9	mg.kg^{-1}
		Sandy loam/loam	5 - 18	mg.kg^{-1}
		Clay	12 - 21	mg.kg^{-1}
		Peaty clay	11 - 33	mg.kg^{-1}
		Peat	5 - 25	mg.kg^{-1}
Water *	Natural concentration		1 - 2	μg. l^{-1}
	Groundwater	National	1.5 - 10	μg. l^{-1}
		Local (glauconite)	10 - 50	μg. l^{-1}
	Surface water	1986	1 - 3	μg. l^{-1}
		1976	2 - 15	μg. l^{-1}
	Seawater		1.5 - 3	μg. l^{-1}
	Rainwater (av.)		0.6	μg. l^{-1}
Sediment (adjusted contents at 50%<16μm)		fresh	5 - 60	mg.kg^{-1}
		marine	14 - 35	mg.kg^{-1}
		pore water fresh	2 - 45	mg.m^{-3}
Air	Natural concentrations		1 - 2	ng.m^{-3}
	Rural areas		2 - 7	ng.m^{-3}
	Urban-industrial areas		4 - 16	ng.m^{-3}
	Indoor air	no cig. smoke	2 - 8	ng.m^{-3}
		with cig. smoke	10 - 50	ng.m^{-3}
Food and drinking water		daily inorg.As	15 - 58	μg/day
		daily total As	25 - 300	μg/day

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* total contents, dissolved 70-90% In some waters the percentage dissolved As is lower (West Scheldt: 7-10 mg.m^{-3} tot. vs. 2.3-5 mg.m^{-3} dissolved)

5. EFFECTS

This chapter contains a summary of a background report on the possible effects of arsenic exposure on man and the environment. This background report (Hesse et al., 1989), containing all references used, is included as an appendix (Appendix Effects). In this chapter only references are given that are relevant in deriving toxicological limit values.

5.1. HUMAN TOXICITY

Based on experiments with animals fed diets with a low As-content arsenic has been suggested to be an essential element to mammals. However, since human data are lacking and the biological role of arsenic is not known, arsenic is considered as a non-essential element.

On the basis of kinetics and toxicity arsenic compounds can be divided into three groups; water soluble inorganic compounds, insoluble arsenic compounds (including poorly soluble compounds) and organic compounds.

5.1.1. Kinetics and metabolism

- Absorption

Following oral exposure the absorption of soluble inorganic compounds from the gastro-intestinal tract is rapid and nearly complete.

Data on the amounts excreted indicate that appr. 80-100% of these compounds is absorbed in animals and humans. The absorption of trivalent arsenic is greater than that of pentavalent arsenic. Insoluble and pooorly soluble compounds are less readily absorbed. The absorption of organic arsenic as present in seafood (mainly arsenobetaïne) is nearly complete. The uptake of other organic compounds depends amongst other things on the lipophility.

From experimental studies and occupational data it was estimated that the absorption of water soluble inorganic arsenic compounds following inhalation is 30% to 60%. The absorption percentage of insoluble compounds is lower. The average absorption for total (soluble and insoluble) inorganic arsenic is estimated at 20%.

Dermal absorption has been described but quantitative data are lacking.

- Distribution

Once absorbed, arsenic is widely distributed in the body via the blood. In most animal species, including man, the absorbed arsenic is rapidly cleared from the blood to various tissues. Shortly after exposure the highest concentrations are found in liver, kidneys, spleen, skin and lung. The arsenic concentration declines rapidly, with the exception of skin, lungs and hair, which contain relatively large amounts of proteins with SH-groups to which trivalent arsenic binds. The disappearance from blood in rat is much slower resulting from the strong affinity of the heamoglobin of the rat for arsenic. Inorganic arsenic can cross the placenta, both in animals and humans.

- Biotransformation and excretion

Excretion in the urine is the predominant elimination route of arsenic. Within a few days 60-90% of both inorganic and organic arsenic is excreted by the kidneys. A small percentage is eliminated via the bile and by other routes; sweat, desquamation of the skin and hair. The major excretion products are dimethyl arsinic acid and methyl arsonic acid (65% and 20%, respectively, of the total quantity excreted). Small amounts of inorganic arsenic are excreted unchanged. Methylation, representing a route of detoxification, probably mainly takes place in the liver. The (partly enzymatic) detoxification shows saturation at higher exposure levels, resulting in strongly elevated blood levels.
Organic arsenic from seafood is excreted unchanged. Interconversion of trivalent and pentavalent arsenic has been observed in some animal experiments.

5.1.2. Toxicity

Acute and subacute toxicity

- Animal studies

The toxicity of inorganic arsenic compounds varies depending on the chemical form and oxidation state. In general trivalent arsenic is more toxic than pentavalent arsenic. Oral LD50-values have been reported from 10 to 300 mg $As.kg^{-1}$ b.w. for inorganic arsenic. Lower LD50-values are found for readily (water)soluble compounds.

Oral LD50-values for phenoxarsine oxide (PXO) and phenarzaine oxide (PZO) (industrial biocides) were found to be appr. 10 and 24 mg $As.kg^{-1}$ b.w., respectively. A single oral dose of PXO (10 mg $As.kg^{-1}$ b.w.) resulted in hepatotoxic effects in rats. A similar dose of PZO did not cause any toxic effects.

A subacute oral dose (through a gavage) of 7.6 mg $As.kg^{-1}$ b.w. as arsenic trioxide caused dermal effects in rats. No toxic effects were found at a dose of 1.5 mg $As.kg^{-1}$ b.w. In rats, subacutely exposed to drinking water containing 40 mg $As.l^{-1}$ and more, an increase in relative kidney weight was found. In rabbits, exposed to dimethyl methane arsenate in the diet (0.8 mg $As.kg^{-1}$b.w.) for 7-8 weeks, effects in the liver were observed.

The LC50 for arsine in mice by inhalation has been estimated to be 500 $mg.m^{-3}$ after 2.4 min. Exposure to 75 $mg.m^{-3}$ arsine during 30 min. may be lethal for mice. Inhalation of PXO and PZO in concentrations of 0.3-0.6 mg $As.m^{-3}$ (5 h a day) during 30 days did not cause any toxic effects in rats. No mortality was found in rats inhalatorily exposed to aerosols containing an estimated concentration of 840 $mg.m^{-3}$ of dimethyl arsinic acid.

- <u>Human studies</u>

Acute effects in humans are in general the same as in animals. Lethal oral dose for man is 70-180 mg arsenic trioxide (0.8 - 2.3 mg $As.kg^{-1}$ b.w.). Inhalation of 25 $mg.m^{-3}$ arsine gas during 30 min. may be fatal.

Subacute effects include fever, anorexia and insomnia. In a large group of infants, exposed to pentavalent inorganic arsenic via milk powder for two to three weeks, hearing loss, EEG-abnormalities, liver enlargement and eye-abnormalities were observed in addition to some cases of mortality. The total intake per infant was estimated to be 1.3-3.6 mg arsenic per day. More than 400 people were exposed for two to three weeks to soy sauce contaminated with an inorganic arsenic compound (probably calcium arsenate). The average daily intake was 3 mg As. Facial oedema, anorexia, neuritis and effects on the respiratory tract and the skin were observed.

(Sub)chronic toxicity (excluding carcinogenicity)

- Animal studies

In animals chronically exposed to inorganic arsenic effects in the liver, skin, cardiovascular and nervous system were found. Liver cirrhosis and necrosis occurred in rats orally exposed to 0.7-4.7 mg As.kg^{-1} b.w. during 50-250 days. In rats given drinking water containing arsenic trioxide at levels of 0.13 to 62.5 mg As.l^{-1} (0.01-5 mg As. kg^{-1} b.w.) for 7 months a dose-dependent proliferation of the bile duct was found. In another study (performed by the same group), however, in which rats were exposed to arsenous acid in drinking water at concentrations up to 6.1 mg As.kg^{-1} b.w. per day) no toxic effects were found.

In a chronic feeding study changes in electrocardiograms were observed in cats exposed to 1.5 mg As.kg^{-1} b.w. No toxic effects were found in rats daily fed a shrimp diet containing 14 mg As.kg^{-1} (0.7 mg As.kg^{-1} b.w.) as organic arsenic. On the basis of this study the semi-chronic toxicity of organic arsenic from marine organisms seems to be low.

Disturbances in the functional state of the central nervous system were found in rats exposed to aerosols containing arsenic trioxide (3.7 μg As.m^{-3}) during three months. In a group of rats continuosly exposed to arsenic trioxide aerosols containing 60 or 120 μg As.m^{-3} no increased mortality was found after one year of observation.

Embryotoxic and teratogenic effects have been reported in hamsters, rats and mice after exposure to both tri- and pentavalent inorganic arsenic. After a single oral dose of 48 mg.kg^{-1} b.w. of arsenate embryotoxic and teratogenic effects were found in mice. At this dose also maternal toxicity was found. At lower oral doses (up to appr. 25 mg As.kg^{-1} b.w. as arsenite), being toxic to the mothers, no teratogenic effects occurred. Intravenous injections of 3 mg As.kg^{-1} b.w. or more caused teratogenic effects. In a 7-generation rat study on arsanilic acid no teratogenic effects were observed.

In an inhalation study with mice a concentration of 22 mg As.m^{-3} as arsenic trioxide resulted in embryotoxic and teratogenic effects. On the basis of these studies it is concluded that no embryotoxic and teratogenic effects occur without maternal toxicity.

- Human studies

Long-term oral intake of inorganic arsenic may result in among other things effects in skin, liver and cardiovascular and nervous system.

Hyperkeratosis and, to a lesser degree, hyperpigmentation are characteristic skin lesions (mainly on soles and palms), occurring following long-term exposure to inorganic arsenic via drinking water or drugs. These lesions have been reported from various parts of the world where drinking water contains naturally high arsenic levels. For example, from an epidemiological study in Mexico, the calculated relative risk of the development of several different skin disorders varied from 3.6 (ulcerative zones) to 36 (keratosis). The As-content in the drinking water of exposed persons was 0.41 mg $As.l^{-1}$, that of the control population was 0.007 mg $As.l^{-1}$. The arsenic was for 70% in its pentavalent form (Cebrian et al., 1983). Among a group of 262 patients orally treated with large doses arsenite (sometimes more than 10 mg arsenic per day for several years) a positive dose-response relationship was observed between the total intake of arsenic and the incidence of hyperkeratosis. Multiple keratosis may develop into precancerous states.

High prevalences of the Blackfoot disease (BFD), a specific cardiovascular disorder associated with long-term oral arsenic exposure, have been reported in areas with a naturally high As-content (0.01-1.82 mg $As.l^{-1}$) in drinking water. A positive dose-response relationship was found between the total As-intake and the BFD incidence. Other substances present in the water may have played a role in causing BFD. Peripheral vascular diseases (like the Raynaud syndrome and acrocyanosis) have been desribed in populations exposed during appr. 15 years to an average of 0.6 $mg.l^{-1}$ in the drinking water.

Neurological effects have been described in patients exposed to inorganic arsenic.

Electromyographic abnormalities were reported among persons using drinking water containing more than 0.05 mg $As.l^{-1}$ for a long time. Other factors that would predispose people to develop EMG abnormalities were not taken into account.

Other symptoms of arsenic intoxication are disturbances of the haematopoietic system and, possibly, effects on resistance to bacterial infections. Effects on the respiratory tract have been suggested resulting from oral exposure. Among patients treated with arsenite effects on liver have been reported.

On the basis of a number of drinking water studies the Joint Expert Committee on Food Additives (JECFA, 1983) of the World Health Organization (WHO) concluded that after long-term intake of drinking water containing 1 mg As.l^{-1} toxic effects are very likely to occur. Slight effects in groups at risk may not be ruled out after long-term consumption of drinking water containing 0,1 mg As.l^{-1}. On the basis of a daily consumption of 1,5 l this corresponds with a long-term daily intake of 0.15 mg As per person. The risk of effects occurring following this intake is not negligible.

Data on chronic exposure to arsenic by inhalation are mainly available from studies among occupationally exposed groups.

Exposure to relatively high arsenic levels (up to 7000 μg As.m^{-3} , but mostly less than 500 μg As.m^{-3}) resulted in lesions of the membranes of the respiratory tract and perforation of the nasal septum among smelter workers. In arsenite treated patients and in occupationally exposed persons (to e.g. arsenic) effects on the liver (cirrhosis and consequently portal hypertension) have been found. In serum of workers exposed to relatively low arsenic levels (below 13 μg As.m^{-3},as a 6-h average) no significant increase in liver enzymes (indicative for liver damage) was found. Long-term inhalation of dust containing arsenic trioxide resulted in changes in the peripheral blood circulation among smelter workers (Lagerkvist et al., 1986). The same group showed slight neurologic and (not significant) electromyographic abnormalities (Blom et al., 1985). During this study the concentration was appr. 50 μg As.m^{-3}. In previous years the level was considerably higher (up to 500 μg As.m^{-3}). The authors were not able to determine whether the symptoms resulted from present exposure or from exposure in the past. However, the authors concluded that the probability of clinical neuropathy occurring may be considered slight at exposure levels of 50 μg As.m^{-3} or less. With regard to cardiovascular diseases some studies indicated an increased mortality, whereas in other studies no excess in cardiovascular mortality was found. Further local skin irritation

and sensibilization may occur in smelter workers. From the above-mentioned studies it is concluded that at a concentration of appr. 50 $\mu g.m^{-3}$ slight toxic effects may occur in occupationally exposed persons.
Data on teratogenicity of arsenic in humans are too limited to draw conclusions. In women who worked at a copper smelter during pregnancy an increased frequency of spontaneous abortion and deformities was observed. However, other chemical pollutants were present and exposure concentrations were not given.

- <u>Mechanisms of toxicity</u>

Arsenate is analogous to phosphate. Therefore it may replace phosphate in the respiratory chain and so uncouple the oxidative phosphorylation. Furthermore, in the tissues arsenate may be metabolized into arsenite, which is more toxic. The most important mode of action of arsenite is blocking enzymes that have a sulfhydryl group in the active center.
The most toxic form of arsenic is arsine gas (AsH_3). Arsine combines with the haemoglobine of erythrocytes, resulting in lysation of these cells. The degradation products of these cells may block the action of the kidneys.

5.1.3. Genotoxicity

From genotoxicity tests it appeared that inorganic arsenic compounds do not induce gene mutations. But on the other hand in both animal and human cells increased frequencies of SCE's and chromosome aberrations were found in vitro. However, in the only in vivo experiment in mice no induction of chromosome aberrations could be found in bone marrow cells or in spermatogonia. Organic arsenic compounds did not induce gene mutations nor a higher frequency of SCE's in vitro.
In peripheral lymphocytes from people exposed to inorganic arsenic higher frequencies of chromosome aberrations and SCE's were demonstrated. However, because of the fact that these persons were simultaneously exposed to other substances, it can not be ruled out that the observed effects must be ascribed to other factors than arsenic exposure.
A number of studies indicate that the increased frequency of SCE's and chromosome aberrations found in vitro only occur if arsenic is present during the S-phase of the cell-cyclus. Besides arsenic has been shown to be

incorporated into the RNA molecule (and therefore probably also into DNA) instead of posphorus. Arsenic has also been reported to inhibit DNA repair mechanisms. The unusual genotoxic profile (induction of chromosome aberrations in vitro but not of gene mutations) might be due to the strong interaction of arsenic with sulfhydryl groups of enzymes.

5.1.4. Carcinogenicity

- Animal studies

Various arsenic compounds, mainly inorganic, have been tested for carcinogenicity in experimental animal studies. Long-term oral (rats and mice) or inhalatory (rats) exposure did not result in increased tumour incidences. Subchronic intratracheal administration did not result in an increase in the number of tumours in rats. In two studies it appeared that hamsters develop lung adenomas (benign) upon intratracheal administration. In one of these experiments the doses used were found to be toxic to the lung-cells as well.

In a number of studies the development of malignant tumours was reported following treatment with inorganic arsenic. After repeated subcutaneous or intravenous administration in mice lymphocytic leukaemia as well as lymphomas were found. After intratracheal application of arsenic-containing mixtures to rats lung tumours were observed; these mixtures contained also copper sulphate and calcium hydroxide or tinoxide ore.

On the basis of these data it was concluded that there is inadequate evidence for carcinogenicity in animals with respect to exposure routes relevant for humans. The International Agency for Research on Cancer (IARC, 1980, 1987) arrived at the same conclusion.

A mechanistic study indicated that both arsenite and arsenate do not have initiating activity in male rats (in a kidney model), but have tumour promoting activities with respect to kidney tumours in rats initiated by diethyl nitrosamine.

- Human studies

Long-term oral exposure to inorganic arsenic is associated with an increased risk for skin cancer. Furthermore, several studies indicated evidence of an increased incidence of cancer of the respiratory tract in association with exposure to inorganic arsenic by inhalation. No data are available on the carcinogenicity of organic arsenic compounds.

- *Oral exposure*

An increased incidence of skin cancer following oral exposure of inorganic arsenic via drinking water or drugs has been described in a number of studies. Some of these studies will be described below.
In a survey carried out among more than 40,000 inhabitants from an area in Taiwan with high natural concentrations of arsenic in drinking water (10-1820 μg As.l^{-1}; 500 μg As.l^{-1} on average) a positive dose-response relationship was observed between the total intake and the prevalence of skin cancer (Tseng, 1977). Contribution to the development of skin cancer by other compounds present in the water cannot be ruled out. No excess of skin cancer was found in a population of the USA at a concentration in the drinking water up to 2150 μg As.l^{-1} (16.5 μg As.l^{-1} on average) (Morton et al., 1976). However, these results are not necessarily contradictary, since the average arsenic concentration in the last-mentioned study were about 30 times lower. In a study (in Taiwan) increased cancer mortality of bladder, kidney, skin, lung, liver and colon was reported at a total daily intake of 1 mg and more. Results of a patient-control study, performed in Mexico, indicate an elevated risk for cancer of the bladder, lung and liver. The prevelance of skin cancer increased as the total dose ingested increased in a group of 262 patients chronically treated with As-containing drugs. In this study, however, a large number of persons did not respond to the call for a check-up and a reference group was lacking. In another study increased incidence of benign and malignant skin tumours was reported among patients, of which some were exposed to a total dose of 10 g As and more (appr. 170 mg As.kg^{-1} b.w.).
On the basis of the epidemiological data the IARC (1980) concluded that there is sufficient evidence that inorganic arsenic compounds are skin carcinogens in humans after oral exposure. Data suggesting an increased risk for cancer at other sites were considered to be inadequate for

evaluation. The WHO (JECFA, 1983) acknowledges the association between exposure to inorganic arsenic from drinking water and increased skin cancer risk, but assumes that there are no carcinogenic effects without toxic effects.

- *Inhalatory exposure*

Several occupational studies indicated an increased incidence of cancer of the respiratory tract among workers exposed to inorganic arsenic.
Increased cancer risk to the respiratory tract was found among man, working in a pesticide (containing inorganic As) producing unit. A positive dose-response relationship was found between exposure and respiratory cancer. The contribution of arsenic itself is difficult to determine, since in most cases simultaneous exposures occurred, to trivalent and pentavalent arsenic as well as to other compounds.

In one study ceiling exposure seemed to be more important than TWA-exposure in the increased incidence of lung cancer. It was suggested that when exposure is kept below 500 μg As.m^{-3} the risk of lung cancer will be very low. Increased incidences of cancer of the respiratory tract were also reported among workers handling pesticides containing inorganic arsenic.

In most of the epidemiological studies mentioned above simultaneous exposure to other substances occurred. In the "drinking water" studies besides arsenic other substances (including organochlorides and ergot alkaloïds) were identified in the water. Exposure to other substances than arsenic was reported in studies on copper smelter workers (sulphur dioxide) as well as in studies on workers handling pesticides (other pesticides). It can therefore not be ruled out that the observed effects may be ascribed to causes other than exposure to arsenic.
In spite of these facts it is concluded that inorganic arsenic compounds are skin and lung carcinogens in humans. Data suggesting an increased risk to cancer at other sites are considered to be inadequate for evaluation. No data could be found regarding the carcinogenicity of organic arsenic compounds.
The IARC (1980, 1987) considered the evidence, that inorganic arsenic compounds are skin and lung carcinogens in humans, to be "sufficient". With

regard to oral exposure the JECFA concluded that there is no carcinogenic risk without toxic effects. With regard to the lung cancer risk, on the basis of four smelter studies (using a "non-threshold extrapolation method) the EPA calculated "unit risks" in the range 1.25 to 7.6 x 10^{-3} (mean 4.3 x 10^{-3}). A "unit risk" of 3.0 x 10^{-3} was proposed by the WHO as a "conservative" estimate of the lung cancer risk.

In 1945 airborne arsenic levels were reported of up to 40 mg $As.m^{-3}$. Original exposure data (obtained from one of these studies) were calculated to 8-h time-weighted averages (TWA) over 40 years of exposure. If workers exposed for less than one year were excluded, no clear dose-response relationship was observed below a TWA of 90 μg $As.m^{-3}$. Updating these data and extending them with additional data over a 9-year period, it appeared that the new derived mortality data did not result in a dose-related increase of cancer mortality. It is not known to what extent the exposure levels in the follow-up period were comparable to those of the orginal cohort.

Studies concerning the relation between the spraying of pesticides containing inorganic arsenic compounds and lung cancer were contradictory. Several case reports described skin and lung cancers in vineyard workers suffering from chronic arsenic intoxication. Among inhabitants living in an area with a high usage level of pesticides, no increase in these cancers has been found. Also no relationship could be found between exposure to lead arsenate in the past and the occurrence of cancer in vineyard workers. Among copper smelter workers exposed to inorganic arsenic an increased risk of lung cancer has been reported several times. A number of studies indicated that particularly peak concentrations (5000 μg $As.m^{-3}$ and more) are essential in increasing the risk and the exposure period is a less important factor. Also in these studies there was simultaneous exposure and data on smoking habits and age-distribution in the groups were not always taken into account. One of these studies suggested that workers whose exposure was kept below 500 μg $As.m^{-3}$ demonstrated no excess in lung cancer mortality.

On the basis of epidemiological studies the IARC (1980) concluded that there is sufficient evidence that inhalatory exposure of inorganic arsenic may cause lung cancer in humans. Insufficient evidence exists with respect to the induction of cancer in other organs. A number of studies have been evaluated by two expert groups: one of the US Environmental Protection Agency (EPA, 1983), the other of the World Health Organization (WHO, 1987). On the basis of relevant studies the carcinogenic potency, expressed as the "unit risk" (defined as the additional cancer risk due to a lifetime exposure to 1 μg $As.m^{-3}$) of inorganic arsenic was calculated. Based on five different data sets the EPA (1983) estimated unit risks ranging from 1.25 to 7.6×10^{-3}, with a geometric mean of 4.3×10^{-3}. In the Air Quality Guidelines the WHO (1987) arrived at a unit risk of 3.0×10^{-3}, proposed as a "conservative" estimate.

5.2. ECOTOXICITY - AQUATIC ORGANISMS

5.2.1. Biotransformation and bioavailability

The role of algae in arsenic biochemistry is significant. Several studies indicate that arsenate can be taken up by algae because of its similarity to essential phosphate. At higher phosphate concentrations the arsenate uptake decreases. The algae metabolize the arsenate into less toxic compounds. At higher trophic levels arsenic is predominantly present in the organic form. Arsenobetein is the dominant form, particularly in marine species.

The availability of arsenic from sediments is low. The iron/arsenic ratio correlates better with the arsenic levels in the organisms than with the arsenic levels in the sediment. A positive correlation has been found between arsenic levels in tubificides (worms) and the arsenic concentration in pore water from sediments of the Rhine and the Meuse. This correlation was not found between arsenic levels in the worm and the arsenic concentration in the sediments.

5.2.2. Bioaccumulation and biomagnification

Bioaccumulation of arsenic in aquatic organisms is low to moderate. Bioconcentration factors (BCF = concentration in organisms/ concentrations in water) were generally higher in organisms of lower trophic levels than in organisms of higher trophic levels. BCF's (based on wet weight) of phytoplankton, invertebrates and fish were reported of up to 400, 700 and 150, respectively. A field study into the arsenic concentrations in organisms from polluted and "normal" lakes showed that the BCF increases with decreasing water concentrations. There are indications that organic arsenic in higher organisms is taken up via the food. Biomagnification, however, is not likely to occur. For example lower arsenic contents were found in fish than in zooplankton living in the river basins of the Rhine and Meuse.

5.2.3. Toxicity

- Freshwater organisms

Short-term toxicity tests with inorganic arsenic have resulted in L(E)C50-values ranging from 206 μg As.l^{-1} for the crustacean *Bosmina longirostris* to 82,500 μg As.l^{-1} for the fish *Pimephales promelas* . For organic arsenic three LC50-values were obtained from one study, ranging from 417 to 1412 μg As.l^{-1}. The acute toxicity of arsenite to the waterflea *Daphnia magna* decreases significantly in the presence of sediment.

Relevant long-term tests with inorganic arsenic resulted in NOL(E)C-values ranging from 10 to 10,000 μg As.l^{-1}. The highest and lowest NOEC-values were found in the same study under identical test conditions for the algae *Ankistrodesmus falcatus* and *Selenastrum capricornutum* , respectively. The large differences in susceptibility of algae to arsenic have been confirmed in other studies. The arsenate toxicity to (macro)algae is inversely related to the concentration of phosphate present. In the absence of phosphate the arsenate toxicity to algae is comparable to the arsenite toxicity. In one study the chronic toxicity of inorganic and organic arsenic compounds was tested in organisms from various taxonomic groups (molluscs, crustaceans, insects and fish). No effects on survival and reproduction were found at the highest tested concentrations of 1,000 μg As.l^{-1}.

On the basis of the results of both short- and long-term toxicity tests, no difference in the toxicity of trivalent and pentavalent inorganic arsenic can be found. Toxicity data on organic arsenic are too limited to allow comparison between inorganic and organic arsenic. However, the data suggest that organic arsenic compounds (expressed as As) are more toxic in short-term tests than inorganic compounds.

- Marine organisms

Data on the toxicity of arsenic to marine organisms are limited and mainly concern inorganic arsenic compounds. Relevant short-term tests have resulted in L(E)C50-values ranging from 232 µg As.l^{-1} for the crustacean *Cancer magister* to 28,500 µg As.l^{-1} for the fish *Limanda limanda* . In one test the effects of arsenate, arsenite and methane arsonate on the filtering rate of the mussel *Perna perna* was examined. Arsenite appeared to be the most and arsenate the least toxic to this mussel. Effects of arsenic have been reported on the development of sea urchins (appr. at 1 mg As.l^{-1}) and several estuarine organisms (> 1 mg As.l^{-1}).
Only one long-term test resulted in a NOEC-value; in a 14-d test a NOEC-value of 34.6 µg As.l^{-1} was found for the macrophyte *Champia parvula* , based on growth and reproduction. At the highest concentration tested in other long-term experiments (ranging from 9 to 3,600 µg As.l^{-1}) no effects were found in organisms of different taxanomic groups.
Marine algae, like freshwater algae, show wide ranges of sensitivity to arsenic. The toxicity reducing effect of phosphate was also found for marine algae. The species composition of a natural phytoplankton assemblage was altered by adding 5 µg As.l^{-1}, both under laboratory and field conditions.

5.3. ECOTOXICITY - TERRESTRIAL ORGANISMS

5.3.1. Accumulation

With regard to the accumulation in terrestrial organisms the data are limited to one study on various earthworm species. This study indicated that arsenic levels in earthworms from contaminated soils (up to 33 mg

As.kg^{-1} d.m.) are comparable to those in earthworms from uncontaminated soils.

5.3.2. Toxicity

Data on the toxicity to terrestrial organisms mostly concern microbe-mediated processes. In one study the influence of arsenic trioxide (As[III]) and arsenate (As[V]) on a number of micro-organisms and on the activity of microbe-mediated processes (dehydrogenase activity) was investigated in sandy and in alluvial soil. At the lowest concentrations tested, 500 mg As[III].kg^{-1} d.m. and 1,000 mg As[V].kg^{-1} d.m., effects were still observed in both soils (Maliszewska et al., 1985). Near a smelter the abundancy of bacteria, actinomycetes, fungi and nematodes increased with decreasing arsenic concentration in the soil (972-163 mg As.kg^{-1}). The concentration of various other metals in this soil decreased with increasing distance from the smelter as well. The hatching of *Heteroderma* species (round-worms) was inhibited in solutions containing 225 mg As.l^{-1} as arsenite and 300 mg As.l^{-1} as arsenate. The NOEC-values for As[III] and As [V] are below 500 and 1,000 mg As.kg^{-1} d.m., respectively.

5.4. TOXICITY TO AGRICULTURAL CROPS AND LIVESTOCK

5.4.1. Agricultural crops

- Accumulation

In one study the arsenic levels in a number of crops were invetigated, grown in "normal" soils in the Netherlands, containing an average of 10 mg As.mg^{-1} d.m. Levels in potatoes, vegetables and apples varied from 0.001 to 0.014 mg As.kg^{-1} f.w. (fresh weight). Cereals (especially oats) contain relative large amount of arsenic (0.045-0.189 mg As.kg^{-1} f.w.). The arsenic contents of cattle-fodder crops (grass, silage maize and sugar beet) varied from 0.17 to 0.51 mg As.kg^{-1} d.m., respectively.

Potatoes grown on (soil mixed with) municipal waste compost, sewage sludge or dredgings from contaminated river silt contained levels of up to 69 mg

As.kg^{-1} d.m., which was considered not elevated in comparison with normal values of 0.013 mg As.kg^{-1} f.w. In a heavily contaminated mining area with 144-892 mg As.kg^{-1} in the soil, the levels in the crops were 0.01-0.93 mg As.kg^{-1} d.m. A positive relationship was found between arsenic levels in soil and those in the edible parts of beetroots, lettuce, onions and peas. Arsenic levels in potatoes grown on soils containing concentrations of up to 70 mg As.kg^{-1} d.m., were below the proposed maximum permissible level of 0.10 mg As. kg^{-1} f.w.

- Toxicity

In a pot experiment the toxicity of arsenic differed with the availability of arsenic, and so with the solubility of the arsenic compound tested. Adding up to a total of 18 mg As.kg^{-1} as Fe-, Al-, Na- or Ca-arsenate to sandy soil (background concentration is 8 mg As.kg^{-1}) caused no effects on the growth of corn. Adding 100 mg As.kg^{-1} of the Al-, Na- and Ca-compounds caused more than 50% growth inhibition, whereas no effects were found when $Fe(H_2AsO_4)_3$ was applied (Woolson et al., 1971). In another experiment a significant reduction in soybean yields were reported at applied rates of 12 mg As.kg^{-1} (calculated)(background concentration of the soil used was not given). Concentrations of 80 and 320 mg As.kg^{-1} d.m. as lead arsenate and of 122 mg As.kg^{-1} as arsenite added to soil caused significantly reduced yields of various crops.

In a field experiment with the wild plant *Urtica dioica* growth was significantly inhibited upon addition of 30 mg As.kg^{-1} of lead arsenate and sodium cacodylate [$(CH_3)_2AsO(ONa)$]. At added concentrations of 5 mg As.kg^{-1} d.m. from both compounds no effects were observed. In this experiment also no background concentration was given (Otte et al., 1988).

Among plants differences in sensitivity to arsenic have been observed. Observations on one wild plant species provided some evidence that arsenic tolerance may be developed in plants. In these plants the phosphate uptake system was altered in such a way that arsenic can no longer be taken up by this system. Taking into account the arsenic background concentration in the soil, the highest concentration in these experiments at which no effects were observed, was appr. 20 mg As.kg^{-1} d.m.

5.4.2. Livestock

Accumulation from food into organs and tissues was examined in lambs and cows for various arsenic compounds. Arsenic was applied as a water soluble compound, in sewage sludge or in dredgings from harbours. In lambs fed for 3 months a diet containing about 2 mg $As.kg^{-1}$ d.m. the arsenic levels in muscle-, liver- and kidney tissues were elevated with appr. a factor 10 to 13, 24 and 50 μg $As.kg^{-1}$ f.w., respectively. These tissues also contained the highest As-concentrations in cows given 33 mg As from arsenic trioxide daily for three months. The arsenic levels in muscle, liver and kidney tissue of these cows were 30, 100 and 160 μg $As.kg^{-1}$ f.w. compared to 5, 10 and 35 μg $As.kg^{-1}$ f.w. in control animals (daily intake is 3.4 mg As). Several studies indicated that both inorganic and organic arsenic compounds are not (or hardly) excreted into cows milk. A daily intake of 33 mg As as arsenic trioxide for three months did not cause elevated arsenic levels in the milk or blood of cows. A slight elevation of arsenic levels in milk occurred (0.015-0.026 mg $As.kg^{-1}$) after exposure to 1.6-3.2 mg $As.kg^{-1}$ b.w. from arsanilic acid and arsonic acid.

In chronically intoxicated cattle (intake not reported) concentrations of 7.0-70, 5-53 and 0.07-1.5 mg $As.kg^{-1}$ f.w. were measured in liver, kidney and milk, respectively.

- Toxicity

Pesticides containing arsenic were frequently reported to cause acute poisoning in domestic animals. Organic arsenics, used as growth promotant caused acute poisoning in domestic animals. A subacute exposure to 490-2,900 mg $As.kg^{-1}$ as arsenic trioxide caused 50% mortality in a group of more than 6,000 cattle within a week. Toxic effects were similar to those found in experimental animals. Furhtermore, the milk production was drastically reduced.

Intake of 22.8 mg $As.kg^{-1}$ b.w. as arsenite and arsanilic acid for 8 weeks caused no toxic effects in sheep, whereas 45.6 mg $As.kg^{-1}$ b.w. of the same compounds resulted in effects. After a daily intake of 5 mg $As.kg^{-1}$ b.w. during 3 weeks no adverse effects were found in horses and cattle. In chickens toxic effects were observed after exposure to 18 mg $As.kg^{-1}$ b.w. as arsenic pentoxide for 60 days; a dose of 18 mg $As.kg^{-1}$ b.w. did not cause any adverse effects. Data on chronic toxicity are hardly available. Low levels of arsenic seemed to have beneficial effects on the coat of animals. Application of 1,000 mg $As.kg^{-1}$ as arsaniline acid to cattle in the diet caused toxic effects. The growth promotant level recommended is 100 mg $As.kg^{-1}$ diet.

5.5. SUMMARY AND TOXICOLOGICAL LIMIT VALUES

5.5.1. Humans

Arsenic is considered to be a non essential element.

Epidemiological studies have demonstrated positive dose-response relationships between exposure to inorganic arsenic compounds and cancer risk. Long-term intake of inorganic arsenic via drinking water (with naturally high As-levels) or medication has been associated with an increased skin cancer risk. An increased cancer risk of the respiratory tract has been found in several studies among populations occupationally exposed to inorganic arsenic by inhalation. Although in most of these studies simultaneous exposure to other substances occurred (it can therefore not be ruled out that the observed effects were due to factors other than arsenic exposure) it is concluded that inorganic arsenic can cause tumours in humans (similar conclusion was drawn by the IARC a.o.).

From animal experiments it appeared that inorganic arsenic applied via exposure routes relevant for humans is not carcinogenic in animals. A mechanistic study showed that both tri- and pentavalent arsenic have tumor promoting activities on kidney tumours induced by diethyl nitrosamine.

Arsenic has an unusual genotoxicity profile; in *in vitro* tests a clear induction of chromosome aberrations is observed, but not of gene mutations. There are indications that other mechanisms than direct DNA damage are involved.

From metabolism studies it appeared that in both animals and humans inorganic arsenic is methylated to organic arsenic compounds; this process probably mainly takes place in the liver. At higher exposure levels this detoxification process becomes saturated resulting in higher blood arsenic

levels. Epidemiological studies demonstrated that an increased lung cancer risk correlated stronger with peak exposure than with cumulative exposure.
On the basis of all results (epidemiological studies, animal experimental studies, genotoxicity tests, metabolism studies) it is concluded that, with regard to the carcinogenic effect of arsenic, there is insufficient evidence to use a "non-threshold" extrapolation method for risk assessment. It should be noted that the EPA, in contrast with the point of view that is formulated in this document, did apply a "non-threshold" extrapolation for cancer risk assessment in recent reports for both oral (EPA, 1987) and inhalatory (EPA, 1983) exposure. However, the EPA noticed that a linear extrapolation may overestimate the risks from low level arsenic exposure. For oral exposure the WHO assumed the existence of a threshold (JECFA, 1983), but not for inhalatory exposure (WHO, 1987). As a comparison the values that were derived by these agencies using a "non-threshold" extrapolation are also given.

In animal experimental studies no embryotoxic or teratogenic effects are reported without the occurrence of maternal toxicity. One study on the offspring of women working at a copper smelter indicates an increased frequency of spontaneous abortion and malformations in the offspring. In smelters exposure to a mixture of chemicals occurs, no conclusions can, therefore, be drawn from this study.

Since appropriate long-term animal experimental studies are lacking for both oral and inhalatory exposure, the formulation of a toxicological limit value for arsenic is based on epidemiological data.

- <u>Oral exposure</u>

To assess a toxicological limit value for oral exposure the epidemiological studies, in which exposure took place via drinking water, are considered to be most relevant. On the basis of the effects observed in these studies it was, however, not possible to establish a dose without effect as a basis for a recommended limit value.

On the basis of these drinking water studies (a.o. by Tseng, 1977 and Cebrian et al., 1983) the WHO (JECFA, 1983) also concluded that a threshold value exists for the carcinogenic effect of arsenic. It is assumed that carcinogenic effects only occur at levels at which toxic effects are observed as well. On the basis of the studies mentioned the WHO derived

that intake of drinking water containing 1 mg As.l^{-1} toxic effects are likely to occur. After long-term consumption of water containing 0,1 mg As.l^{-1} there is a risk of effects occurring in groups at risk. Assuming a daily consumption of 2 l of water and a mean body weight of 70 kg* this corresponds with a daily intake of 2.0 μg As.kg^{-1} b.w. (JECFA, 1983).
In the present document the WHO approach is adopted in principle. However, it should be emphasized that an intake of 2.0 μg As.kg^{-1} b.w. per day should not be considered as an "acceptable daily intake" (as defined by the WHO) because effects can not be ruled out after a long-term daily intake of this amount of arsenic.
The WHO did not establish a limit value for organic arsenic compounds. Assuming that these compounds are at least a factor 10 less toxic than the inorganic ones, it is concluded that effects are unlikely to occur after long-term daily intake of 20 μg As.kg^{-1} b.w.

Using a "non-threshold" extrapolation and the data from the study by Tseng (1977) the EPA calculated the risk for skin cancer. The "unit risk" [the lifetime risk due to 1 μg As.kg^{-1} b.w. of arsenic intake from water] was estimated to be appr. 1.5 x 10^{-3} (1 and 2 x 10^{-3} for male and female, respectively) (EPA, 1987). Assuming an acceptable risk of one extra case of skin cancer per million persons exposed for a lifetime, this risk corresponds with a daily intake of 6.7 x 10^{-4} μg As.kg^{-1} b.w.

- <u>Inhalatory exposure</u>

On the basis of occupational studies it was not possible to establish a dose-response relationship for the carcinogenic effect of inorganic arsenic. In a number of studies "threshold values" of 90 or 500 μg As.m^{-3} are described. Exposure to peak concentrations may also be significant in the development of lung cancer.

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* The RIVM normally assumes a daily consumption of 1,5 l water and a mean body weight of 60 kg; corresponding to 2,5 μg As.kg^{-1} b.w.

Toxic effects described among occupationally exposed persons include those effects on peripheral blood vessels and the nervous system; slight toxic effects have been reported at appr. 50 μg As.m^{-3} (Lagerkvist et al., 1986; Blom et al., 1985). In these studies, however, it was not made clear whether these effects had to be ascribed to present or past exposure (up to 500 μg As.m^{-3}). It is assumed that at 5 μg As.m^{-3} these effects will not occur in the working environment. Applying an extrapolation factor of 10 (according to the Health Council, 1985) a concentration of 0.5 μg As.m^{-3} is proposed as a toxicological limit value for the general population.
In the Air Quality Guidelines (WHO, 1987) a "unit risk" of 3.0×10^{-3} is proposed as a "conservative" estimate of the lung cancer risk by inhalatory exposure. Starting from an acceptable risk of one extra case of lung cancer per one million persons exposed for lifetime, this risk corresponds to a concentration of 3.3×10^{-4} μg As.m^{-3} (0.3 ng As.m^{-3}).

5.5.2. Aquatic environment

At present no generally accepted methods are available for the extrapolation of the results of laboratory single species toxicity studies to natural ecosystems. Therefore, different theoretical methods (Slooff et al., 1986; Kooijman, 1987; Van Straalen, 1987) are used provisionally to derive toxicological limit values for the aquatic environment. The applied extrapolation methods are extensively described in a report of an advisory board of the Health Council on the ecotoxicological risk evaluation of chemical compounds (Gezondheidsraad, 1988). With rgard to the extrapolation methods the procedure proposed by this advisory board is followed in principle.
In the method according to Slooff et al. (1986), calculated concentrations are based on the lowest toxicity value available that is considered reliable with respect to the test performance and the results of other tests. In the method of Slooff et al. an L(E)C50 is used to calculate an NOEC for single species ($NOEC_{ss}$) as well as an NOEC for ecosystems ($NOEC_{eco}$). Using an NOEC, only an $NOEC_{eco}$ can be calculated. The values calculated (which need to be divided by an uncertainty factor depending on the formula used) are considered to be "safe" concentrations for a long-term exposure.

In the method according to Kooijman (1987) all relevant L(E)C50-values derived from long-term experiments are used. From these data a HCS ("hazardous concentration for sensitive species") is calculated; the probability of the long-term L(E)C50 of the most susceptible species being below the HCS, being equal to an arbitrarily chosen value (for example 10%). Since chronic toxicity data are not available for arsenic, short-term toxicity data have been applied.

The method according to van Straalen (1987) is similar to that according to Kooijman and uses all relevant NOEC-values. The calculated HC5 ("hazardous concentration for 5% of the species") is that concentration at which adverse non-lethal effects will probably occur in up to 5% (chosen arbitrarily in this evaluation) of the species in an ecosystem exposed. This concentration is considered to be a toxicological limit value.

The results of these extrapolation methods are presented in table 5.1. for both freshwater and seawater. The L(E)C50-values and NOEC-values used in the extrapolation methods according to Kooijman (1987) and Van Straalen (1987) are printed **bold** in the tables 2.2, 2.3, 2.4 and 2.5 of the Appendix "Effects". In the event of two or more NOEC-values for one single species being available, only one value has been used to make the results independent of the distribution of the susceptibilities within a species. A reliable and representative value for a species was chosen on the basis of test performance (reliability, test medium, exposure time, effect parameters) and test results. The values used in the method according to Slooff et al. (1986) are underlined in these tables. Data on short- and long-term toxicity of organic arsenic compounds are far too limited to be used in assessing limit values for freshwater and seawater.

The distribution of long-term NOEC-values for freshwater organisms is shown in figure 5.1; the only NOEC-value for seawater organisms has been marked with a cross.

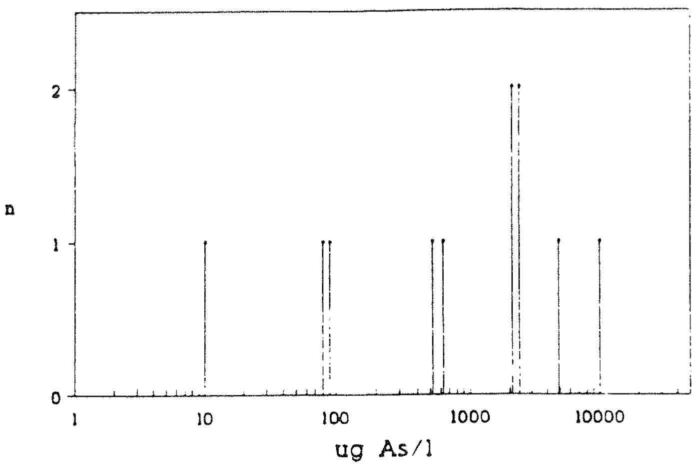

Figure 5.1. *Distribution of NOEC-values of inorganic arsenic in freshwater*

Tabel 5.1. *Calculated concentrations (in µg As.l^{-1}) of inorganic arsenic in freshwater and seawater based on the extrapolation methods according to Slooff et al. (1986), Van Straalen (1987) en Kooijman (1987)*

	Freshwater	*Seawater*
Lowest relevant L(E)C50 *(short-term tests)*	*874*	*232*
Slooff et al.: $NOEC_{ss}$: UF	*32.7 : 25.6 =* ***1.3***	*9.3 : 25.6 =* ***0.36***
$NOEC_{eco}$: UF	*68.0 : 85.7 =* ***0.8***	*23,3 : 85,7 =* ***0.27***
L(E)C50-values *(short-term tests)*	+	+
Kooijman (1) HCS	*0.5*	*0.0095*
Lowest relevant NOEC *(long-term tests)*	*10*	*34.6*
Slooff et al.: $NOEC_{eco}$: UF	*30.2 : 33.5 =* ***0.9***	*86.7 : 33.5 =* ***2.6***
NOEC-values *(long-term tests)*	+	-
Van Straalen (2) : HC5	***6***	-

+/- : sufficient and insufficient data, respectively
UF : Uncertainty factor

[1] Dm from table 1 in Kooijman (1987) at d = 0.1; theorethical number of species in the ecosystem ("n") is 1000.

[2] Dm from table 1 in Kooijman (1987) at d = 0.05.

Freshwater

Based on the data available it is concluded that there is no difference in toxicity between tri- and pentavalent inorganic arsenic to freshwater organisms. Therefore, one toxicological limit value for arsenic is proposed, which applies to the sum of tri- and pentavalent inorganic arsenic.

Because sufficient long-term NOEC-values for arsenic are available the toxicological limit value will be based on these data. Applying the method of Van Straalen (1987) to the available NOEC-values a toxicological limit value of 6 $\mu g\ As.l^{-1}$ is derived for freshwater. Using the method according to Slooff et al. (1986) an $NOEC_{eco}$ of 30 $\mu g\ As.l^{-1}$ is calculated on the basis of the lowest NOEC-value of 10 $\mu g\ As.l^{-1}$. If this value is divided by the UF of 33.5 a "safe" concentration of 0.9 $\mu g\ As.l^{-1}$ is obtained for freshwater.

The difference between the values calculated using the methods according to van Straalen and Slooff et al. is less than a factor 10. Therefore, in accordance with the proposal of the Health Council (1988), the value calculated with the method according to van Straalen (6 $\mu g.l^{-1}$) is recommended as a toxicological limit value. This value is based on total arsenic concentrations under laboratory conditions. Since these conditions generally differ from those in surface waters because of the difference in the amount of particulate matter content, the calculated concentration of 6 $\mu g.l^{-1}$ is considered to be dissolved arsenic (particulate size < 0.45 μm).

Based on L(E)C50-values from short-term toxicity tests an HCS of 0.5 $\mu g\ As.l^{-1}$ has been calculated using the method according to Kooijman (1987). Using the method according to Slooff et al. (1986) on the basis of the lowest L(E)C50-value an $NOEC_{ss}$ and $NOEC_{eco}$ of 32.7 and 68.0 $\mu g\ As.l^{-1}$, respectively, are calculated. Dividing these values by the uncertainty factor results in concentrations of 1.3 and 0.8 $\mu g\ As.l^{-1}$, as dissolved arsenic, considered safe for individual species and ecosystems, respectively.

Seawater

Data on the toxicity of inorganic arsenic to marine organisms are limited, especially with regard to chronic toxicity. Only one NOEC-value from long-term tests being available, the method according to van Straalen can not be applied for risk assessment. Using the method according to Slooff et al., based on the lowest L(E)C50-value from short-term tests, a $NOEC_{ss}$ and $NOEC_{eco}$ of 9.3 and 23.3 μg $As.l^{-1}$, respectively, have been calculated. Dividing these values by the uncertainty factor results in the "safe" concentrations of 0.36 and 0.27 μg $As.l^{-1}$ for single species and ecosystems, respectively. Using the method according to Kooijman (1987) on the available L(E)C50-values results in an HCS of 0.0095 μg $As.l^{-1}$. On the basis of the lowest NOEC-value a $NOEC_{eco}$ of 86.7 μg $As.l^{-1}$ has been calculated, using the method according to Slooff et al. The "safe" value for marine organisms is found by dividing this value by the UF; this results in a concentration of 2.6 μg $As.l^{-1}$.

It is not possible to compare the data on marine organisms with those on freshwater organisms, because the data set on seawater are too limited. However, there does not seem to be a consistent difference: the lowest relevant L(E)C50-value for freshwater organisms is higher (factor 3) compared to that for seawater organisms, whereas the lowest NOEC-value for freshwater is lower (factor 3) than for seawater. Therefore, the toxicological limit value proposed for freshwater is provisionally recommended for seawater as well: 6 μg $As.l^{-1}$ "dissolved" inorganic arsenic.

5.5.3. Terrestrial environment

Data on the accumulation in, and toxicity to terrestrial organisms are too limited to allow assessment of a toxicological limit value.

With regard to the toxicity of arsenic to plants two experiments, in which arsenic was added to the soil as water soluble compounds, are considered to be usable. In the first experiment an added concentration of 30 mg $As.kg^{-1}$ d.m. as lead arsenate or sodium cacodylate [$(CH_3)_2AsO(ONa)$] caused a significant growth inhibition in one wild plant species (Otte et al., 1988). In the second study no effects were found at appr. 20 mg $As.kg^{-1}$

d.m., consisting of a background concentration of 8 mg As.kg^{-1} d.m. and an added concentration of 10 mg As.kg^{-1} d.m. from Fe-, Al-, Na-, and Ca-arsenate. At an added concentration of 100 mg As.kg^{-1} d.m. from Al-, Na-, and Ca-arsenate more than 50% growth inhibition was found. When arsenic was added as Fe-arsenate, at an added concentration of 100 mg As.kg^{-1} d.m. no or only slight effects on growth were observed (Woolson et al., 1971). These effect concentrations are significantly lower than the lowest concentrations tested with terrestrial organisms.

Only one study was available on the toxicity of arsenic to terrestrial organisms. This study was limited to micro-organisms. At the lowest concentration tested (500 mg As.kg^{-1} d.m. from arsenic trioxide and 1000 mg As.kg^{-1} d.m. from arsenate), effects were still observed on the number of micro-organisms and on the activity of microbe-mediated processes in both sandy and alluvial soils (Maliszewski et al., 1985).

On the basis of these data higher plants seem to be the most sensitive to arsenic. The highest concentration without effects appears to be 20 mg As.kg^{-1}d.m. (including the background concentration), the lowest concentration at which effects are observed being 30 mg As.kg^{-1} d.m. (background concentration excluded).

Accumulation of arsenic in agricultural crops is relatively low and the toxicity of arsenic to livestock is low. It is concluded, therefore, that exposure to a concentration which causes no effect in higher plants will not cause any adverse effects in humans and animals.

6. EMISSION REDUCTION MEASURES

Developments whether or not policy oriented have resulted in a considerable decrease in arsenic emissions to soil, water and air the past ten years. In the discussion of emission reduction measures the most important sources identified in chapter 2 will be emphasized. It involves the use of coal and waste incineration (air), emissions resulting from phosphate fertilizer production (water) and manure (soil). Furthermore, attention is given to the contribution by deposition and waste flows. Successively, the autonomous developments will be discussed as well as the supplementary emission reduction measures, and where possible the costs involved. Considering the nature of the data the business economic consequences of measures possibly to be taken will not be discussed.

6.1. AUTONOMOUS DEVELOPMENTS

6.1.1. Emissions to air

- Energy generation with coal

In future the amount of coal used to this end will increase (see table 6.1.). This applies especially to the generation of electricity. The amount of coal to be used depends on the energy variant; in the gas variant (CPB, 1985) the coal consumption in 2010 will be 24 Mtonnes. Because smoke gas desulphurization is compulsory, the As emission will increase less strongly. The emission per unit combusted coal will decrease by appr. 60% (deduced from Meij, 1988). At an increase of the coal consumption to 24 Mtonnes in 2010, which means a fourfold increase of that in 1987, the arsenic emisson will increase from 0.4 to 0.6 tonnes. It may, however, not be ruled out that in future large coal-dust boilers will be substitued by coalgas fired ones. The combustion gases from these installations will probably contain less dust and consequently less arsenic than those from the ones currently used. In view of the uncertainty with regard to the application of coal gasification this has not been taken into consideration in this document.

- Waste incineration

The amount of waste to be incinerated is expected to increase in future. It is estimated that the amount of urban waste to be incinerated in the year 2000 will be appr. 120% higher than that in 1986 (Nagelhout, 1989). The amount of construction, demolition and remaining waste will also increase. It is to be expected that in future all sewage purification sludge will be incinerated, because its application will then be prohibited.

As with combustion of coal, smoke gases will relatively be more purified than they are at present. This will be realized by the application of a smoke gas purification system installed after the electrofilter. The amount of dust per tonne incinerated waste will decrease by appr. 90%. Since the arsenic emitted is practically completely bound to the fly ash this also applies to arsenic. In view of the expected increasing amount to be incinerated and the more effective purification of the smoke gases the arsenic emission to air will decrease from 0.3 tonne in 1986 to 0.1 tonne in 2010 (see table 6.1.).

Table 6.1. Overview of future arsenic emissions to air at autonomous developments (tonnes/year)

Source	Fuel consumption (Mtonnes)			Emission factor (g/tonnes)			Emission (tonne)		
	1987	2000	2010	1987	2000	2010	1987	2000	2010
COAL CONSUMPTION									
Power plants:									
- coal variant	6.0	19.4	25.7	0.066	0.027	0.027	0.40	0.52	0.68
- gas variant	6.0	10.7	12.0	0.066	0.027	0.027		0.28	0.32
- nuclear variant	6.0	13.2	8.0	0.066	0.027	0.027		0.35	0.21
Industry:									
- coke factories	0.24	1.73	1.92	0.000	0.000	0.028	0.00	0.00	0.00
- remaining industry	0.24	8.58	10.35	0.066	0.027	0.027	0.02	0.22	0.27
FUEL OIL CONSUMPTION:									
- Power stations	0.28	0	0	0.100	0.100	0.100	0.03	0	0
- Remaining industry	0.34	0	0	0.100	0.100	0.100	0.03	0	0
- Refineries	1.13	1.46	1.46	0.100	0.100	0.100	0.11	0.15	0.15
SUBTOTAL (gas variant)	8.2	22.5	25.7				0.59	1.52	1.63
WASTE INCINERATION:									
- Urban waste	2.34	5.1	8.9 *	0.10	0.04	0.04	0.23	0.1	0.1
- Chemical waste	0.23	0.2	0.2	0.10	0.04	0.04	0.02	pm	pm
- Remaining waste	0.10	0.81	2.34 *	0.10	0.04	0.04	0.01	pm	pm
- Constr./demolit. waste	0.10	0.36	0.55	0.10	0.04	0.04	0.01	pm	pm
- Sewage plants sludge	0.1	6.2	6.2	0.10	0.04	0.04	0.01	pm	pm
SUBTOTAL:	2.9	12.7	18.2				0.28	0.1	0.1
REMAINING EMISSIONS:									
Pigiron and steel industry							0.04	0.04	0.04
Non-ferro metal industry							0.06	0	0
Remaining industry							0.038	0.04	0.04
Fireplaces/multiburners/smoking							0.03-0.08	0.04	0.05
TOTAL:							1.04-1.09	1.74	1.86

* These data have been deduced from those for the year 2000 (Afval 2000, RIVM/LAE) by means of extrapolation of the growth curves for the various waste flows drawn up for the year 2000, to the year 2010

6.1.2. Emissions to water

- Fertilizer industry

It will be difficult to estimate the extent of the future arsenic emissions by the fertilizer industry. The current productive capacity will probably be maintained or decrease slightly. There is a possibility, however, of the emission being reduced by the application of a different production process for the preparation of phosphoric acid. This process is being developed to decrease the cadmium emission. The exact consequences will not yet be clear, however. It is assumed that the emission will remain constant.

- Deposition

The extent of future arsenic emissions to surface water as a result of deposition is dependent on the environmental policies in the countries surrounding the Netherlands in connection with the (economic) development in those sectors being mainly responsible for the arsenic emissions to air (energy generation with coal, metallurgical industry and waste incineration). The international developments are not known. The deposition is assumed to remain constant.

- Dephosphatization of waste water

At present dephosphatization of waste water only takes place at a small scale. In future it will increase considerably. In 1995 the phosphate load to surface water in the Netherlands will have to be reduced by 50%. To this end the number of dephosphatizing purification installations need to be increased. Studies into the side-effects of dephosphatization on micropollutions is currently being carried out. So far it may be concluded that the mercury and zinc emissions are reduced by dephosphatization. The effect of dephosphatization on the emission of arsenic compounds is not yet clear. To be able to pronounce upon this further research needs to be carried out. If during dephosphatization conditions can be created where arsenic can adsorb on sludge, some effects are to be expected on the arsenic emissions. In this document it is assumed that the efficiency of purification will remain the same.

- Remaining industry

Changes in production processes will cause the emission from the remaining industry to decrease to appr. 0.7 tonne annually in 2010.

- Remaining sources

It is assumed that in future more households and small companies will be connected to sewage plants (RIVM, 1988) an increase from 85% to appr. 95%

In table 6.2. the above-mentioned has been summarized.

Table 6.2. Emission of arsenic to surface water in 1985 and 2010 at autonomous developments (tonnes/year)

Source	*Emission*	
	1985	*2010*
Industry:		
- Fertilizer industry	*5 - 10*	*5 - 10*
- Remaining industry	*0.7 - 1.1*	*0.7*
SUBTOTAL (rounded)	*6 - 11*	*6 - 11*
Sewage plants:		
- Public installations	*0.57*	*0.64*
- Private installations	*0.22*	*0.25*
- Sewage water run over	*0.1*	*0.1*
SUBTOTAL (rounded)	*0.9*	*1*
Remaining sources:		
- Domestic waste water	*0.2*	*0.05*
- Leaching out impregnated wood	*1*	*1*
- Landfills	*<0.01*	*<0.01*
SUBTOTAL	*1.2*	*1.1*
DEPOSITION (wet + dry):	*2 - 3*	*2 - 3*
TOTAL (rounded)	*10 - 16*	*10 - 16*

6.1.3. Emissions to soil

- Diffuse loads

It is not clear how diffuse emissions to soil will develop in future. In 1987 the flow in the form of deposition was an important factor. However, since appr. 5% of the deposition results from Dutch emissions, future deposition will mainly depend om international developments with regard to arsenic emissions to air. At present it is not yet clear in what way this policy will develop.

The amount of arsenic which is applied to the soil in the form of manure will in future probably decrease as a result of the manure legislation. Whether this decrease will be effected through export of manure or through increasing import of raw materials for concentrate is not clear. A decrease in the deposition as well as the application of low As phosphate fertilizer may also lead to a lower As content in manure (see 6.1.2.). Since the current amount of arsenic in manure is not known, it is not well possible to make an exact estimation of its future extent. It is estimated at 8 - 10 tonnes in 2010.

In the developments mentioned in table 6.3. it is assumed that in future the purification sludge will be incinerated first, as a result the emissions to soil will be nil.

- Waste

The total waste flow will increase in the period 1985 - 2010 from appr. 27 Mtonnes to appr. 45 Mtonnes; the waste flow to the soil, however, will decrease from appr. 11 to appr 8 Mtonnes. This is caused by the increasing amount of waste that is incinerated and by the increased recycling of various waste flows. The amount of waste to be incinerated will in this period increase from 2.6 million tonnes in 1985 to appr. 5.1 million tonnes in 2010.

A future decreasing amount of PVC in domestic waste will result in a decrease of arsenic as well. To what extent is not known. In future the greatest arsenic flow to the soil will also originate from industry. The metallurgical industry remains an important factor, contributing considerably to the arsenic flow in the form of jarosite. Besides arsenic, jarosite contains heavy metals and has for years been deposited as chemical waste, for which no appropriate processing methods have been developed as yet. Research into this subject is currently being carried out, mainly aimed at recovering sulphuric acid and ammonia from ammonium jarosite and the production of iron-containing raw materials in the form of iron(III)-oxide. Arsenic remains in the mother liquor as a contaminant.

Table 6.3. Current and future diffuse arsenic loads of the soil (tonnes; autonomous developments)

Source	*Amount* *1985*	 *2010*	*Reference*
To agricultural soil:			
Purification sludge-comm.	*0.55*	*0*	*CBS, 1989c; RIVM, 1988*
Purification sludge-private	*0.14*	*0*	*Martens, 1989; RIVM, 1988*
Compost-comm.	*0.56*	*0*	*CBS, 1989c; RIVM; 1988*
Compost-private	*0.044*	*0*	*Martens, 1989; RIVM; 1988*
Fertilizer	*2.3-4.4*	*2.3-4.4*	*CBS, 1989b*
*Manure **	*13-15*	*13-15*	*CBS, 1989b*
Deposition	*8*	*8*	
SUBTOTAL (rounded)	*25-30*	*23-39*	
To remaining soil:			
Purification sludge-private	*0.1*	*0*	*Martens, 1989*
Domestic waste water	*0.05*	*0.05*	*CBS, 1989b; Feenstra and Van der Most, 1985*
Deposition	*7*	*7*	
Pellets of shot	*0.84-3.4*	*0.84-3.4*	*CCRX, 1989*
SUBTOTAL (rounded)	*8-10*	*8-10*	
Leaching out:			
Materials/waste products	*0.005*	*0.005*	*RIVM estimate, 1989*
Landfills	*0.013*	*0*	*Nagelhout et al., 1989*
TOTAL (rounded)	*33-40*	*31-49*	

** The amount of arsenic ending up on the soil via manure has been calculated from the amount of arsenic in the roughage and concentrate being fed to the Dutch livestock annually, allowing for a slight accumulation in the animals*

The contribution from waste flows originating from coal will in future increase considerably because of the greater consumption of coal for the purpose of energy generation. Furthermore, the flow of waste incineration slag will increase as a result of larger-scale future domestic waste incineration.

In table 6.4. the future developments are indicated of the amount of arsenic ending up on the soil in the form of waste if no additional measures are taken to reduce these flows.

Table 6.4. Future arsenic emissions to soil in the form of waste (autonomous developments)

Waste flow	Total waste flow (Mtonnes)		Total waste flow to soil (Mtonnes)		Total arsenic load of soil (Mtonnes)		References
	1985	2010	1985	2010	1985	2010	
Shredder waste	0.07	0.65	0.03	0	0.6	0	RIVM, 1988; Meijer, 1989
Urban waste	7.1	10	4.95	0	29	0	Nagelhout et al., 1989
Chemical waste	1.0	1.0	0.1	0.1	3.2	3.2	
Construction/demolit. waste	7.7	9	4.4	0.54*	2.2	0.27	
Remaining waste	1.4	1.9	1.1	0.84*	4.8	3.7	
Industrial waste	15	22.5	4	6	261-318	390-475	RIVM, 1988
Dredgings	1.4	1.4	1.4	1.4	18	18	RIVM, 1988
Drinking water sludge	0.10	0.14	0.1	0.14	4.6	6.6	RIVM, 1988, Wieringa and Laan, 1989
Purification sludge	4.7	7.1	4.6	0	3.2	0	RIVM, 1988
Fly ash:							
-coal combustion	0.57	1.14	0.06	0.24	3.2	6.4	RIVM, 1988; Meij, 1988
-waste incineration	0.07	0.18	0.04	0.1	1.3	3.2	RIVM, 1988; Nagelhout, 1989
-chem. waste incineration	0.01	0.01	0.01	0.017	0.2	0.33	RIVM, 1988
-sewage plant sludge incin.	0	0.01	0.003	0.006	0.01	0.18	
Slag:							
-coal combustion	0.06	0.12	0	0	0	0	RIVM, 1988; Meij, 1988
-waste incineration	0.66	2.6	0.18	0.45	5.9	14.6	RIVM, 1988; Nagelhout, 1989
-chem. waste incineration	0.01	0.01	0.01	0.017	0.52	0.9	RIVM, 1988
-sewage plant sludge incin.	0.001	0.04	0.001	0.02	0.02	0.74	
TOTAL:	40	58	21	10	337-395	448-533	

In determining these flows the intermediate gas scenaria was used

* Deduced from data for the year 2000 by means of extrapolation to 2010

6.2. POSSIBILITIES TO FURTHER EMISSION REDUCTION

6.2.1. Emission to air

With respect to the autonomous developments few additional measures are to be taken resulting in a considerable emission reduction to air.

Combustion of coal

Import of coal with a lower arsenic content results in a decrease of arsenic emissions to air. In calculating the emission for the various variants an average As content of 3.65 $mg.kg^{-1}$ coal was used. The country of origin is important with respect to the As content of coal. For instance coal from Australia has a content of appr. 2.4 $mg.kg^{-1}$ and coal from the US appr. 10.9 $mg.kg^{-1}$ (CBS, 1989a). The use of coal with a content of 2.4 $mg.kg^{-1}$ results in a decrease of the emission to air from 0.35 to 0.21 tonnes per year in 2010.

Combustion of fuel oil

Heavy fuel oil-fired installations may be equipped with an installation for the reduction of dust emission. The efficiency of such an installation is appr. 50% (Ros and Slooff, 1987), resulting in a decrease of the emission in 2010 from appr. 0.15 to 0.07 tonnes.

6.2.2. Emissions to water

Fertilizer industry

Besides by the application of a different production process the arsenic emission may also decrease by the use of low-arsenic ores. This option is actually considered by a major fertilizer producing company. The arsenic content of phosphate ore varies from 5-25 $mg.kg^{-1}$, the content of the ore currently used ranges from 10-20 $mg.kg^{-1}$. The emission may be decreased by appr. 50% by using low-arsenic phosphate ore, conditions being that sufficient low-arsenic ore be available. This may not be the case.

Leaching out of arsenic impregnated wood

Leaching out of arsenic may decrease by the application of alternative substances for the preservation of wood. In West Germany, for instance, chromium-copper-boron-salt based agents are used instead of arsenic. Technically as well as economically these agents are equal to arsenic-containing salts.

By impregnating wood with a steam fixation process leaching out of arsenic to water decreases in special cases (Berbee, 1989). The extent in which depending on, among other things, the kind of wood. In 1990 DBW/RIZA are expected to report on further studies concerning this subject.

Deposition

International measures to reduce arsenic emission to air are of vital importance for the deposition of arsenic to water and soil.

Emission reduction measures such as the application of dust filters whether or not combined with smoke gas desulphurization may contribute considerably to the reduction of arsenic emission to air. Considering the difference in emission between Dutch sources and certain sources abroad, emission abroad may decrease substantially as a result of which the diffuse load through deposition will also decrease.

6.2.3. Emissions to soil

Landfills

By sealing off the landfills underneath, leaching out of arsenic to soil is prevented. Arsenic should be prevented from entering surface water.

6.3. COSTS OF MEASURES AND BUSINESS ECONOMIC CONSEQUENCES

As is shown in 6.2. any possible further reduction of arsenic emission to air, water and soil is rather limited. The business economic consequences of the measures mentioned have, therefore, not been described.

6.3.1. Emissions to air

Reduction of arsenic emission as a result of combustion of fuel oil requires an estimated investment of MDfl. 100; the annual costs are appr. MDfl. 50 (Ros and Slooff, 1987).

6.3.2. Emissions to water

The application of the steam fixation process involves an annual cost of Dfl. 7.50 - Dfl. 10.- per m^3 impregnated wood. Furthermore, an investment of appr. Dfl. 100,000.- is required at an annual production of appr. 10,000 m^3 wood. In addition to these adjustments to the process a purification installation may also be installed in the impregnating plants to purify rainwater polluted with impregnating agents. The purified rainwater is stored in a cellar and largely reused for the production of salt mixtures and steam for the impregnating process.

6.4. SUMMARY AND CONCLUSIONS

The future arsenic emission to air is not expected to increase substantially. The emission increase as a result of the increasing amounts of coal to be combusted and waste to be incinerated, will mainly be compensated by improved smoke gas purification.

It is not yet clear whether the future load of the surface water from Dutch sources will decrease. The load will probably not increase.

The diffuse load of the soil will remain more or less constant, unless the import of arsenic via air and the related deposition increases with the arsenic emissions abroad.

The load of the soil caused by arsenic in waste will rise through the growing industrial activities and amounts of residual substances originating from coal combustion and domestic waste incineration.

Any possible further reduction of the load to air, water and soil by taking additional measures is limited.

7. EVALUATION

7.1. EXCEEDING OF THE CURRENT STANDARDS AND GUIDELINES

7.1.1. Soil and groundwater

The current reference value for (total) arsenic in soil [15+0.4 (L+H)] $mg.kg^{-1}$ (29 $mg.kg^{-1}$ d.m. for standard soil, see section 1.2.1.) appears generally not to be exceeded by anthropogenic influences. Data with respect to agricultural areas where previously sodium arsenite was applied in the cultivation of potatoes are not available. The reference value is exceeded in river clay under the influence of the Rhine and the Meuse, as well as in polders raised with harbour sludge. The reference value and even the C-value (50 $mg.kg^{-1}$ d.m., total arsenic) are exceeded many times naturally in soils rich in iron in the east and south of the Netherlands (total appr. 50.000 ha).
The current reference value with respect to groundwater (10 $\mu g.l^{-1}$ total arsenic) is in general not exceeded either as a result of anthropogenic emissions. Data on As concentrations in agricultural areas where previously arsenic was applied are not available. Model calculations, however, show that exceeding of the reference value and even of the B-value (testing value for further studies: 30 $\mu g.l^{-1}$) in those areas may not be ruled out. Based on these model calculations it is assumed that the As concentrations may further increase at these locations in the years to come. Exceeding also occurs in the deeper (40-300 m) groundwater in North Brabant; this, however, concerns a natural increase of arsenic, originating from glauconite layers in Belgium. These elevated arsenic contents in groundwater generally do not pose a problem, since during aeration in the drinking water preparation process coprecipitation of arsenic to iron takes place.

7.1.2. Surface water and sediment

The total contents in surface water are far below the basic quality (50 $\mu g.l^{-1}$) and the standard for the preparation of drinking water (20 $\mu g.l^{-1}$)

The quality objectives (dissolved and total content 12.5 and 16 $\mu g.l^{-1}$, respectively) recently proposed in the 3rd Memorandum Water Management (3e Nota Waterhuishouding) is in general not exceeded. However, locally, exceeding may occur near discharge points of sources as is described in chapter 2.

With regard to the contents in sediments it may be stated that the current provisional C-value (100 $mg.kg^{-1}$ d.m.) as well as the quality objectives (83 $mg.kg^{-1}$ d.m., total arsenic) proposed by the DBW/RIZA (1989) are seldom exceeded (occasionally in the Ghent-Terneuzen Canal). The arsenic contents in the sediments of state-owned waters approximate the reference value (29 $mg.kg^{-1}$ d.m.), those in non-state-owned waters and in the Wadden Sea are generally well below this value.

7.1.3. Air

For the air compartment in the Netherlands no standards or guidelines have (yet) been formulated.

7.2. RISKS AND GROUPS AT RISK

7.2.1. Risks for man

Generally it should be stated that no international consensus has (yet) been reached with regard to the risks of arsenic. In this report it has been concluded that inorganic arsenic compounds may cause skin and lung tumours in man following oral and inhalatory exposure, respectively.

Furthermore, based on the total toxicological results (from epidemiological studies, chronic animal experiments, the genotoxicity profile and metabolism studies) it is concluded that with respect to the carcinogenic effects of arsenic there is insufficient evidence to justify the use of a non-threshold extrapolation in the standard-setting.

In contrast, however, the EPA considers arsenic as a carcinogen without a threshold value and, based on this assumption, arrives at acceptable values that are below the natural levels. The WHO assumes a threshold value with respect to oral exposure but not with respect to inhalatory exposure.

- *Oral exposure*

With respect to oral exposure the WHO approach is adopted in this report. Based on epidemiological studies, where exposure took place via drinking water, the WHO arrived at a provisional maximally acceptable daily intake of 2 μg inorganic arsenic per kg body weight. Using a different parameter magnitude for the daily drinking water consumption (1.5 l instead of 2 l) and the average body weight (60 kg instead of 70 kg) results in a slightly higher maximally acceptable daily intake: 2.5 μg inorganic arsenic per kg body weight. This amount may not be considered as an ADI (acceptable daily intake): at a long-term daily intake of this dose the possibility of effects occurring may not be considered negligible. For a person of 60 kg this dose means an ingested amount of 120 μg inorganic arsenic per day (150 μg conform RIVM assumptions). For organic arsenic compounds it is assumed that the toxicity is at least a factor 10 lower.

In figure 7.1. the intake is shown of total, inorganic and organic arsenic based on 20 duplicate 24-hour feeds in the Netherlands with the highest total arsenic concentrations of a total of 311 feeds studied (according to Vaessen and Van Ooike, 1988).

With regard to the contents of inorganic arsenic it may be stated that (a) no relationship appears to exist between the total As content and the fraction inorganic As in food, and (b) the exposure is at least a factor 2 below the maximally acceptable intake. However, with respect to the less toxic organic arsenic there appears to exist a relationship with the total arsenic; appr. 70-90% of the arsenic is organically bound. The current maximal exposure levels are below the maximally acceptable daily intake (1200 μg organic arsenic per day).

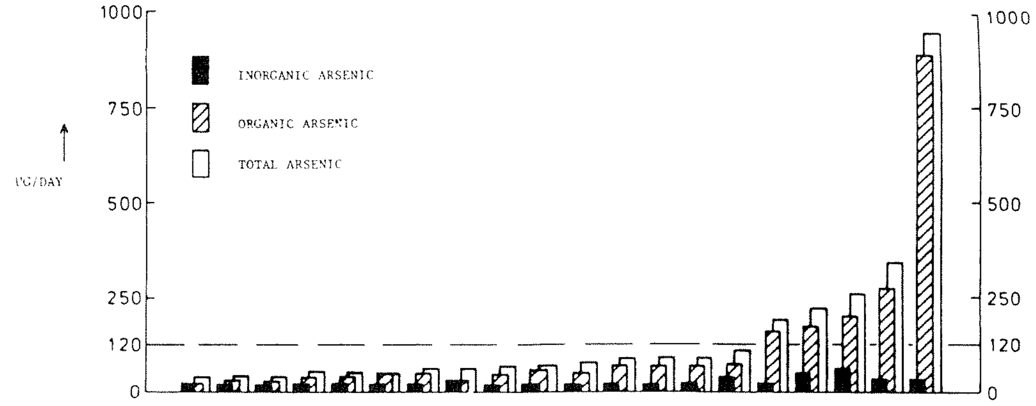

Figure 7.1. Comparison of the maximally acceptable intake of inorganic arsenic (120 µg per day) with the daily intake as determined for 20 duplicate 24-hour feeds with the highest arsenic concentrations of a total of 311 feeds studied, collected in the Netherlands in 1976-1978 and 1984-1985

Based on the above-mentioned it is concluded that the current arsenic load of food and drinking water in the Netherlands will not possibly cause any adverse effects on human health.

- *inhalatory exposure*

The toxicological recommended value for inhalatory exposure is formulated based on (toxic) effects observed in occupationally exposed groups. Assuming a concentration at which in the working environment marginal effects are still observed, using an extrapolation factor of 100, a concentration of 500 ng $As.m^{-1}$ has been proposed, at which no effects are expected to occur in the general population. The large-scale annual average arsenic concentration in the Netherlands is over a factor 100 below this. In smoky rooms the deduced concentration level is not exceeded either.

7.2.2. Risks for ecosystems

- *Aquatic environment*

With respect to the aquatic environment in accordance with the extrapolation procedure proposed by the Health Council (1988) based on chronic single-species studies, a toxicological recommended value of 6 $\mu g.l^{-1}$ (dissolved inorganic arsenic) was formulated for freshwater. A similar value was advised for the marine environment since no consistent differences in toxicity for arsenic could be observed in the two environments.

The dissolved As content in the fresh surface water in the Netherlands has in the past 10-15 years generally decreased to a level of 1-3 $\mu g.l^{-1}$. This level is below the recommended value and approximates the natural background of 1-2 $\mu g.l^{-1}$. In the pore water of the anaerobic part of the sediment the concentrations are an order of magnitude higher than those in surface water. Organisms living in sediment which do not live in the upper aerobic layer (appr. 1 cm) but in deeper ones (appr. 1-10 cm) are possibly harmed by these higher concentration levels.

In the marine environment elevated As concentrations are found near the coast and in the delta area. The highest levels were observed in the West Scheldt (2.3-5 $\mu g.l^{-1}$ dissolved), but these are also below the recommended value. Any adverse effects caused by As loads of surface water may, therefore, be ruled out, with the exception of locations in the immediate vicinity of discharge points (e.g. New Waterway?).

- *Terrestrial environment*

Although the information available is inadequate to formulate a well-founded recommended value, it is adequate to state that higher plants are probably the most sensitive to arsenic. The highest no-effect level amounted to 20 mg total $As.kg^{-1}$ d.m. (background plus added value); the lowest added concentration still causing any effects amounted to 30 mg $As.kg^{-1}$ d.m.. In nature reserves and in agricultural areas not influenced by the big rivers the range of the As contents amounts to appr. 1-33 $mg.kg^{-1}$ d.m., the highest concentrations being found in peaty/clay soils. The contents in river clay (river forelands of Rhine and Meuse) and those in harbour sludge polders are higher: 30-70 and 35-125 $mg.kg^{-1}$ d.m. on average, respectively). The ecotoxicological significance of these higher contents is not clear, since the availability of arsenic for biota is closely related to a number of physico-chemical characteristics of the soil, which are given neither in toxicological experiments nor in the soil measuring series. In this framework not only the percentage of organic matter and the lutum content should be considered, but also the iron content and the oxygen concentration (arsenic adsorbs on iron and the two elements change into different forms under reduced conditions) and the phosphate content (competition of P with As for available adsorption sites), and the influence of the pH. The anthropogenically elevated contents (usually less than 100 mg.kg^{-1} d.m.) are generally lower than the naturally elevated contents in areas rich in iron in the east and south of the Netherlands (80-390 mg.kg^{-1} d.m. on average). Also considering the tolerance which may occur, it is, therefore, assumed that the anthropogenic elevation of the As content in the soil in the Netherlands does in genereal not result in unacceptable effects on terrestrial ecosystems.

7.2.3. Effects on agricultural crops and livestock

In view of the slight accumulation in agricultural crops and the low toxicity to livestock arsenic is assumed not to have any low toxicity to livestock arsenic is assumed not to have any adverse effects on these groups at the current As concentrations in the environment.

7.3. ENVIRONMENTAL QUALITY OBJECTIVES

7.3.1. Soil (soils and sediments)

As has been stated inadequate ecotoxicological and human toxicological data are available for the formulation of an effect-oriented standard for soil. In the framework of an integral standard-setting the recommended value for soil and sediments could be deduced from that for surface water. A Kd-value of 25.10 3 l.kg^{-1} (see 4.7.) could be assumed with which a (draft) limit value of appr. 150 mg.kg^{-1} d.m. for sediment as well as for soil could be deduced (see also 7.3.2.). The Kd-value used applies, however, for pH=7.35 and a Fe-content of 2%. In a working paper concerning quality objectives for water and soil (Van de Meent et al., 1990) at desirable and limit value levels a Kd-value of 10.7×10^3 $l.kg^{-1}$ is assumed, based on experimentally determined values. This value being used the deduced value would amount to appr. 64 $mg.kg^{-1}$ d.m. As may be gathered from section 3.2., besides the applied soil properties (organic carbon and lutum) also the pH, the Fe- and P-contents are of importance in the current standard-setting. Depending on the conditions the value to be deduced may vary considerably. It is, therefore, recommended to reconsider the soil standard-setting. It might be considered to determine a maximally acceptable concentration on a general environmental quality level, and the desirable value on a local level.

7.3.2. Water (surface water and groundwater)

For surface water an ecotoxicological recommended value of 6 μg dissolved inorganic As. l^{-1} (roughly corresponding with 8 μg total As. l^{-1}) was proposed. In accordance with the current policy (NMP, 1989) this value need to be considered as a (draft) limit value (the natural level of 1-2 μg dissolved As. l^{-1} could be used as a desirable level). There are no human toxicological reasons to further accentuate this value; the value could also be applied to groundwater.

7.3.3. Air

Inadequate toxicity data are available for the formulation of an effect-oriented quality objective for air. For the time being there does not seem to be any need for the formulation of a standard, considering the difference between the concentration at which no effect is expected to occur in the general population and the maximally occurring exposure concentrations, on a national level (factor >100) as well as near sources (factor >25).

7.4. MEASURING STRATEGIES

With respect to soil and groundwater a general measuring strategy is considered of minor importance. Reasons being (a) the necessity to adjust the current standard-setting and the determination of the physico-chemical soil properties also determining the series, (b) the concentrations in a general sense are known and (c) there are no reasons to assume that in the near future significant changes in a negative sense (higher concentrations) will occur. These items are currently being furtherevaluated by the Project Group Soil Quality in the framework of the determination of the Soil Quality Measuring Network. Considering the results from model calculations it might be advisable to verify if the As concentration in groundwater actually increases in areas where previously arsenic was applied as a defoliant in the cultivation of potatoes.

With respect to surface water the national measuring programme is considered amply sufficient, certainly considering the current concentration levels and their further developments. The sediment may be analysed at the same measuring locations, a lower measuring frequency being sufficient. A more specific measuring campaign is also recommended in which attention is given to the As concentrations in the immediate vicinity of sources.

With regard to the air compartment, considering the limited importance of arsenic in outdoor air, measurements are considered of little importance.

7.5. FEASIBILITY OF ENVIRONMENTAL QUALITY REQUIREMENTS

The concentrations in the Dutch environment are largely determined by the natural background and/or emissions from abroad. The effects of autonomous developments and (possible) national measures on the concentration levels on a national scale are, therefore, extremely limited.

With regard to the soil the current reference value and even the C-level will be exceeded in great parts of the south and east of the Netherlands. In view of the fact that it concerns a natural situation, it is not important if the environmental quality requirements are met, but whether the formulation of more practically-oriented environmental requirements is feasible (see 7.2.2.). The effects of anthropogenic activities on the groundwater balance and the consequences for the arsenic fluxes are pointed

out. Through changes in oxidizing/reducing conditions the mobility of arsenic in the soil is changed considerably.
It is assumed that the As content in the soil will not increase significantly through diffuse loads, even if the deposition would increase through increasing As emissions abroad. On the other hand the load at landfill sites will increase through an increase in industrial activities and the amount of waste from coal combustion and domestic waste incineration.
With respect to surface water the current and proposed quality requirements are (generally) met. The levels in the big rivers and in the Northern Delta basis have decreased the past 15-20 years and are at present approximating the natural background level. It is assumed that the load and consequently the arsenic concentration in surface water and sediment will not increase.
For air no environmental quality requirements have either been formulated or porposed.

7.6. CONCLUSIONS AND RECOMMENDATIONS

Although insufficient data for an adequate risk assessment are available, the conclusion appears to be justified that the risks related to the current arsenic concentrations in the environment and those expected to occur in the near future in the Netherlands are in general limited.
For man the current arsenic levels are within the toxicological limits. In surface water the arsenic contents have decreased thus that the current levels generally approximate the natural background levels, risk areas with regard to damage to aquatic organisms and ecosystems being limited to the immediate vicinity of sources. A possible risk to organisms living in the anaerobic zone of the sediment might be the prevailing relatively high arsenic concentrations. With respect to the soil the anthropogenic contribution to the contents in soil and groundwater is, in comparison with the natural arsenic levels, so low, that it is assumed not to cause any effects on terrestrial organisms and ecosystems. For reasons mentioned effects on agricultural crops and livestock are not expected to occur either. Possibly with the exception of parts of the river forelands and some harbour sludge polders, as well as agricultural areas which previously were treated with As-containing pesticides.

Based on the above-mentioned data the following recommendations were made:

- Verification of the results from model calculations with regard to the behaviour of arsenic in and the arsenic load of agricultural areas, previously treated with arsenic-containing pesticides as a defoliant in the cultivation of potatoes, and the further determination of the risks for the multifunctionality of the soils.
- Reconsideration of the current soil standards (A-, B- and C-values) and the standards for the use of organic fertilizers (purification sludge in particular).
- Determination of the nature (organic/inorganic) and the concentration levels of arsenic in purification sludge in certain areas (fish processing industry). Assessment of the possible drawbacks for the soil quality at agricultural use of this sludge.

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APPENDIX to Report no. 710401004

INTEGRATED CRITERIA DOCUMENT ARSENICUM

EFFECTS

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CONTENTS

INTRODUCTION

Data in the present Appendix are underlying those in the chapter on "effects" (chapter 5) of the "Integrated Criteria Document Arsenic". The Criteria Document, prepared by the National Institute of Public Health and Environmental Protection in The Netherlands, comprises a systematical survey and a critical evaluation of the most important data on the "priority substance" arsenicum, as much as possible with regard to the specific situation in The Netherlands. The information in the Criteria Document will serve as a scientific basis for an "effect oriented policy" in The Netherlands, especially with regard to the general population and aquatic and terrestrial ecosystems.

The Criteria Document, including the present Appendix, has been written on behalf of the Ministry for Housing, Physical Planning and Environment, Directorate Substances and Risk-management. By order of this principal in the present Appendix considerable reference has been made to previous reviews; these reviews are marked in the text by "R". However, in many cases data presented in reviews are too limited for evaluation. Therefore the original publications have also been studied whenever it appeared necessary; this applies especially with regard to data which are used in the risk assessments.

Extensive reviews on chemobiokinetics and metabolism in mammals and on effects on mammals have been published by the World Health Organization (IPCS, 1981-R) and the U.S. Environmental Protection Agency (EPA, 1983-R, 1987-R). Reviews on more specific items are mentioned in the sections in question.

The data which are considered to be necessary for a risk assessment for the general population, are described in chapter 1. Data on the impact of arsenic on aquatic and terrestrial organisms are described in chapter 2 and chapter 3, respectively. In chapter 4 data on agricultural crops and livestock are described. Chapter 5 contains the risk assessment for man and the environment.

An online literature search has been conducted in september 1988, especially in order to retrieve recent publications.

human studies

After oral exposure, as in animals, soluble arsenic compounds are almost completely absorbed in humans. From data on urinary and faecal excretion, absorption was estimated to be 80-90%. From an experiment with finely powdered arsenic selenide it appears that undissolved particles are poorly absorbed. In general the absorption of trivalent arsenic is greater than that of pentavalent arsenic.

The absorption of "seafood arsenic" (mainly arsenobetaïne) is also fast and almost complete (80%-95%). Arsenic from meat of animals fed additives containing arsenic, for example arsanilic acid, is absorbed for about 40% (IPCS, 1981-R, EPA, 1983-R).

- Respiratory absorption

animal studies

Two studies indicate that arsenic in the form of an aerosol is absorbed by experimental animals. In hairless mice (used to minimize oral intake of arsenic deposited on the fur) exposed for several weeks to fly ash with a particle size < 10 μm containing 0.18 mg $As.m^{-3}$ increased tissue levels of arsenic were demonstrated. In rats exposed to condensation areosols containing 0.001, 0.004 or 0.046 mg $As.m^{-3}$ from arsenic trioxide for three months increased tissue levels were found in the two highest exposure groups. In both studies it was not possible to differentiate between inhaled and ingested arsenic (IPCS, 1981-R).

About 60% of arsinegas is absorbed by mice exposed by inhalation (IARC, 1980-R). After intratracheal administration of a solution of ^{74}As-labelled sodium arsenate (0.1-4 mg $As.kg^{-1}$ bw) to rats a rapid and complete absorption was reported. Dimethyl arsinic acid is absorbed for more than 95% within 15 min. after intratracheal instillation in rats (IPCS, 1981-R).

Human studies

With regard to the absorption of arsenic after inhalation by humans data are available from terminally ill patients or occupationally exposed persons.

1.1.2. Transport and distribution

- animal studies

After absorption arsenic compounds are mainly transported via the blood, from which it is cleared rapidly to tissues. In most animal species one or two days after administration of inorganic arsenic less than about 6% of the dose remains in the blood. The distribution pattern of the rat, however, differs from that of other species (including humans). The haemoglobin of the rat has a strong affinity for arsenic, about 50% of the dose accumulating in erythrocytes. For this reason the half-life of inorganic arsenic (both trivalent and pentavalent) in the blood of rats is relatively long, namely 60 to 90 days (i.e. the life-span of erythrocytes) (IPCS, 1981-R, EPA, 1983-R).
There are no data on blood arsenic levels of organic arsenic from seafood. With regard to the clearance of dimethyl arsinic acid (DMAA) the same difference is seen between the rat and other species. After three months about 10% of the dose was still present in the blood of rats, whereas DMAA, as major metabolite of inorganic arsenic, might be expected to be cleared from the blood fairly rapidly. Less than 6% of doses of 4 different organo-arsenic drugs, given intravenously to rabbits, remained in the blood 2 h after administration (IPCS, 1981-R).

After oral exposure of mice to arsenite or arsenate (single doses of 0.4 or 4 mg As.kg^{-1} bw) highest arsenic levels were found in kidneys, liver and bile. After exposure to arsenite most tissues contained higher amounts of arsenic than after exposure to arsenate, especially the liver and the biliary gland. After intraperitoneal injections of lower doses (up to 25 μg As.kg^{-1} bw) in rabbits these differences were not seen. After parenteral exposure of several animal species to inorganic arsenic compounds an initial acumulation in liver, kidney, spleen, skin and lung was found. In most organs the concentration declined rapidly, with the exception of the skin. This could be explaned by the reaction of trivalent arsenic with sulphydryl groups of proteins, which are abundant in the skin. Arsenite and arsenic trioxide were found to pass the blood-brain barrier in many species. The concentration in the brain, however, was lower than in the

In humans arsenic is usually found in higher concentrations in hair and nails than in other parts of the body. These two matrices contain high amounts of keratin with SH-groups to which arsenic binds (IPCS, 1981-R). In populations exposed via drinking water mean arsenic levels of hair and urine showed increasing concentrations with increasing exposure level. The blood arsenic levels did not increase until the water contained about 400 $\mu g\ As.l^{-1}$ (Valentine et al., 1979).

1.1.3. Elimination

- animal studies

In all species (except the rat) absorbed inorganic arsenic is excreted rapidly, mainly in the urine. Experiments with arsenic trioxide in various species show that between 70 and 85% of the dose (0.1-1 mg $As.kg^{-1}$ bw) , administered by various routes, was excreted in the urine. Elimination in the rat occurs very slowly because of the accumulation in erythrocytes mentioned earlier. The elimination in dogs follows a two phase model, with halflife-times of 6 hours and 2.4 days. The amount of arsenic excreted into the bile differs between animal species. Trivalent arsenic is excreted to a lesser extent than pentavalent arsenic. Elimination via the lungs is of minor or no importance.

Arsenobetaine administered to animals is also eliminated rapidly. In general between 50%-90% of the given dose will be excreted in the urine within three days. In contrast, arsanilic acid is eliminated predominantly in the faeces. In rats and guinea pigs the influence of the molecular structure of some arsenic-containing drugs on the elimination kinetics was demonstrated. Drugs with functional hydrophilic groups, that facilitates elimination without biotransformation, are eliminated rapidly (65-90% in 24 h). Hydrophobic drugs were excreted slowly (20-50% in 24 h), predominantly into the bile (IPCS, 1981-R).

- human studies

In humans urine is also the predominant excretion route. The excretion of inorganic arsenic after administration of a single dose amounts to 30%

In a few reports an interconversion of the two valency forms of arsenic was suggested. Methylated forms of arsenic, which could have influenced the results, were not taken into account, so no conclusions can be made sofar. When DMAA was given orally to rats it appeared to be mainly unchanged. Some demethylation seems to occur. Organic arsenic compounds used as feed additives and as drugs are converted to more easily excretable compounds (IPCS, 1981-R).

- human studies

Following ingestion or inhalation of inorganic arsenic between 70% and 85% is excreted as methylated arsenic. The major excretion products are dimethyl arsinic acid and methyl arsonic acid accounting for about 65% and 20%, respectively, of the total quantity excreted. A minor part of inorganic arsenic is excreted unchanged; after drinking arsenite-rich wine this appears to be 10% of the dose. The pattern of biotransformation was reported to be independent of route of uptake and probably takes place in the liver. However, the intestinal flora could also play a role in this process (Wibowo et al., 1982-R).

Several studies indicate that organic arsenic compounds, mainly from seafood, are excreted without prior biotransformation. Arsenobetaine, which accounts for 70% of the arsenic in seafood, was demonstrated in the urine after consuming of rock lobster tails (IPCS, 1981-R).

Summary and conclusions "kinetics and metabolism"

In the present report arsenic is considered to be nonessential.

After oral exposure the absorption percentage of water soluble inorganic arsenic compounds is circa 80% to 100% in animals and 80% to 90% in humans. The absorption of "seafood arsenic" (mainly arsenobetaïne) is nearly complete. From experimental studies and occupational data it was estimated that the absorption of water soluble inorganic arsenic compounds after inhalation is 30% to 60%. The absorption percentage of insoluble compounds is lower after both exposure routes.

In most species, including man, the absorbed arsenic is rapidly cleared from the blood to various tissues, with initial accumulation in liver, in guinea pigs and rats were 24 and 40 $mg.kg^{-1}$ bw (7 and 12 mg $As.kg^{-1}$ bw), respectively. Those for phenarzarsine oxide were 77 and 83 $mg.kg^{-1}$ bw (23 and 25 mg $As.kg^{-1}$ bw), respectively (IPCS, 1981-R). After a single oral dose of PXO (10 mg $As.kg^{-1}$ bw) to rats hepatotoxic effects were observed. When rats were given a similar dose of PZO no toxic effects were seen.

In a subacute study rats were given arsenic trioxide at doses of 1.5 or 7.6 mg $As.kg^{-1}$ bw per day by gavage for 40 days. Rats receiving the higher dose showed hairloss and eczema, hyperplasia and hyperkeratosis of the skin. Bleeding, ulceration and crustforming also occurred. No adverse effects occurred in the animals from the dose lower group. Dysfunction of the bloodbrain barrier was indicated in rats fed 290 mg $As.kg^{-1}$ bw as arsenite for 35 days. In rats given 40, 85 or 125 mg $As.l^{-1}$ in drinking water for 6 weeks an increase in relative kidney weights was found (IPCS, 1981-R, IARC, 1980-R).

An impaired resistance to viral infections has been reported in mice subacutely exposed to inorganic arsenic by different routes; subcutaneously (2-4 mg $As.kg^{-1}$ bw), in drinking water (75-150 $mg.kg^{-1}$ bw) or via intraperitoneal injection (1.8 mg $As.kg^{-1}$ bw). Impaired kidney function was also described in rabbits intravenously exposed to 0.6 mg $As.kg^{-1}$ for 2-12 weeks three times a week (IPCS, 1981-R).

The most toxic form of arsenic seems to be arsine gas (Coddington, 1986-R). The LC50 for arsine in mice by inhalation has been estimated to be 500 $mg.m^{-3}$ after 2.4 min. Exposure to 75 $mg.m^{-3}$ arsine during 30 min may be lethal for mice. In a subacute study rats were exposed to aerosols containing dimethylarsinic acid. It was estimated that the concentration in the aerosols was approximately 840 $mg.m^{-3}$. No increased mortality occurred. In another study rats were inhalatory exposed to PXO and PZO (0.3-0.6 mg $As.m^{-3}$) 5 h a day during 7 or 8 weeks. No toxic effects were seen except for cellular infiltration of the portal tracts of the liver (IPCS, 1981-R).

Intraperitoneal injection of arsine in mice resulted in a LD50-value of 2.5 mg $As.kg^{-1}$ bw (IARC, 1980-R). The intraperitoneal LD50-values of methanearsonic acid disodium salt and dimethylarsinic acid in mice were about 550 $mg.kg^{-1}$ bw. Guinea pigs given 0.07 mg $As.kg^{-1}$ bw as sodium arsenate intraperitoneally for 2 months showed effects on the ear (leading during the convalescence. Dermatitis occurred in some patients treated with arsobal (organic arsenic compound) for trypanosomiasis (IPCS, 1981-R). Inhalation of arsine concentrations of 25 $mg.m^{-3}$ for 30 min. can be fatal. Exposure to airborn arsenic compounds (mainly arsenic trioxide) in a smelter caused irritation of the respiratory tract; conjunctivitis and dermatitis (IARC, 1980-R).

1.2.2. Long-term exposure (subchronic and chronic toxicity, exclusive of carcinogenicity)

- Animal studies

In rats fed arsenate at a concentration of 50 mg $As.kg^{-1}$ bw for 10 weeks a decrease in haematocrit and haemoglobin was found. Liver cirrhosis and necrosis and bile duct proliferation were seen in rabbits fed daily doses of 0.7-4.7 mg $As.kg^{-1}$ bw as lead-, copper- or sodium arsenate for 50-250 days (IPCS, 1981-R). In rats given drinking water containing arsenic trioxide at levels of 0, 0.125, 12.5 or 62.5 mg $As.l^{-1}$ for 7 months liver injury and a dose-dependent proliferation of the bile duct was found (Ishinishi et al., 1980). In another study (performed by the same group), however, in which rats were exposed to arsenous acid in drinking water at concentrations up to 62.5 $mg.l^{-1}$ (corresponding to a maximum dose of 6.1 mg $As.kg^{-1}$ bw per day) for 54 weeks no toxic effects were found as related to growth, physical appearance or haematology. Slight temporary biochemical changes related to liver changes were observed at the 15th week, but no differences with the controls were found thereafter. It was suggested that: "an adaptation to the toxic effects of arsenic does seem to occur, at least, in rats" (Hisanaga, 1982).

In an oral study in which rats were fed a "shrimp diet" containing about 14 mg $As.kg^{-1}$ (0.7 mg $As.kg^{-1}$ bw) for 12 months no toxic effects were seen. On the basis of this study the toxicity of "seafood arsenic" seems to be relatively low.

Minor changes in kidney function and histology were found in rats orally exposed to calcium- and leadarsenate at doses of 1.5 and 1.25 mg $As.kg^{-1}$ bw, respectively, for two years. In a chronic feeding study, which was

arsenite (12, 23 or 26 mg $As.kg^{-1}$ bw) on one of days 8 to 15 of gestation. The lowest dose did not cause embryotoxic or teratogenic effects and no maternal toxicity was observed. At the higher doses both embryotoxic effect and maternal toxicity was found. Low incidences of malformations (exencephaly and open eyes) occurred in these groups (Baxley et al., 1981). Teratogenic effects were seen after intraperitoneal injections of 45 $mg.kg^{-1}$ bw sodium arsenate in mice (11 mg $As.kg^{-1}$ bw, based on hydrated sodium arsenate) (Hood and Bishop, 1972). In rats given arsenate (5-12 mg $As.kg^{-1}$ bw) teratogenic effects included eye-defects (IPCS, 1981-R). Intravenous injections of 3 mg $As.kg^{-1}$ bw or more as arsenate to hamsters on the 8th (or 9th) day of gestation caused several teratogenic effects. An increased incidence of exencephaly, anencephaly, renal agenesis, rib and genito-urinary abnormalities was found. Both resorption and malformation rates in the fetus increased with increasing doses of arsenate (IPCS, 1981-R, Friberg, 1986-R).
In a three-generation experiment mice were exposed to 5 mg $As.kg^{-1}$ diet (about 0.5 mg $As.kg^{-1}$ bw) in the form of arsenite. Except for a reduced litter size no abnormalities were found. In a 7-generation study rats were fed diets containing 0.01, 0.02, or 0.05% arsanilic acid (3.5, 7, or 17.5 mg $As.kg^{-1}$ diet). No teratogenic effects were seen. The litter sizes and the survival of the pups were found to increase significantly (IPCS, 1981-R).

The embryotoxic effects of inhaled arsenic were studied in mice. Pregnant mice were exposed to 0.21, 2.3 or 22 mg $As.\ m^{-3}$ as arsenic trioxide for 4 h per day during days 9 through 12 of gestation. At the highest concentration a significant increase in the number of fetuses with retar-ded growth was observed. The number of fetuses with skeletal malformations was also significantly increased at the highest concentration. The average fetal weight decreased at all concentrations compared to the controls (Nagymajtenyi et al., 1985).

- <u>Human studies</u>

Chronic effects of arsenic may involve many organs or organ systems, including the skin, the liver, the respiratory and gastrointestinal tract,

hyperkeratosis. Multiple keratosis can occur and develop into precancerous states (IPCS, 1981-R).
Hyperkeratosis and hyperpigmentation are rarely seen among smelter workers exposed to inorganic arsenic by inhalation, but have been reported in other occupational situations. In smelter workers dermatosis due to local irritation may occur (IPCS, 1981-R).

effects on the respiratory tract
Effects of arsenic on the respiratory system have been reported primarily as a result of occupational exposure. A few reports describe respiratory effects after oral exposure. In one study, for example, a high frequency of chronic cough and a history of bronchopulmonary disease were reported among inhibitants of Chili exposed to arsenic via drinking water. Because arsenic can suppress the immune response (see "other effects") these effects may be due to an impaired resistence to infections (IPCS, 1981-R, EPA, 1983-R).
After inhalatory exposure to relative high arsenic concentrations in smelters, lesions of the mucous membranes in the respiratory tract and perforation of the nasal septum were reported. Among copper smelter workers in Sweden, exposed to arsenic levels up to 7 mg As.m^{-3} in some workplaces, but generally not to a higher level than 0.5 mg As.m^{-3}, two types of respiratory syndroms were found. Symptoms of the upper respiratory tract (septum perforation, laryngitis and changes in nasal mucosa) were mainly found in workers exposed to arsenic in the "crude or refined form". The other syndrom, which included tracheobronchitis and signs of pulmonary insufficiency, was found among workers with mixed exposure to arsenic and sulfur dioxide. No control group was used and smoking habits were not included (IPCS, 1981-R, EPA, 1983-R).

effects on the liver
Chronic oral intake of inorganic arsenic has been associated with the development of portal hypertension, malignant liver disease and cirrhosis. Long-term intake of drugs containing inorganic arsenic (mainly Fowler's solution containing arsenite) resulted in portal hypertension and sometimes in liver cirrhosis. In patients treated with organic arsenic (arsobal for treatment of trypanosomiasis) liverdamage can occur as side-effect.

Peripheral vascular diseases have rarely been reported among occupationally exposed populations or among patients receiving arsenic medications. In one study, however, some changes in peripheral circulation were found among copper smelter workers with long-term exposure to arsenic dust (mainly containing arsenic trioxide). An increase in vasospastic reactivity as well as phenomenon of Raynaud were found, which could be due to functional alternations in the vessels of the hand. It was estimated that at time of study the maximal daily absorption did not exceed 300 μg. It was not possible to determine if the increased reactivity was due to past (significantly higher) or present exposure (Lagerkvist et al., 1986). A few studies indicated an increased mortality from cardiovascular diseases among smelter workers (Lee and Fraumeni, 1969; Lee-Feldstein, 1983), but in other studies no excess in cardiovascular mortality was found (EPA, 1983-R).

effects on the nervous system

Peripheral and central nervous system (CNS) injury are important effects after inorganic arsenic exposure. Neurological involvement is demonstrated by several sensory changes and considerable muscle tenderness. Varying degrees of motor weakness, more severe at proximal parts are seen, which can develop into paralysis of affected muscle groups or extremities. Other symptoms of arsenic intoxication are headache, sleepiness, and in severe cases loss of memory and confusion. Changes in personality are also reported (EPA, 1983-R; IARC, 1980-R).

A high percentage of electromyographic (EMG) abnormalities was reported among persons using drinking water containing more than 0.05 mg $As.l^{-1}$. Non of the controls showed these abnormalities. Several factors that would predispose people to have EMG abnormalities were not taken into account (IPCS, 1981-R). From a neurological examination of a population in Alaska it appeared that a daily ingestion between 1 and 4521 μg arsenic from well water did not result in clinical or subclinical neuropathy (Kreiss et al., 1983). Among patients having taken anti-asthmatic herbal preparations containing inorganic arsenic, more than 50% had neurological complications, mostly sensimotor polyneuropathy. The recommended daily dose for these patients contained several mg arsenic trioxide or arsenic sulfide. Other symptoms of arsenic poisoning were also seen in this group. In patients treated with arsobal (organic arsenic) for trypanosomiasis (dose not given) the smelter during pregnancy compared to the population living nearby. (In this population there was no variation in total or specific frequencies). Frequencies of spontaneous abortion were studied in populations located at different distances from the smelter. In the population located near the smelter a significant increase in abortion (11% of the pregnancies) was found compared to the population living further away (7.6%). It must be kept in mind that the population living closest to the factory includes many employees of the smelter. A decreased birthweight in the population living near the smelter was also reported. The studies indicate that workers at the smelter and probably people living close by the smelter have an increased risk of genetic damage (Nordström, 1978, 1979). Ingestion of 30 ml of a rat poison containing 1.32% arsenic as arsenic trioxide by a woman in the 30 th week of pregnancy caused death of the infant (Lugo et al., 1969).

Summary and conclusions "short- and long-term toxicity"

The toxicity of arsenic compounds depends on the chemical form and the oxidation state. In general, the inorganic arsenic compounds appear to be more toxic than organic compounds and trivalent inorganic compounds more toxic than the pentavalent ones. Oral LD50-values range from 10 to 130 mg $As.kg^{-1}$ bw for inorganic arsenic compounds and from 700 to 2,800 $mg.kg^{-1}$ bw for organic compounds. The lethal oral dose for humans ranges from 70 to 180 mg arsenic trioxide (0.8-2.3 mg $As.kg^{-1}$ bw). Inhalation of 25 $mg.m^{-3}$ arinegas may be lethal.

After long-term exposure to inorganic arsenic in both animals and humans effects are seen on several organs or organ systems. In a subchronic study on rabbits fed 0.7 to 4.7 mg $As.kg^{-1}$ per day as arsenate liver cirrhosis and (a dose-dependent) proliferation of the bile duct was found. Subchronic exposure to arsenic trioxide via drinking water (doses up to about 6 mg $As.kg^{-1}$ bw) caused the same symptoms. In a chronic study (rats exposed through drinking water to same doses) with arsenous acid no toxic effects were appearent. In rats orally exposed to 1.5 mg $As.kg^{-1}$ bw per day as arsenate for 2 years minor changes in kidney function were found. In a chronic feeding study in which cats were given 1.5 mg $As.kg^{-1}$ bw as

slight subclinical neuropathy was found. It was estimated that at time of study the concentration was about 50 µg $As.m^{-3}$ and that maximal daily absorption did not exceed 300 µg. It was, however, not possible to determine if the effects were due to present or past exposure, when it was significantly higher (up to 500 µg $As.m^{-3}$). The risk on clinical neuropathy is considered to be very low when exposure is kept below 50 µg $As.m^{-3}$. In workers exposed to levels below 13 µg $As.m^{-3}$ (as a 6-h average) no significant increase in liver enzymes was found.
One study on the offspring of women working at a smelter indicates an increased frequency of spontaneous abortions and malformations in the offspring. The women were also exposed to other toxic substances, so no conclusion can be drawn on the role of arsenic.

1.3. GENOTOXICITY

- Results from tests:

Various inorganic arsenic compounds have been tested for genotoxicity in a variety of test systems. Data on the genotoxicity have been reviewed among others by Jacobson-Kram and Montalbano (1985), IARC (1980, 1987) and IPCS (1981). According to Jacobson-Kram and Montalbano the following conclusions can de drawn ;

1. Arsenic is either inactive or extremely weak for the induction of gene mutations *in vitro*.
2. Arsenic induces chromosome aberrations and sister chromatid exchanges (SCE's) in a variety of cell types, including human cells, *in vitro;* trivalent arsenic is approximately an order of magnitude more potent than pentavalent arsenic.
3. Arsenic does not appear to induce chromosome aberrations *in vivo* in experimental animals.
4. Several studies suggest that humans among others exposed to arsenic demonstrate higher frequencies of SCE's and chromosomal aberrations in peripheral lymphocytes.

DNA) in place of posphorus (Kay, 1965). Inorganic arsenic has also been reported to inhibit DNA repair enzymes (Rossman et al., 1977, Jung and Trachsel, 1970).

The unusual genotoxic profile (i.e. the induction of chromosome aberrations *in vitro* but not of gene mutations) might be due to the strong interaction of arsenic with sulfhydryl groups of enzymes.

Summary and conclusions "genotoxicity"

From *in vitro* genotoxicity tests it appeared that inorganic arsenic compounds do not induce gene mutations. But on the other hand in both animal and human cells increased frequencies of SCE's and chromosome aberrations were found. However, in the only *in vivo* experiment (mice) no induction of chromosome aberrations could be found in bone marrow cells or in spermatogonia. Organic arsenic compounds did not induce gene mutations nor a higher frequency of SCE's *in vitro*.

In peripheral lymphocytes from people exposed to inorganic arsenic higher frequencies of chromosome aberrations and SCE's were demonstrated. However, because of the fact that these persons were simultaneously exposed to other substances, it can not be ruled out that the positive results must be ascribed to other factors than arsenic exposure.

Possible explanations for the unusual genotoxic profile of inorganic arsenic are the inhibition of DNA repair enzymes, the substitution of arsenic instead of phosphorus in the DNA-backbone, or the interaction of arsenic with sulfhydryl groups of enzymes.

On the basis of the data above mentioned it is concluded that, as far as exposition-routes which are relevant for humans are concerned, there is inadequate evidence for the carcinogenicity of arsenic in experimental animals. The "International Agency for Research on Cancer" (IARC, 1980, 1987) arrived at the same conclusion.

1.4.2 Human studies

Data on the carcinogenicity of inorganic arsenic in humans have been reviewed by the IPCS (1981), the EPA (1983), the IARC (1980, 1987), Pershagen (1981) and the WGD (1984). With regard to organic arsenic compounds no data could be found.

- Cancer of the skin

Chronic oral exposure to inorganic arsenic has been associated several times with an increased incidence of skin cancers. This relationship was found in several studies on populations living in different parts of the world which were exposed through naturally contaminated drinking water. Long-term ingestion of drugs containing inorganic arsenic was associated with an increased incidence of skin cancer as well. The medicines contained mainly arsenite. It is assumed that the arsenic present in drinking water is of inorganic nature, but its valence is often not known. A short overview of epidemiologic studies on orally exposed populations is given in table 1.2. A number of studies will be described below.

A general survey was carried out among more than 40,000 inhabitants from an area of Taiwan with high concentrations of arsenic in well water. The arsenic level of water was reported to range from 0.01 to 1.8 mg $As.l^{-1}$ (mainly from 0.4 to 0.6 mg $As.l^{-1}$, mean 0.5 mg $As.l^{-1}$). Skin cancers were found in 428 of 40,421 examined inhabitants. The most common type was intra-epidermal carcinoma and the body areas most frequently involved were unexposed surfaces. Nearly all patients had multiple skin cancer. A positive dose-response relationship was demonstrated between intake and the prevalence of skin cancer (Tseng, 1968, 1977). A control-group was not included in this study. In another study cancer mortality rates in the same

Additives" (JECFA) concluded that "there is epidemiological evidence of an association between the overexposure of humans to inorganic arsenic from drinking water and increased cancer risk". Additionally, the JECFA stated: "Human exposure to levels of arsenic below those which cause arsenicism do not appear to carry a carcinogenic risk" (JECFA, 1983).

- Cancer of the respiratory tract

There is substantial evidence of increased incidence of cancer of the respiratory tract in association with exposure to inorganic arsenic (and other substances) by inhalation. Epidemiological studies on smelter workers demonstrate in most cases a positive relation between exposure to inorganic arsenic and respiratory cancer. Similar results were found in groups handling pesticides containing inorganic arsenic. A short overview of epidemiologic data is given in table 1.3. and a number of studies will be described below.

exposure to pesticides
The mortality of workers exposed to arsenic was compared to that of workers of the same factory who had no exposure to arsenic. Several pesticides containing arsenic were produced. Airborn arsenic levels were reported up to 40 mg $As.m^{-3}$. A positive dose-response relationship was found between the total dose of arsenic and lung cancer mortality. In the exposed group 16 of the 28 deaths due to respiratory cancer occurred in workers exposed less than one year. An increase in cancers of the lymphatic system and haematopoietic system, other than leukaemia was also found (Ott et al., 1974). It should be noted that only those workers who were employed until death or who retired were used. This means a loss of data especially on those exposed to low concentrations (IPCS, 1981-R). Corresponding to the exposure categories made by Ott et al. 48-h time- weighted averages (TWA) were calculated over 40 years of exposure. If workers exposed for less than one year were excluded, no clear dose-response relationship was observed below a TWA of 90 μg $As.m^{-3}$ daily (Blejer and Wagner, 1976, evaluated in reviews). A retrospective cohort analysis on the same population resulted in a relative risk (RR = 3.5) of lung cancer in the exposed group compared to U.S. mortality rates. The relative risk of lymphatic and haematopoietic

well informed. The authors concluded that the specific cause of increased mortality remains to be identified because there was also no difference in smoking habits (Wicklund et al., 1988). The negative finding of this study was consistent with a study in which no relationship could be found between exposure to arsenates (either lead or calcium arsenate) and lung cancer in agricultural setting (Barthel et al., 1981, cited by Wicklund, 1988).

exposure in smelters

Among copper smelter workers exposed to high levels of arsenic an increased risk of lung cancer has been reported many times (see table 1.3).

In one study the mortality rates among more than 8,000 white male smelter workers in the USA were compared with that of the white population of the same states. A positive dose-reponse relationship was found between SMR and estimated degree of arsenic exposure, with the SMR in the highest exposure group being 800. The results are, however, difficult to interpret because of the fact that a positive dose-response relationship was also found between exposure to sulphur dioxide and respiratory cancer risk. Data on smoking habits and age-distribution in the groups were not given (Lee and Fraumeni, 1969). The mortality of the same cohort was studied again with a broader follow-up period (1938-1977). Relative exposure categories were made. The mortality due to lung cancer increased with an increasing degree of arsenic trioxide exposure and with an earlier period of first employment (Lee-Feldstein, 1983). The risk of lung cancer also increased (lineair) with cumulative exposure indices (Lee-Feldstein, 1986). In another study a sample of 1,800 men of the cohort originally studied by Lee and Fraumeni was examined. Three indices of arsenic exposure were developed; TWA [<100 μg As.m^{-3}, 100-499 μg As.m^{-3}, 500-4999 μg As.m^{-3} and >5000 μg As.m^{-3}], 30-day ceiling and cumulative. A dose-response relationship existed between arsenic exposure and respiratory cancer. Ceiling arsenic exposure seemed to be more important than TWA exposure. It appeared that workers whose exposure was kept below 500 μg As.m^{-3} demonstrated no excess in lung cancer mortality. Smoking habits, although contributing to the respiratory cancer incidence did not appear to be as important as arsenic. Sulphur dioxide and asbestos (indirect estimated) did not appear to be important in the excess of respiratory cancer, although sulphur dioxide and arsenic exposures could not be separated completely (Welch et al., 1982, Lamm and Lederer, 1984).

several reports it was suggested that inorganic arsenic compounds should be considered as co-carcinogens (Ishinishi et al., 1977; Rossman, 1981; Lee et al., 1988). In one study the tumor promoting potential of arsenite, arsenate and dimethyl arsinic acid in the kidneys of rats was determined. It was found that arsenite and arsenate did not have any initiator activity in "intact" male Wistar rats, but significantly promoted diethylnitrosamine (DENA)-initiated renal tumors in these animals. Dimethyl arsinic acid had neither initiator nor promotor activity. A dose dependent effect (not significantly) for arsenite promotion in partially hepatectomized DENA-initiated rats was found at concentrations of 40, 80 and 160 mg $As.l^{-1}$ in the drinking water (EPA, 1987b).

Summary and conclusions "carcinogenicity"

From experimental studies it appeared that long-term oral or inhalatory exposure to inorganic compounds did not result in increased cancer incidences. After repeated intratracheal administration the development of benign lung tumours were reported. An increase in number of malign tumours were observed after intravenous or subcutanous administration as well as after exposure to arsenic containing mixtures. It was concluded that there is inadequate evidence for carcinogenicity in animals for exposure-routes relevant for humans. The IARC (1980, 1987) arrived at the same conclusion.
A mechanistic study indicated that both tri- and pentavalent inorganic arsenic have "tumor promoting" activities. In some other studies it was suggested that inorganic arsenic should be considered as a co-carcinogen.

In epidemiological studies an association was found between long-term oral exposure to inorganic arsenic and skin cancer. Several studies on populations exposed through drinking water or medication showed increased skin cancer incidences. Most studies also showed positive dose-response relationships; for example the "drinkingwater" study from Taiwan [among more than 40,000 people using water containing up to 1,820 μg $As.l^{-1}$ (mean 500 μg $As.l^{-1}$)]. In one study conducted in Oregon, however, no increased skin cancer incidences were found. Mean arsenic levels in this study were much lower (mean 16 μg $As.l^{-1}$) than in the other ones. It is assumed that

Table 1.1 Short summary of experimental carcinogenicity studies. The data are mainly derived from the IARC (1980) and the IPCS (1981). Some recent studies were also summarized, from these studies the references are given at the foot of the table (see "notes").

Species	compound(s) tested	dose/duration	effect	note
Oral exposure ;				
exposure through drinkingwater				
mouse and rat	As_2O_3	within 15 months arsenic level increased from 3 to 26 mg As/l estimated daily intake was between 0.08 and 0.6 mg per animal	-	
mouse	As_2O_3	76 mg As/l for lifespan	-	
mouse and rat	$NaAsO_2$	5 mg As/l for lifespan	-	
mouse	$NaAsO_2$	5.8 mg As/l for 60 weeks	-	
mouse	As_2O_3	15 mg As/l for lifespan, or in addition after 6 months painting with DMBA or croton oil.	-	
mouse	As_2O_3	76 mg As/l for 3-14 weeks did not enhance carcinogenesis induced by 3-methylcholanthrene.	-	
mouse	As_2O_3	76 mg As/l for 40-60 weeks in combination with treatment with crotonoil, DMBA or urethane.	-	
exposure through the diet				
mouse	DMAA	46 mg/kg bw. per day at an age of 7-28 days and than for 18 months 121 mg/kg diet	-	
mouse	AA	500 then 250 mg/kg diet AA or 338 then 169 mg/kg diet potassium arsenite and two skin applications of DMBA and crotonoil	-	
rat	$Pb_3(AsO_4)_2$ $Ca_3(AsO_4)_2$	daily doses of about 2 mg As for 2 years	-	
rat	$NaAsO_2$ or Na_3AsO_4	up to 250 mg As/kg diet as sodium arsenite (25 mg As/kg bw) or up to 400 mg As/kg diet as sodium arsenate (40 mg As/kg bw) for two years.	-	
rat	$Pb_3(AsO_4)_2$ Na_3AsO_4	up to 400 mg As/kg diet as lead arsenate or 100 mg As/kg diet as sodium arsenite (40 or 10 mg As/kg bw, respectively) for 29 months either with or without additional administration of NDEA 5 days a week for 29 months.	-	
dog	$NaAsO_2$ or Na_3AsO_4	up to 125 mg As/kg diet (3.1 mg As/kg bw) for 2 years.	-	
Exposure by gavage				
mouse	As_2O_3 or	weekly doses of drugs containing arsenic trioxide for 5 months total dose ± 220 mg As/kg bw.	m	(1)
Dermal exposure				
mouse	$KH_2(AsO_2)_2$	10 weekly administrations, total dose 8.7 mg As after 25 days once weekly applications of croton oil.	-	(2)
mouse	$KH_2(AsO_2)_2$	8 applications of arsenite over 5 days (total dose 1.2 mg arsenite per animal), followed by 2 weekly applications of croton oil.	-	
		1 application of DMBA, followed by applications of arsenite (total dose 64 mg arsenite per animal per week).	-	
mouse	Na_3AsO_4	2 weekly application of arsenate for 60 weeks with or without additional treatment with crotonoil, DMBA or urethane.	-	
Exposure by inhalation				
mouse	$NaAsO_2$	1% (w/w) aqueous solution of sodium arsenite 20-40 min/day 5 days a week for 55 weeks	-	(3)

Tabel 1.2 Overview of epidemiological studies concerning long-term oral exposure to inorganic arsenic (through drinking water) and skin cancer risk.

Studied population (reference)	Chemical info/ source	Dose/duration	Effects / dose-response relationship
A group of skin cancer patients, Oregon case-control study (Morton et al., 1976)	drinking water from different sources	mean: 16 μg $As.l^{-1}$, range: 0-2150 μg $As.l^{-1}$	No positive relationship was found between arsenic levels in drinking water and skin cancer incidence.
general survey of more than 40000 inhabitants from south-west Taiwan (Tseng, 1977)	artesian well water	mean: 500 μg $As.l^{-1}$, range: 0-1,800 μg $As.l^{-1}$	A positive dose-response relationship was found between arsenic levels in drinking water and incidences of skin cancer.
998 exposed vs. 1488 non exposed persons from North Mexico (Cebrian et al., 1983)	well water, for 70% pentavalent arsenic	exposed: 0.41 mg $As.l^{-1}$ control: 0.007 mg $As.l^{-1}$	21.6% of the exposed population showed at least one of the cutaneous signs of chronic arsenic poisoning against 2.2% of the control population. RR's were 3.6 to 36 for developing cutaneous lesions in the exposed group compared to the control group.
residents of a BFD-endemic area in Taiwan different villages (Chen et al. 1985)	high arsenic artesian well water, valence state unknown	0.35-1.14 mg $As.l^{-1}$ estimated total daily intake as high as 1 mg	SMR's for cancers of bladder, kidney, skin, lung, liver and colon were 1100, 772, 543, 320, 170, 160 resp for males and 2009, 1119, 652, 413, 229 and 168 resp. for females. A dose-response relationship was observed between SMR's of the cancers and BFD-prevalence rate of the villages.
204 cancer patients 368 controls, Taiwan, case-control study (Chen et al., 1986)	either artesian well water or shallow well water was used, valence state unknown	artesian: 0.35-1.14 mg $As.l^{-1}$; shallow: 0-0.04 mg $As.l^{-1}$	A positive dose-response relation was observed between exposure to artesian well water and cancers of bladder, lung and liver. O.R.'s were 3.90, 3.39 and 2.67 for those who used well water for ≥ 40 yr compared to those who never used well water.
67 exposed persons vs. 96 non exposed persons rural West-Bengal India (Guha Mazumder et al., 1988)	tubewell water, valence state unknown	exposed group: 0.2-2 mg $As.l^{-1}$ c-group: ≤ 0.5 mg $As.l^{-1}$	exposed group: 93% showed evidence of chronic dermatosis and hepatomegaly control group: 6.3% had non-specific hepatomegaly, no skin lesions.

Table 1.3 (continued)

studied population (reference)	exposition to and concentration(s)	results	remarks
Exposition in smelters			
8000 copper smelter workers, Montana follow-up 1938-1963 (Lee & Fraumeni, 1969)	arsenic trioxide and sulphur dioxide, relative exposure categories	smelterworkers had an excess mortality from respiratory cancer (observed 147 versus 44.7 expected; SMR=329, $p<0.01$). The risk increased in proportion to the degree of exposure to arsenic and sulphur dioxide. SMR's were 239, 478 and 667 in those with heavy, medium and light exposure. The excess was 8-fold for workers who were for more than 15 years heavily exposed to arsenic.	no data on smoking habits and actual exposure levels. effects of arsenic or sulphur dioxide difficult to seperate.
idem follow-up 1938-1977 (Lee-Feldstein, 1983, 1986)	idem	a dose-response relationship was found beteen degree of exposure to arsenic trioxide and risk of lung cancer. Highest SMR was found for men heavily exposed and employed before 1925 (SMR=800), from another analysis by the same author it appears that the cancer mortality increases linear with cumulative exposure index, with SMR from 200 to 900.	
idem (a sample of 1800 workers) follow-up 1938-1977 (Welch et al., 1982)	idem TWA-categories were made; <100; 100-499; 500-4999 and >5000 µg As.m^{-3}	A dose-response relationship was found between exposure categorie and respiratory cancer risk. Ceiling exposure seemed to be more important than TWA-catergories. It was suggested that exposure <500 µg As.m^{-3} did not increase lung cancer risk.	
copper smelter workers Tacoma Washington, follow-up 1950-1971 (Milham & Strong, 1971, evaluated in reviews)	arsenic, among others	there were 40 observed lung cancer deaths versus 18 expected, SMR=222, $p<0.001$	methodof calculating expected deaths inadequately described (IARC, 1980).
metal workers Japan, follow-up 1949-1971 (Tokudome & Kuratsume, 1976, evaluated in reviews)	arsenic, among others	an sign. increased mortality for lung cancer(trachea, lung and bronchus) was found (29 observed vs. 2.44 expected; SMR=1189, p,0.01). A positive dose-response relationship was found between SMRs for lung cancer and duration of employment as well as between estimated levels of exposure and SMR. There was also an increase in mortality from coloc cancer (3 vs. 0.6) and in mortality from liver cancer (11 vs. 3.26).	no detailed information about smoking habits, age distribution and exposure (IPCS, 1981).
residents living near a copper smelter, Sweden case-control study (Axelson et al., 1978, evaluated in reviews)	arsenic, among others	an overall OR of 4.6 was found for lung cancer mortality among smelters. Using a composite score OR's were 2.1, 5.9 and 8.8 for 'low', 'medium' and 'high' exposures. There was also a significant increased risk of leucemia and myeloma among smelters.	Excess lung cancer did not correlate with exposure to Ni, Pb, Cu, Bi, An, Se or sulphur dioxide.

2 ECOTOXICITY - AQUATIC ORGANISMS

2.1 BIOTRANSFORMATION AND BIOAVAILABILITY

The role of algae in arsenic biochemistry is significant. Arsenate (the predominant form of arsenic in the marine environment) can be taken up by algae because of its similarity to essential phosphate. Several studies indicate that inorganic arsenic is reduced and methylated by phytoplankton. The algal turnover reduces the toxicity of arsenic by producing non-reactive, stable and methylated compounds. Organic arsenic compounds are less toxic to algae and the other organisms to which arsenic can transferred via the foodchain. (Sanders, 1979a, Edmonds and Francesconi, 1987, Lunde, 1972). In higher organisms (invertebrates and fish) arsenic is predominantly present in the organic form. The major compound found in especially marine organisms was arsenobetaîne. Two arsenical compounds [2-hydroxy-3-sulphopropyl-5-deoxy-5-(dimethylarsenoso)furanoside and 2,3-dihydroxypropyl-5-deoxy-5-dimethyl-arsenoso)furanoside] which could be intermediates in the chain from arsenate to arsenobetaîne were identified in the seaweed *Ecklonia radiata* (Edmonds and Francesconi, 1981). The arsenic concentrations of marine macroalgae vary significantly. This is probably due to metabolic differences and not to variations in the total mount of available arsenic in the surrounding water. Considerable quantities of the arsenic in the algae were in the inorganic form (Sanders, 1979a). The seaweeds *Fucus spiralis* and *Ascophyllum nodosum* were found to accumulate more arsenate than arsenite. An increase in temperature resulted in an increased arsenic uptake, whereas variation in salinity or pH had no effects. The uptake of arsenate was reduced by phosphate (Klumpp, 1980a).

The availability of arsenic from estuarine sediments, with total arsenic concentrations ranging from 2 to 2,800 mg $As.kg^{-1}$, to several benthic organisms was studied. In general, the arsenic levels in the deposit feeder *Scrobicularia plana* (bivalve), the polychaete worm *Nereis diversicolor* and the seaweed *Fucus vesiculosus* correlated with the arsenic concentration in the sediment. In the case of *S. plana* the iron/arsenic ratio in the sediment correlated more significant with arsenic levels in the organism than did the arsenic concentration alone. With a decreasing iron concentration in the animals from uncontaminated water (Bryan et al., 1980). Bioconcentration factors (BCF's) were generally higher in organisms of lower trophic levels than in organisms of higher trophic levels. BCF' (based on wet weight) of phytoplankton, invertebrates and fish were reported up to 400, 700 and 150, respectively (Mance et al., 1984).

Laboratory experiments indicated that arsenic is bioconcentrated by aquatic organisms but not biomagnificated. In one experiment stoneflies, snails, amphipods and trout were (separately) exposed to about 100 and 1,000 μg $As.l^{-1}$ from arsenic trioxide, arsenic pentoxide, sodium dimethyl arsenate (SDMA) and disodium methylarsenate (DSMA). BCF's were calculated by dividing the residue tissue concentration (dry weight) by the corresponding exposure concentration. The highest accumulation (BCF=131) in the stonefly *Pteronarys dorsata* was found after 28 days exposure to 100 μg $As(III).l^{-1}$. In the snails *Helisoma campanulata* and *Stagnicola emarginata* BCF's of 99 and 92, respectively, were found after 28 days exposure to 89 μg $As(V).l^{-1}$. A BCF=219 was found in *Daphnia magna* after exposure to 96 μg $As(III).l^{-1}$ for 21 days. The concentration in amphipods and trout were not elevated compared to controls. It can, however, be doubted if the exposure time was long enough to equilibrate the uptake (Spehar et al., 1980). The uptake and accumulation of several arsenic compounds from the water by *Poecilia reticulata* was studied by keeping the fish in labelled solutions of arsenic acid, methylarsonic acid, dimethylarsinic acid, arsenic trioxide, methylarsonous acid, dimethylarsinious acid and trimethylarsine. It was found that only trimethylarsine can be readily taken up by fish. An exact value could not be given because methylarsine is volatilized and oxidized in aqueous solutions and because trimethylarsines are sorpted by the skin of fish (Stary et al., 1982). Marine bivalve molluscs *Argopecten irradians* accumulated arsenic resulting in a BCF of about 10 (based on wet weight) for concentrations up to 3.0 mg $As.l^{-1}$ (just not toxic) (Nelson et al., 1976). Highest concentrations of arsenic in the green sunfish *Lepomis cyanellus* were found in the gallbladder and bile. Levels increased from about 35 to 78 to 159 mg $As.kg^{-1}$ fresh weight during 2.4- and 6-day exposure to 60 mg $As.l^{-1}$ as arsenate (Sorensen et al., 1979). Data on polychaete worms living in sediments of estuarines, indicated that some species can accumulate arsenic to levels around 200 $mg.kg^{-1}$ dry weight, even under low arsenic concentrations. A very remarkable worm is the

Accumulation (and elimination) of arsenic was studied in a foodchain consisting of the macroalga *Fucus spiralis*, the snail *Littorina littoralis* and the predatory snail *Nucella lapillus*. The diet appeared to be the main source of arsenic in this foodchain. The results indicated that arsenic is not biomagnificated. The arsenic taken up was easily eliminated by the organisms. (Klumpp, 1980b). In an experimental food chain radioarsenic was used to identify various chemical forms of arsenic accumulated from food or water. The chain consisted of phytoplankton *Dunaliella marina*, zooplankton *Artemia salina* and a shrimp *Lysmata seticaudata*. In *D. marina* only organic arsenic could be detected after exposure both via water and via food. The arsenic taken up from the water by *L. seticaudata* was largely converted to arsenite, after uptake via food arsenic was mainly found in the organic form. The shrimp contained both organic and inorganic arsenic after exposure by water. It was suggested that arsenic was converted by the intestinal microflora (Wrench et al., 1979). The accumulation and biomagnification of the organic arsenic compounds cacodylic acid (CA) and dimethylarsine (DMA) was studied in a model ecosystem consisting of fish, daphnids, snails and algae. The organisms were exposed for 3, 29, 32 and 32 days, respectively. It appeared that lower food chain organisms (algae and daphnids) bioaccumulated more CA and DMA than did higher food chain organisms (snails and fish) and that both CA and DMA did not biomagnify (Isensee, 1973). It should be noted that in this experiment the exposure time for fish may not have been enough to establish an equilibrium.

2.3 TOXICITY

2.3.1 Freshwater organisms short-term

The relevant short-term toxicity tests with freshwater organisms resulting in L(E)C50-values are summarized in table 2.2.

- Inorganic arsenic

Short-term tests with freshwater organisms have resulted in L(E)C50-values between 206 μg As.l^{-1} for the crustacean *Bosmina longirostris* and 82,500 μg As.l^{-1} for the fish *Pimephales promelas* for inorganic arsenic compounds.

The growth of the alga *Micocoleus vaginatus* was not affected at the highest concentration tested (100,000 µg $As.l^{-1}$) (Vocke et al., 1980). The chronic toxicity of several inorganic (and organic arsenic) compounds was tested in organisms from various taxonomic groups; molluscs, crustaceans, insects and fish. No effects were found at the highest tested concentrations of 1,000 µg $As.l^{-1}$ from arsenic trioxide, arsenic pentoxide or sodium arsenite (Spehar et al., 1980). The parr-smolt transformation appeared to be the most sensitive life stage of the Coho salmon *Oncorhynchus kisutch* . Based on biochemistry and development the NOEC-value was reported to be 76 µg $As.l^{-1}$ from arsenic trioxide (Nichols et al., 1984).

Additional information

The wide range in sensitivity of algae to inorganic arsenic had been found in several other studies as well. A concentration of 75 µg $As.l^{-1}$ as arsenate in the medium significantly inhibited the growth rates of the algae *Melosira granulata* and *Ochromonas vallesiaca* . The alga *Chlamydomonas reinhardtii* required 750 µg $As.l^{-1}$ as arsenate for the same degree of depression, whereas *Cryptomonas erosa* and *Anabaena variabilis* were unaffected up to 7,500 µg $As.l^{-1}$ as arsenate. It was shown that depletion of phosphate increased the toxicity of arsenate (Planas and Healey, 1978). The primary productivity of *Skeletonema costatum* was inhibited by arsenate at 67 nM when the concentration of phosphate was kept low. A phosphate enrichment > 0.3 µM decreased the toxicity of arsenate. Dimethylarsinic acid (organic arsenic) was less toxic than the inorganic compounds (Sanders, 1979b). The effect of arsenic trioxide on the ovarian function and spermatogenesis of the freshwater fish *Colisa fasciatus* was studied. Exposure to 1.5 mg $As.l^{-1}$ for 15 and 30 days and exposure to 10.6 mg $As.l^{-1}$ for 15 days showed no effect on the development of oocytes, whereas exposure to 10.6 mg $As.l^{-1}$ for 30 days decreased the development of oocytes. With regard to spermatogenesis similar results were obtained, with the same test concentrations and exposure times. Only after 30 days exposure to 10.6 $As.l^{-1}$ an effect was seen (Shukla and Pandey, 1984a, 1984b). Arsenic had a significant effect on the temperature tolerance and survival of newly hatched fry *Esox masquinongy* .Fry in the swim-up phase (8-14 days post hatch) were exposed to 0-5.0 mg $As.l^{-1}$ as arsenite. In all tests, including the control, a significant decline in the Critical Thermal Maxima (CTM) occurred during this period. The control group

As.l^{-1} from disodiummethylarsenate and sodium dimethylarsenate (Spehar et al., 1980). The toxicity of several dietary arsenic compounds in juvenile *S. gairdneri* was tested. Even the highest doses organic arsenic did not cause any effects. Dimethylarsinic acid and arsanilic acid provided NOEC-values of ≥1497 and ≥1503 mg As.kg^{-1} diet, respectively (Cockell and Hilton, 1988).

2.3.3 Freshwater sediment systems

The presence of sediments significantly decreased the toxicity of arsenite to *Daphnia magna*. In two series consecutive tests with several arsenite concentrations a difference was seen in the 48-h survival of neonate *D. magna* (< 24-h in age) tested in beakers with or without sediment. Survival in the beakers with sediment increased over time. Arsenite in the water column apparently became adsorbed to sediment through time, becoming less available to *D. magna*. For example, 47 mg As.l^{-1} as arsenite caused 100% mortality in repetitive tests until day 20. This percentage decreases to 37% at day 28. Alkaline phosphatase, a principle enzym released by *D. magna*, was shown to decrease in activity (Burton et al., 1987).

2.3.4. Marine organisms short-term

Data on relevant short-term toxicity tests with marine organisms resulting in L(E)C50-values are presented in table 2.4.

- Inorganic arsenic

The short-term tests with marine organisms have resulted in L(E)C50-values between 232 μg As.l^{-1} (48-h) for larval stages of the crustacean *Cancer magister* to 28,500 μg As.l^{-1} (96-h) for the adult fish *Limanda limanda* . For adult crustaceans a 96-h LC50-value of 17,000 μg As.l^{-1} was reported.

Additional information

The effects of arsenate and arsenite on the filtering rate of the mussel *Perna perna* was examined after one hour of exposure. A difference in toxicity between tri- and pentavalent arsenic was found. Sodium arsenate did not affect the filtering rate significantly at levels up to 150 mg As.l^{-1}, whereas a

Additional information

The toxicity of arsenic to marine algae has been studied several times. Marine algae show, just like freshwater algae, wide ranges of sensitivity to arsenic. Single species tests were conducted to study the effect of up to 100 µg $As.l^{-1}$ as arsenate on the growth of seven species of marine phytoplankton. *Cylindrotheca closterium* was most resistance (NOEC $\geq$ 100 µg $As.l^{-1}$). *Thalassiosira pseudonana* and *Tetraselmis contracta* were slightly affected (growth depression 27 and 19%, respectively). *Skeletonema costatum* was greatly affected (growth depression 60%). The other species *Isochrysis galbana*, *Amphidinium carterae* and *chaetoceros pseudocrinitum* were most affected; their growth was terminated by arsenate (Sanders and Vermersch, 1982). An arsenate concentration of 75 µg is inhibitory to the growth of the green alga *Chlamydomonas* (Christensen and Zielski, 1980).

In a number of studies it was found that phosphate enrichment reduced the uptake of arsenate by algae. Under low phosphate conditions the growth of algae was affected at lower doses. Studies indicated that both arsenite and arsenate inhibit cellular functions at low levels, but in different ways. Arsenite is thought to react with SH-groups of proteins. Arsenate competes with phosphate for transport into the cell. It inactivates the phosphate active transport mechanism and may inhibit glucose metabolism. In addition it competes with phosphate in the oxidative phosphorylation. Therefore the extracellular concentration of phosphate may be important in the toxic effect of arsenate (Sanders, 1979b). The influence of phosphate on the toxicity of arsenate and arsenite on the macroalga *Champia parvula* was also investigated in the following study. Phosphate had no effect on the toxicity of arsenite. At a concentration of 212 µg $As.l^{-1}$ the growth of *C. parvula* was significantly less than that for the control, but the effect was independent of phosphate concentrations. The toxicity of arsenate was inhibited by phosphate. When no phosphate was added all plants died at 1,290-9,330 µg $As(V).l^{-1}$, whereas with an addition of 4,5 µM phosphate the growth in all treatments was equal to that in the control (Thursby and Steele, 1984).

The effect of arsenate on the growth rate of a natural phytoplankton assemblage was tested at concentrations of 5, 15 and 25 µg $As.l^{-1}$. The NOEC-value based on growth was <5 µg $As.l^{-1}$ after 72 h. The species composition was

From sediments relatively little arsenic is taken up into biota. The iron/arsenic ratio in sediments correlated more significantly with the arsenic levels in the organisms than did the arsenic concentration in the sediment alone.

- Bioconcentration/biomagnification

Arsenic is a natural component of aquatic systems and is bioaccumulated in small to moderate amounts. In organisms of lower trophic levels higher bioconcentration factors (BCF = concentration in organisms/ concentration in water) are found than in organisms of higher trophic levels. BCFs up to 400, 700 and 150 are reported for algae, invertebrates and fish, respectively. In one study the BCF was found to increase with a decreasing concentration of arsenic in the water. There are indications that the organic arsenic in higher trophic levels are taken up from by food. Biomagnification does not seem to occur.

- Toxicity

Freshwater organisms

Short-term toxicity tests with inorganic arsenic compounds have resulted in L(E)C50-values ranging from 206 μg $As.l^{-1}$ for the crustacean *Bosmina longirostris* to 82,500 μg $As.l^{-1}$ for the fish *Pimephales promelas*. The relevant long-term toxicity tests with freshwater organisms have resulted in NOE(L)C-values between 10 and 10,000 μg $As.l^{-1}$. The lowest and highest NOEC-values were found for the algae *Ankistrodesmus falcatus* and *Selenastrum capricornutum* in the same study, using identical testconditions. This wide variance in sensitivity of algae to arsenic has been found in several other studies as well. The toxicity of arsenate to algae decreases in the presence of phosphate, whereas the toxicity of arsenite seems to be independent of phosphate. Chronic exposure to about 1,000 μg $As.l^{-1}$ as arsenic trioxide, arsenic pentoxide, sodium arsenite, disodium methylarsenate and sodium dimethylarsenate did not cause toxic effects in molluscs, crustaceans, insects and fish. In the presence of sediment the acute toxicity of arsenite to *Daphnia magna* decreased.

Table 2.2. Short-term "single species" toxicity tests - freshwater organisms

Organism	A	Test-type	Test-subst.	Test-water.	pH	Hardness	Exp.-time	Criterion	Result $\mu g\ As.l^{-1}$	Reference
Inorganic arsenic compounds										
Aschelminths										
Philodina roseola		S	$NaAsO_4$ x $7H_2O$	n.m.	-	-	96-h	LC50	**13,000**	[1] Schaefer & Pipes '73
Molluscs										
Aplexa hypnorum	-	S	As_2O_3	lake	7.4/ 7.7	56	96-h	LC50	**18,560**	Holcombe et al. '83
Crustaceans										
Bosmina longirostris (24-h)	+	S	Na_2HAsO_4	s.w.w.	6.8	120	96-h	$EC50_i$	**206**	[2] Passino & Novak '84
Ceriodaphnia dubia < 24-h old	+	R	Na_2AsO_4 x $7H_2O$	r.h.w.	8.1	165	48-h	LC50	**1,450**	Spehar & Fiandt '86
Daphnia magna	-	S	$NaAsO_2$	tap	8.1	40	26-h	$EC50_i$	3,000	Crosby & Tucker '66
Daphnia magna	-	R	Na_3AsO_4	lake	7.7	45	48-h	LC50	**7,400**	Biesinger & Christensen '72
Daphnia magna	+	S	$NaAsO_2$	a.w.w.	7.2/ 8.1	48	48-h	$EC50_i$	**1,500**	[3] Lima et al. '84
Daphnia pulex (24-h)	+	S	Na_2HAsO_4	s.w.w.	6.8	120	48-h	$EC50_i$	**12,040**	Passino & Novak '84
Gammarus pseudolimnaeus	+	CF	$NaAsO_2$	a.w.w.	7.2/ 8.1	48	96-h	$EC50_i$	**874**	Lima et al. '84
Insects										
Tanytarsus dissimilis	-	S	As_2O_3	lake	7.2/ 7.7	37	48-h	LC50	**73,485**	Holcombe et al. '83
Fish										
Channa punctatus fingerlings	-	S	As_2O_3	tap	7.2	124	96-h	LC50	**10,830**	[4] Shukla et al. '87
Colisa fasciatus fingerlings	-	S	As_2O_3	tap	7.1	-	96-h	LC50	**6,100**	[4] Pandey & Shukla '82
Coregonus hoyi fry 15-19 d	+	S	As_2O_3	r.s.w	-	40-48	96-h	LC50	19,700	[5] Passino & Kramer '80
Coregonus hoyi fry 22-d	+	S	As_2O_3	r.s.w.	-	40-48	96-h	LC50	**12,900**	Passino & Kramer '80
Esox masquinongy fry swim-up stage	+	S	$NaAsO_2$	tap	7.2/ 7.7	-	96-h	LC50	**1,100**	Spotila & Paladino '79
Esox masquinongy 5-w old	+	S	$NaAsO_2$	tap	7.2/ 7.7	-	96-h	LC50	**16,000**	Spotila & Paladino '79
Jordanella floridae fry	+	CF	$NaAsO_2$	a.w.w	7.2/ 8.1	48	96-h	LC50	**14,400**	[6] Lima et al. '84
Lepomis cyanellus	-	S	Na_3AsO_4	well	-	-	48-h	LC50	**54,000**	Sorensen '76
Pimephales promelas	+	S	As_2S_3	r.w	7.2/ 7.9	40-48	96-h	LC50	82,400	[7] Curtis et al. '79
Pimephales promelas	+	CF	$NaAsO_2$	a.w.w.	7.2/ 8.1	48	96-h	LC50	**14,100**	[8] Lima et al. '84
Pimephales promelas 30-d old	+	CF	Na_2AsO_4 x $7H_2O$	l.s.w.	7.4	44	96-h	LC50	**12,600**	Spehar & Fiandt '86
Organic arsenic compounds										
Ictalurus lacustris	-	S	MSMA	tap	-	-	96-h	LC50	1,412	Anderson et al. '75
Micropterus dolomieu fingerlings	-	S	MSMA	tap	-	-	96-h	LC50	417	Anderson et al. '75
Procumbaru spp.	-	S	MSMA	tap	-	-	96-h	LC50	509	Anderson et al. '75

Table 2.3. Long-term "single species" toxicity tests with As - freshwater organisms

Organism	A	Test-type	Test-subst.	Test-water.	pH	Hardness	Exp.-time	Criterion	Result $\mu g\ As.l^{-1}$	Reference
Inorganic arsenic compounds										
Bacteria										
Pseudomonas putida		S	$Na_2HAsO_4 \times 7\ H_2O$	n.m	-	-	16-h	$NOEC_g$	**4,860**	[9] Bringmann & Kuhn '80
Algae										
Ankistrodesmus falcatus		S	Na_2HAsO_4	n.m.	7.0	-	14-d	$NOEC_{g[c]}$	**10**	[10] Vocke et al. '80
Asterionella formosa	+	CF	AsO_4	s.l.w.	8.2	-	24-d	$NOEC_g$	**86**	[11] Conway '78
Micocoleus vaginatus		S	Na_2HAsO_4	n.m.	7.0	-	14-d	$NOEC_{g[c]}$	>100,000	[12] Vocke et al. '80
Scenedesmus obliquus		S	Na_2HAsO_4	n.m.	7.0	-	14-d	$NOEC_{g[c]}$	<10	Vocke et al. '80
Scenedesmus quadricauda		S	Na_2HAsO_4	n.m.	-	-	7-d	$NOEC_g$	**2,350**	[9] Bringmann & Kuhn '80
Selenastrum capricornutum		S	Na_2HAsO_4	n.m.	7.0	-	14-d	$NOEC_{g[c]}$	**10,000**	Vocke et al. '80
Protozoa										
Entosiphon sulcatum	-	S	Na_2HAsO_4	n.m.	-	-	72-h	$NOEC_g$	**2,400**	[9] Bringmann & Kuhn '80
Macrophytes										
Lemna paucicostata (6746)	-	S	As_2O_3	n.m.	4-7	-	7-d	$NOEC_g$	<1,000	[13] Nasu & Kugimoto '81
Molluscs										
Helisoma campanulata and Stagnicola emarginata	+	CF	As_2O_3	lake	±7.0	42-45	28-d	NOLC	≥961	[14] Spehar et al. '80
Helisoma campanulata and Stagnicola emarginata	+	CF	As_2O_5	lake	±7.0	42-45	28-d	NOLC	≥973	Spehar et al. '80
Crustaceans										
Cerodaphnia dubia < 24-d old	+	CF	$Na_2AsO_4 \times 7H_2O$	l.r.w.	8.2	100	7-d	$NOEC_{r,m,g}$	<1,140	Spehar & Fiandt '86
Cyclops & Diaptomus spp. naplii	-	S	$NaAsO_2$	n.m.	7.6/ 8.8	139	14-d	NOLC	920	Borgmann et al. '80
Daphnia magna	+	S	As_2O_3	lake	±7.0	42-45	14-d	$NOEC_{m,r}$	≥955	Spehar et al. '80
Daphnia magna	+	S	As_2O_5	lake	±7.0	42-45	14-d	$NOEC_{m,r}$	≥931	Spehar et al. '80
Daphnia magna — P≤1d -> F[lc]	-	R	$NaAsO_4$	lake	7.7	45	3-wk	$NOEC_{r,g,b}$	**520**	[15] Biesinger & Christensen '72
Daphnia magna <24-h old	+	R	$NaAsO_2$	a.w.w.	7.2/ 8.1	48	28-d	$NOEC_{r,g}$	**633**	Lima et al. '84
Gammarus pseudolimnaeus	+	CF	As_2O_3	lake	±7.0	42-45	14-d	NOLC	≥88	[16] Spehar et al. '80
Insects										
Pteronarcys dorsata larvae	+	CF	As_2O_3	lake	±7.0	42-45	28-d	NOLC	≥961	Spehar et al. '80
Pteronarcys dorsata larvae	+	CF	As_2O_5	lake	±7.0	42-45	28-d	NOLC	≥973	Spehar et al. '80
Fish										
Jordanella floridae egg/fry	+	CF	$NaAsO_2$	a.w.w.	7.2/ 8.1	48	31-d	$NOEC_{h,m,g}$	**2,130**	[17] Lima et al. '84
Oncorhynchus kisutch parr-smolt transformation	-	CF	As_2O_3	n.m	8.2	69	6-m	$NOEC_{b,d}$	**76**	[18] Nichols et al. '84
Pimephales promelas egg/fry	+	CF	$NaAsO_2$	a.w.w.	7.2- 8.1	48	29-d	$NOEC_{h,m,g}$	**2,130**	[17] Lima et al. '84
Pimehales promelas	+	CF	$Na_2AsO_4 \times 7H_2O$	l.s.w.	7.4	44	32-d	$NOEC_{g,r,m}$	<3,330	Spehar & Fiandt '86

Table 2.4. Short-term "single species" toxicity tests - marine organisms

Organism	A	Test-type	Test-subst.	Remark medium	Salinity o/oo	Exp.-time	Criterion	Result $\mu g\ As.l^{-1}$	Reference
Inorganic arsenic compounds									
Macroalgae									
Plumaria elegans sporelings	-	S	$NaAsO_2$	s.w	-	18-h	LC50	>580	[20] Boney et al. '59
Molluscs									
Crassostrea gigas embryo	+	S	As_2O_3	n.s.w.	34	48-h	$EC50_d$	**326**	Martin et al. '81
Crassostrea virginica embryo	-	S	$NaAsO_2$	s.s.w.	25	48-h	LC50	**7,500**	Calabrese et al. '73
Mytilus edulis embryo	+	S	As_2O_3	n.s.w.	34	48-h	$EC50_d$	**3,000**	Martin et al. '81
Crustaceans									
Argopecten irradians	-	R	$NaAsO_2$	n.s.w.	25	96-h	LC50	**3,490**	Nelson et al. '76
Cancer magister zoea I stage larvae	+	S	As_2O_3	n.s.w.	34	48-h	$EC50_d$	**232**	Martin et al. '81
Scylla serrata		S	As_2O_3	s.s.w.	-	96-h	LC50	**17,000**	Krishnaja et al. '87
Fish									
Chelon labrosus	+	CF	As_2O_3	d.s.w.	34.5	96-h	LC50	**27,300**	Taylor et al. '85
Limanda limanda	+	CF	As_2O_3	d.s.w.	34.5	96-h	LC50	**28,500**	Taylor et al. '85

d = development

n.m. = nutrient medium
d.s.w. = diluted seawater
s.s.w. = synthetic seawater

List of abbreviations on p. 55

[20] at 577 $\mu g.l^{-1}$ 0% of the sporelings died.

3 ECOTOXICITY - TERRESTRIAL ORGANISMS

3.1 ACCUMULATION

The correlation between arsenic concentration in earthworm species and that in soil from 20 different, contaminated and uncontaminated, sites was determined. The arsenic concentrations ranged from 0.77 to 3.5 mg $As.kg^{-1}$ dry weight (dw) in the uncontaminated soils and from 1.4 to 33 mg $As.kg^{-1}$ dw in the contaminated soils. In general, arsenic levels in the earthworms from the uncontaminated soils were not elevated compared to those from contaminated soils. Earthworms collected from uncontaminated sites contained trace amount to 1.5 mg $As.kg^{-1}$ dw. The concentrations in earthworms from contaminated soils ranged from trace amounts to 0.81 mg $As.kg^{-1}$ dw. The correlation coefficient between As levels in worms and those in soils was found to be +0.45 ($p<0.05$). This value could have been strongly influenced by one high value; *Pheretima* sp. contained arsenic levels of 10 mg $As.kg^{-1}$ dw with a corresponding soil level of 20 mg $As.kg^{-1}$ dw. Only a third of the variation in the arsenic concentration in earthworms could be explained by the concentrations in the soil (Beyer and Cromartie, 1985).

3.2 TOXICITY

- Microbe-mediated processes

Soil acidification seemed to have an effect on the availability of metals and on the biological activity of the soil. A combination of a low pH with a heavy metal pollution increased the toxicity of the heavy metals, as measured by decomposition and mineralisation processes (Tyler, 1983).
The effect of several heavy metals (Pb, As, Cd and Cu) on micro-organisms in soils (not specified) near a secundary lead smelter was studied. Analyses showed that with a decreasing concentration of the heavy metals in the soil (samples to a depth of 10 cm) the population counts of bacteria, actinomyces, fungi, nematodes and earthworms increased. The arsenic concentration in the soil decreased from 972 to 163 mg $As.kg^{-1}$ at an increasing distance of 15 to 180 m from the smelter. The correlation

In one study the influence of spray programs on beneficial anthropods of apple orchards has been investigated. The sensitivity to lead arsenate varied widely among different predator and parasite species. Exposition in this study was variable for the different species (Macphee and Sanford, 1954).

In a field experiment the arsenic content of vegetables was examined 16 years after application of lead arsenate. From 1949 to 1953 lead arsenate was added to a sandy loam soil. In 1969 an arsenic concentration of 275.5 kg $As.ha^{-1}$ dm was measured (0-15 cm depth). Assuming a specific gravity of 1.5 $kg.l^{-1}$ soil, this corresponds to a concentration of 122 mg $As.kg^{-1}$ dm. The number of mites and collembolans were counted in soil samples of 0-12.7 cm depth. The number of collembolans was found to be greater in the treated soil than in the control soil. The number of mites was not changed (Chisholm and Macphee, 1972).

Summary and conclusions "terrestrial organisms"

- Accumulation

With regard to the accumulation of arsenic the data are limited to one study on earthworms. This study indicated that arsenic levels in several earthworm species from contaminated soils were not elevated compared to those in earthworms from uncontaminated soils. Based on these data the accumulation of arsenic in earthworms does not seem to be a problem.

- Toxicity

Data on the toxicity of arsenic to terrestrial organisms or processes are limited and mostly concern microbe-mediated processes. In one study the influence of As(III) and As(V) on the number of micro-organisms and on the activity of microbe-mediated processes was investigated in a sandy and in an alluvial soil. At the lowest concentrations tested (500 mg $As(III).kg^{-1}$ from arsenic trioxide and 1,000 mg $As(V).kg^{-1}$ from arsenate) effects were still seen. Therefore the NOEC-values for As(III) and As(V) were below 500 and 1,000 mg $As.kg^{-1}$ dm, respectively. Round a smelter the populations of

4 TOXICITY TO AGRICULTURAL CROPS AND LIVESTOCK

4.1 AGRICULTURAL CROPS

4.1.1 Accumulation

Arsenic levels in cereals, potatoes, vegetables, fruits, and the major fodder crops (grass, silage maize, and crowns and leaves of sugar beet) grown in The Netherlands and the arsenic concentration in the corresponding "normal" soils were measured. Levels in cereals (0.045-0.189 mg $As.kg^{-1}$ fw), especially oats (0.189 mg $As.kg^{-1}$ fw), were high compared with those of the other crops. Arsenic concentration (in mg $As.kg^{-1}$ fw) were 0.013 in potatoes, 0.001-0.022 in vegetables and 0.004-0.014 in apples. The (mean) arsenic contents of grass, silage maize and (the crowns and leaves of) sugar beet were found to be 0.28, 0.17, and 0.51 mg $As.kg^{-1}$ dm, respectively. The corresponding soils contained 12, 10, and 14 mg $As.kg^{-1}$ dm, respectively (Wiersma et al., 1986). Arsenic levels in potatoes grown under "normal" conditions and in potatoes grown on (soils mixed with) municipal waste compost (MWC), sewage sludge or dregded materials from contaminated harbours were studied. Based on own experiments and data from literature potatoe (tuber) levels of 0.013 and 0.10 mg $As.kg^{-1}$ fresh weight (fw) were considered normal and (too) high, respectively. A maximum permissible level for arsenic in potatoes of 0.10 mg $As.kg^{-1}$ fw was proposed (see Criteria Document). Potatoes grown on a light sandy soil and a heavy fluvial clay soil containing 2 and 12 mg $As.kg^{-1}$ dry matter (dm), respectively, contained about 0.004 mg $As.kg^{-1}$ fw. Potatoes grown on mixtures of these soils with MWC in a soil/MWC ratio of 90/10 or on 100% MWC (containing 4 mg $As.kg^{-1}$ dm) did not result in higher levels in the tubers. Arsenic levels in potatoe tubers grown on mixtures of the sandy and clay soil (with 2 and 12 mg $As.kg^{-1}$ dm, respectively) with sewage sludge in a ratio soil/sludge: 90/10 (by volume) or on 100% sewage sludge (46 mg $As.kg^{-1}$ dm) were 0.012 and 0.004 mg $As.kg^{-1}$ fw for mixtures with the sandy and the clay soil, respectively, and 0.014 mg $As.kg^{-1}$ fw for the sewage sludge. In potatoe tubers grown on sewage sludges from different sites (6-46 mg $As.kg^{-1}$ dm) levels stayed below the (proposed maximum level of) 0.10 mg $As.kg^{-1}$ fw. In an experiment using the same sandy soil mixed with (another) liquid sewage sludge (5 annual contained concentrations up to 3.88 mg $As.kg^{-1}$ dm (mean 0.85). A positive relationship was found between arsenic levels of the edible parts of beetroot, lettuce, onion and pea and both extractable and total arsenic in soil. Arsenic in carrots was not significantly related to soil content. In the case of lettuce soil iron was demonstrated to reduce arsenic uptake by giving relatively low solubility products and by adsorption of arsenic to iron hydroxides). The effect of phosphorus (less arsenic taken up) may be due to the fact that phosphorus is preferred to arsenate by the phosphate uptake system. In spite of the large arsenic amounts in these soils all the vegetables examined were below the level of 1 $mg.kg^{-1}$ fw. The authors conclude that plants act as 'geochemical barriers' in the environment and are only making a small contribution to man's exposure (Xu and Thornton, 1985).

To evaluate the effect of residual arsenic in soils on the yield, quality, and arsenic uptake by vegetable crops a field trial was carried out on Plainfield sand. The caracteristics of the soil used were; 0.7% carbon; 4% silt; 7% clay; pH 5.5 and 4.0 meq.100 g^{-1} cation exchange capacity. Sodium arsenite was added to the soil at amounts of 0, 45, 90, 180 and 720 $kg.ha^{-1}$. This equals concentrations of 0, 20, 40, 80 and 320 mg $As.kg^{-1}$ dm (assuming s.g.=1.5 $kg.l^{-1}$ soil). Arsenic was below detection limits (<0.02 mg $As.kg^{-1}$ fw) in the edible portions of peas and sweet corns. Levels of the above-ground portions of potatoes showed no correlation with the arsenic content in the soil. External contamination by wind blown sand was probably the principal source of contamination for the above-ground portions. The concentrations in potatoe tubers and in snap beans increased with increasing concentrations in the soil. Snap bean (seeds and pods) contained up to 1.5 mg $As.kg^{-1}$ fw. In potatoe tubers the peelings contained up to 83 mg $As.kg^{-1}$ fw, whereas the peeled potatoe tubers contained only (maximal) 0.5 mg $As.kg^{-1}$ fw (Jacobs et al., 1970).

Data on wild plants

In one study the uptake of arsenic by vegetation of the former Rhine estuary, containing 10 to 200 mg $As.kg^{-1}$ dm, was studied. The roots of *Phragmites australis* and *Urtica dioica* contained 0.08-20 and 0.1-4.7 mg $As.kg^{-1}$ dw, respectively. The root/soil accumulation factor was calculated to be 0.006-0.23 and 0.008-0.12 for *P. australis* and *U. dioica* , of 12.2 mg $As.kg^{-1}$ had no significant effect on crop yields during the period covered by this study (1959-1969). In one year (1966) a detrimental effect on beans was found but this was not repeated (Chisholm and Macphee, 1972). In a field study (mentioned in 4.1.1) the arsenic residue toxicity to vegetable crops was investigated. Sodium arsenite was added to the soil up to 320 mg $As.kg^{-1}$. Yields from the 80 and 320 mg $As.kg^{-1}$ treatments were significantly lower than check yields. The yields of potatoes and peas decreased lineair with increasing arsenic concentration in the soil. A small increase in yield was observed at the lowest concentration (20 mg $As.kg^{-1}$), which could be due to a greater availability of phosphate in the soil. The data indicate that with this soil marked yield reductions of peas, snap beans and sweet corn occur at 1 and 10 $mg.kg^{-1}$ NH_4OAc or Bray P-1 extractable (0.025 N HCl; 0.03 N H_4F) arsenic-values, respectively (Jacobs et al., 1970).

Data on wild plants

Among plants differences in sensitivity to arsenic have been observed. One study provided some evidence that arsenic tolerance in the plant *Holcus lanatus* is caused by an altered phosphate uptake system. The tolerance of six clones of *H. lanatus*, measured as root lenght, was determined. The differences in root lenght between tolerant, partially tolerant and non-tolerant were all statistically significant. At low levels of arsenate the high phosphate treatment reduced toxicity of arsenate in non-tolerance but not in tolerant ones. Tolerant plants took up less arsenate than nontolerant ones. The effect of phosphate on the toxicity of arsenate was only clear in nontolerant plants. The results suggest that arsenate is taken up by the phosphate uptake system in nontolerant plants, and that in tolerant plants this system had changed (Macnair and Cumbes, 1987).

In an outdoor experiment two plants *Phragmites autralis* and *Urtica dioica*, were grown on soil (80% garden soil, 20% sand) artificially contaminated with 0, 1, 5 or 30 mg $As.kg^{-1}$ dm as lead arsenate or sodium cacodylate $[(CH_3)_2AsO(ONa)]$. The experiment with *P. australis* lasted two months, that with *U. dioica* one month. The growth of *U. dioica* was significantly inhibited at 30 mg $As.kg^{-1}$ dw from both compounds. The growth of *P. australis* was not affected significantly. At added concentrations of 5 mg $As.kg^{-1}$ dm from both compounds no effects were seen. The difference could be

In a field experiment with the wild plant *Urtica dioica* a significant growth inhibition was found at an added concentration of 30 mg $As.kg^{-1}$ dw as lead arsenate or sodium cacodylate $[(CH_3)_2AsO(ONa)]$. The soil used consisted of 80% garden soil and 20% sand. The backgroundconcentration of arsenic of this soil was not given. At added concentrations of 5 mg $As.kg^{-1}$ dw from both compounds no effects were seen.
Among plants some differences with regard to sensitivity to arsenic were observed. One study with a wild plant indicated that the phosphate uptake system of "tolerant" plants is altered (arsenate can no longer be taken up by this system).
In these experiments the highest concentration without effect, taking into account the backgroundconcentration of arsenic in the soil used, was circa 20 mg $As.kg^{-1}$ dw. The lowest concentration with effect was reported to be 30 mg $As.kg^{-1}$ (exclusive of the backgroundconcentration).

4.2 LIVESTOCK

4.2.1 Accumulation

Experiments were carried out to study the transfer of arsenic from feed into muscle, brain, liver and kidney tissues of fattening lambs. Ten week old lambs were fed arsenic as a soluble compound or such as present in sewage or harbour sludge for 3 months. Part of the lambs (32 out of 48) were kept indoors. The diets contained 1.6 and 2.6 mg $As.kg^{-1}$ dry matter (dm) from arsenic trioxide (for lambs kept indoors and outdoors, respectively), 2.5 mg $As.kg^{-1}$ dm from harbour sludge and 0.9 mg $As.kg^{-1}$ dm from sewage sludge. Control diets contained 0.3 mg $As.kg^{-1}$ dm. Arsenic levels in the various tissues were analysed after three months. Only the soluble arsenic compound and the arsenic from harbour sludge appeared to give some accumulation in muscle, liver and kidney tissues of the animals kept indoors. The tissues contained about 13, 24 and 50 μg $As.kg^{-1}$ fw, compared to 2, 2 and 5 μg $As.kg^{-1}$ fw in tissues of controls. For liver and kidney tissues a dose-dependent accumulation was found. No accumulation was found the brain (only one sample from each group) (Van der Veen and Vreman, 1986). A similar study was carried out to examine the transfer of arsenic

The organic arsenic compounds which have been used as growth promotors, for example arsanilic acid, seem to be less toxic than inorganic arsenic (NRC, 1980).

A group of nearly 6,000 cattle fed for 1-2 days with a mixture containing arsenic trioxide at concentrations between 490 and 2,900 mg $As.kg^{-1}$ was studied. Of this group 1,464 animals died (50% within one week). Acute symptoms were drastic reduction in milk (85%), diarrhoea, dyspnoea, cyanosis, abortion and effects on the nervous system. Chronic exposure resulted in effects on skin and joints, blindness and pathological changes in internal organs (IPCS, 1981). Two adult cows died after ingestion of about 20 g of wood ashes, which contained high amounts of arsenic (780 mg $As.kg^{-1}$ wet weight), chromium and copper (Thatcher, 1985).

Sheep fed lakeweed with a daily dosis of 1.4 mg $As.kg^{-1}$ bw for 3 weeks did not show any adverse effects (IPSC, 1981). Horses and cattle could ingest arsenic up to concentrations of 2.66 and 4 mg $As.kg^{-1}$ bw daily, respectively, without any signs of toxicity. Lead arsenate at a level of 4.68 mg $As.kg^{-1}$ bw in cattle did not result in adverse effects. Potassium arsenite and arsanilic acid fed to sheep for 56 days at concentrations up to 285 mg $As.kg^{-1}$ diet (22.8 mg $As.kg^{-1}$ bw) produced no toxic effects, whereas a concentration of 570 mg $As.kg^{-1}$ diet (45.6 mg $As.kg^{-1}$ bw) resulted in effects as convulsions, weight loss and decreased feed consumption . Arsonic acid fed to sheep for 56 days in concentrations of 1,139 mg $As.kg^{-1}$ diet (91 mg $As.kg^{-1}$ bw) gave no toxic effects. In chickens a dose of 10 mg $As.kg^{-1}$ diet (1.8 mg $As.kg^{-1}$ bw) from arsenic(V)oxide for 56 days did not cause adverse effects. At 100 mg $As.kg^{-1}$ diet (18 mg $As.kg^{-1}$ bw) a decrease in body weight, feed intake and egg production was seen (NRC, 1980). Administration of 350 mg $As.kg^{-1}$ as arsanilic acid to pigs caused various toxic effects (IPCS, 1981). Administration of 1,000 mg $As.kg^{-1}$ diet to swines produced toxic effects after 18 days (NRC, 1980).

Data on chronic toxicity could not be found, but chronic administration of low arsenic levels seemed to have a positive effect on the hair of cattle. After withdrawal of the arsenic this improvement was lost, the animals lost weight and had inflamed eyes. The growth promotant level of arsanilic acid for swines is 100 mg $As.kg^{-1}$ diet (NRC, 1980).

5 RISK ASSESSMENT

5.1 RISK ASSESSMENT FOR MAN

Arsenic is considered to be a non essential element, and is therefore a contaminant.

Epidemiological studies have demonstrated positive dose-response relationships between exposure to inorganic arsenic compounds and cancer risk. Long-term oral intake of inorganic arsenic (through drinking water or medication) has been associated with an increased skin cancer risk. An increased risk on cancer of the respiratory tract has been found in many studies among populations exposed by inhalation. Although in most of these studies simultaneously exposure to other substances occurred, so it can not be excluded that the effects were due to other factors than arsenic exposure, it is concluded that inorganic arsenic can cause tumours in humans.

From experimental studies it appeared that inorganic arsenic is not carcinogenic in animals as far as exposure routes relavant for humans are concerned. A mechanistic study showed that both tri- and pentavalent arsenic have tumorpromoting activities.

Arsenic has an unusual genotoxic profile; in *in vitro* tests a clear induction of chromosome aberrations was observed, but not of gene mutations. Other mechanisms than direct DNA damage could be involved.

From metabolism studies it appeared that in both animals and humans inorganic arsenic is methylated to organic arsenic compounds; this process mainly takes place in the liver. At higher exposure levels this metabolism becomes saturated and this results in higher blood arsenic levels. Epidemiological studies demonstrated that the lung cancer risk correlated stronger with peak exposure than with cumulative exposure.

On the basis of all results (epidemiological studies, experimental studies, genotoxicity tests, metabolism studies) it is concluded that, with regard to the carcinogenic effect of arsenic, there are insufficient arguments to use a "non-threshold" extrapolation method for risk assessment.

of 70 kg* this corresponds with an intake of 2,0 μ As.kg^{-1} bw a day (JECFA, 1983).

In the present document the point of view of the WHO is adopted. However, it must be emphasized that an intake of 2,0 μg As.kg^{-1} bw per day may not be considered as an "acceptable daily intake" (as defined by the WHO) because effects can not be ruled out after long-term daily intake of this amount.

The WHO did not establish a limit value for organic arsenic compounds. Assuming that these compounds are at least a factor 10 less toxic than the inorganic ones, it is concluded that effects are unlikely after long-term daily intake of 20 μg As.kg^{-1}bw.

Using a "non-threshold" extrapolation and the data from the study of Tseng (1977) the "unit risk" [the lifetime risk due to 1 μg As.kg^{-1}bw of arsenic intake from water] was estimated to be circa 1,5 x 10^{-3} (1 and 2 x 10^{-3} for males and female, respectively) (EPA, 1987).

Assuming an acceptable risk of one extra case of skin cancer per million persons exposed lifetime, this risk corresponds with a daily intake of 6,7 x 10^{-4} μg As.kg^{-1} bw.

- <u>Inhalatory exposure</u>

On the basis of the occupational studies it was not possible to establish a usable dose-response relationship. In a number of studies "threshold values" of 90 or 500 μg As.m^{-3} are described. Exposure to peak concentrations also appeared to be of importance.

Toxic effects described among occupationally exposed persons include effects on peripheral bloodvessels and nervous system; slight toxic effects occurred at a concentration of about 50 μg As.m^{-3} (Lagerkvist et al., 1986, Blom et al., 1985). In these two studies it was, however, not possible to determine if the effects had to be described to present or past exposure, when it was significantly higher; up to 500 μg as.m^{-3}.

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* The RIVM normally assumes a daily consumption of 1,5 l water and a mean body weight of 60 kg; this corresponds then to 2,5 μg As.kg^{-1} bw.

long-term NOEC-values. In this method all available data of a kind are used, for example all long-term NOEC-values. Using short-term L(E)C50-values or long-term L(E)C50-values, a HCS ("hazardous concentration for sensitive species") is calculated; at these concentrations there is a probability (for example 10%, arbitrary) that up to 50% of individuals of the most sensitive species will die at short-term and long-term exposure, respectively. At the HCS calculated from long-term NOEC-values, there is a probability that adverse non-lethal effects will occur in up to 50% of individuals of the most sensitive species.

The method according to van Straalen (1987) is similar to that according to Kooijman. In the method of van Straalen all long-term NOEC-values are used, resulting in an HC5 ("hazardous concentration for 5% of the species"). At the HC5 there is a probability of 5% that adverse non-lethal effects will occur in up to 5% of the species exposed. The values of 5% also are arbitrary chosen. The HC5 is considered to be a "threshold" value ("limit" value).

The results of these methods of extrapolation are presented in table 5.1. for both fresh water and seawater. The L(E)C50-values and NOEC-values used in the methods according to van Straalen (1987) and Kooijman (1987) are printed **bold** in the tables 2.2, 2.3, 2.4 and 2.5 in chapter 2 of the present document. In cases of the presence of two or more NOEC-values for one single species, only one value has been used. The "lowest" L(E)C50-values and NOEC-values used in the method according to Slooff et al. (1986) are underlined in these tables. The values used were selected on the basis of both test procedure (reliability, test medium, exposure time, effect-parameters) and representative value for the species involved. Therefore, in all cases only values from primary literature sources have been used in the methods of extrapolation. The distribution of NOEC-values for freshwater organisms is shown in figure 5.1. The only NOEC-value for seawater organisms has been marked by a cross.

Data for the organic arsenic compounds were much too limited to be used in assessing "limit" values.

and surface waters (especially with regard to the difference in particulate matter content), it is assumed that in most tests arsenic was present as "dissolved"-arsenic. For this reason the concentration of 6 μg $As.l^{-1}$ is considered to be "dissolved"-arsenic (< 0.45 μm).

Based on L(E)C50-values from short-term tests a HCS of 0.5 μg $As.l^{-1}$ has been calculated using the method according to Kooyman (1987). Using the method according to Slooff et al. (1986) on the basis of the lowest L(E)C50-value an $NOEC_{ss}$ and $NOEC_{eco}$ of 32.7 and 68.0 μg $As.l^{-1}$, respectively, have been calculated. Dividing these values by the UF results in "safe" concentrations of 1.3 and 0.8 μg $As.l^{-1}$, respectively.

Seawater

Data on the toxicity of inorganic arsenic to seawater organisms are limited, especially with regard to chronic toxicity. Because only one NOEC-value from long-term tests is available, the method according to van Straalen can not be used for risk assessment. Using the method according to Slooff et al., based on the lowest L(E)C50-value from short-term tests, a $NOEC_{ss}$ and $NOEC_{eco}$ of 9.3 and 23.3 μg $As.l^{-1}$, respectively, have been calculated. By dividing these values by the UF's the "safe" concentrations of 0.36 and 0.27 μg $As.l^{-1}$ for single species and ecosystems, respectively, were obtained. Using the method according to Kooyman (1987) on the available L(E)C50-values results in a HCS of 0.0095 μg $As.l^{-1}$. On the basis of the lowest NOEC-value a $NOEC_{eco}$ of 86.7 μg $As.l^{-1}$ has been calculated, using the method according to Slooff et al. The "safe" value for seawater organisms is found when this value is divided by the UF; this results in a concentration of 2.6 μg $As.l^{-1}$.

It is not possible to compare the data on seawater organisms with those on freshwater organisms, because the dataset on seawater is too limited. However, there does not seem to be a consistent difference: the lowest relevant L(E)C50-value for freshwater organisms is higher (factor 3) than for seawater organisms, whereas the lowest NOEC-value for fresh water is lower (factor 3) than for seawater. Therefore, the "limit" value proposed for fresh water is provisionally recommended for seawater as well; 6 μg $As.l^{-1}$ "dissolved"-inorganic arsenic.

5.2.2 Terrestrial organisms

The dataset on accumulation in, and toxicity to terrestrial organisms is too limited to be used in assessing a "limit" value.
With regard to the toxicity of arsenic to plants two experiments, in which arsenic was added as water soluble compounds, were considered to be usable. In the first experiment an added concentration of 30 mg $As.kg^{-1}$ dm as lead arsenate or sodium cacodylate $[(CH_3)_2AsO(ONa)]$ caused a significant reduction in growth of the wild plant *Urtica dioica* (Otte et al., 1988). In this experiment the backgroundconcentration of the soil used was not given. In the second study no effects were found at circa 20 mg $As.kg^{-1}$ dm, consisting of a backgroundconcentration of 10 mg $As.kg^{-1}$ dm and an added concentration of 8 mg $As.kg^{-1}$ dm from Fe-, Al-, Na-, and Ca-arsenate. At an added concentration of 100 mg $As.kg^{-1}$ dm from Al-, Na-, and Ca-arsenate more than 50% growth inhibition was found. When arsenic was added as Fe-arsenate, at an added concentration of 100 mg $As.kg^{-1}$ dm no or only a slight effect was observed (Woolson et al., 1971). These effect concentrations are significant lower than the lowest tested concentrations in terrestrial organisms.
Only one study was available on the toxicity of arsenic to terrestrial organisms and this study was limited to microbe-mediated processes. At the lowest concentration tested, which were 500 mg $As.kg^{-1}$ from arsenic trioxide and 1000 mg $As.kg^{-1}$ from arsenate, effects were still seen on the number of micro-organisms and on the activity of microbe-mediated processes in a sandy and an alluvial soil (Maliszewski et al., 1985).
On the basis of these data higher plants seem to be the most sensitive to arsenic. It appears that the highest concentration without effect is 20 mg $As.kg^{-1}$ dm (including the backgroundconcentration) and the lowest concentration with effect is 30 mg $As.kg^{-1}$ dm, exclusive of backgroudconcentration.

Because of the fact that accumulation of arsenic in agricultural crops is relatively low and the fact that the toxicity of arsenic to livestock is also low, it is concluded that exposure to a concentration which causes no effect in higher plants will not cause adverse effects on humans and animals.

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