National Institute for Public Health
and the Environment
Ministry of Health, Welfare and Sport

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RIVM report 607711006/2012

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This investigation has been performed by order and for the account of the Ministry of Infrastructure and the Environment (I&M), Directorate General for the Environment and International affairs (DGMI), Directorate of Sustainable Production (DP), within the framework of project 607711, Risk in relation to Soil Quality.

Abstract

Proposal for revised Intervention Values for soil and groundwater for the 2nd, 3rd and 4th series of compounds

RIVM has evaluated the so-called Intervention Values for soil and groundwater. This evaluation is necessary in order to implement new scientific knowledge and model improvements in the derivation of these values and therefore to maintain the quality of the standards.
Intervention Values are national soil quality standards which are used to determine seriously contaminated sites. Seriously contaminated sites have to be remediated according to the Soil Protection Act.

The Intervention Values are derived for both soil as well as groundwater. For the derivation of Intervention Values, the concentration in soil and groundwater is determined for which unacceptable risks for humans and the ecosystem exist. The most stringent value of both determines the new intervention value. Additionally, RIVM determined situations in which organisms are exposed to high concentrations of a compound through bioaccumalution in the foodchain, the so-called secondary poisoning.

The first Intervention Values were derived in the nineties for the first of four series of compounds. For 66 compounds from the 2nd, 3rd and 4th series of compounds revision of the Intervention Values is currently needed. Due to limits in time, 16 most urgent compounds were selected. These are, for example, compounds that are often found at contaminated sites. The 16 compounds that have been evaluated are antimony, barium, boron, selenium, thallium, tin (inorganic), vanadium, three organotin compounds, *cis*- and *trans*-dichlororethene, free cyanide, thiocyanate, chloride and sulphate.
For three of these compounds the derived proposals for Intervention Values in this report have become more stringent and the proposals for Intervention Values for four other compounds are stated as less stringent. For several compounds, the derived proposals for Intervention Values were similar to the current Intervention Values. For the remaining compounds which did not have an intervention value yet, a new proposal was derived.

Keywords:
intervention values, risk assessment, standards, soil, groundwater, exposure

Rapport in het kort

Voorstellen voor herziening van interventiewaarden voor bodem en grondwater voor de 2e, 3e en 4e tranche stoffen

Het RIVM heeft de zogeheten interventiewaarden voor bodem en grondwater geëvalueerd. Dit is noodzakelijk om nieuwe wetenschappelijke kennis en modelverbeteringen in de afleiding van deze getallen te verwerken, en daarmee de kwaliteit van de normen te handhaven. Interventiewaarden zijn landelijke bodemnormen die gebruikt worden om aan te geven dat bodems die in het verleden verontreinigd zijn geraakt, volgens de Wet Bodemkwaliteit moeten worden gesaneerd.

De interventiewaarden zijn zowel voor de bodem als voor het grondwater bepaald. Voor beide is bekeken bij welke concentraties risico's voor het ecosysteem en voor de mens ontstaan. De strengste waarde bepaalt de hoogte van de interventiewaarde. Aanvullend is onderzocht wanneer ecosystemen aan te hoge concentraties blootstaan doordat een stof in de voedselketen ophoopt, de zogenoemde doorvergiftiging.

In de jaren negentig zijn voor het eerst de interventiewaarden van vier groepen stoffen in delen (zogeheten tranches) afgeleid. Van 66 stoffen uit de 2e, 3e, en 4e tranche moeten de interventiewaarden worden geëvalueerd. Vanwege een beperkt tijdbestek zijn hieruit alleen de 16 meest urgente stoffen geselecteerd, bijvoorbeeld omdat ze in de praktijk vaak worden aangetroffen. De 16 stoffen zijn: antimoon, barium, boor, selenium, thallium, tin (anorganisch), vanadium, een drietal orgaotins, *cis*- en *trans*-dichlooretheen, vrije cyaniden, thiocyanaat, chloride en sulfaat. Voor drie van deze stoffen is de in deze rapportage voorgestelde interventiewaarde strenger geworden en voor vier stoffen is de voorgestelde interventiewaarde minder streng geworden. Voor enkele stoffen zijn de voorgestelde interventiewaarden niet veranderd. Voor de overige stoffen waarvoor nog geen interventiewaarde was afgeleid, is nu een eerste voorstel gedaan.

Trefwoorden: Interventiewaarden, risicobeoordeling, normen, bodem, grondwater, blootstelling

Contents

Summary

Between 1994 and 1998 Intervention Values have been derived for the 2nd, 3rd and 4th series of compounds. Intervention Values are generic soil quality standards used to classify historically contaminated soils as seriously contaminated under the framework of the Dutch Soil Protection Act. With the implementation of the Intervention Values it was agreed upon that these values would be regularly evaluated. This evaluation is necessary to keep the standards state-of-the art through the implementation of new scientific research data and model improvements.

The 2nd - 4th series of compounds consist of 66 chemicals that were qualified for evaluation of the Intervention Values. Due to limits in time a selection of priority compounds was made. The following 16 compounds are considered priority compounds: antimony, barium, boron, selenium, thallium, tin (inorganic), vanadium, three organotin compounds, *cis*- and *trans*-dichlororethene, free cyanide, thiocyanate, chloride and sulphate.

The Intervention Values are derived by integrating human-toxicological Maximum Permissible Risk (MPR_{human}) values and Ecotoxicological Maximum Permissible Risks (MPR_{eco}). The Serious Risk Concentrations (SRCs) are revised for both soil as well as groundwater. The current proposals for the Intervention Values are derived according to the procedures described for the first series of compounds in 2001 (Lijzen et al.) and are not described in detail in this report. It is worth mentioning that in contrast to the evaluation of the Intervention Values in 2001, a new CSOIL 2000 model (updated in 2006) was used for the current evaluation of the Intervention Values. With respect to the derivation in the nineties the model contains new user scenarios for the derivation of human risk limits.

In addition to direct toxicity, secondary poisoning is a relevant topic to take into account in risk assessment procedures. However, in the procedure for deriving soil quality standards (chapter 3), secondary poisoning is not included. The soil quality standards derived in this report are based on direct toxicity only. In this report, secondary poisoning is described for the sake of completeness. This information can be used later on for the derivation of maximal values in the precautionary framework, if necessary.

Table 1 presents the proposed Intervention Values for soil and groundwater as presented in this report.

Table 1: Proposed Intervention Values in (mg/kg_{dwt}) for soil and groundwater (µg/L).

Compound	**<u>Proposed</u> intervention values for <u>soil</u> [mg/kg_{dwt}]**	**<u>Proposed</u> intervention values for <u>groundwater</u> [µg/L]**
Antimony	150	190
Barium	400	630
Boron	3100	6300
Selenium	5.9	130
Thallium	2.5	1.3
Tin (inorganic)	260	6300
Vanadium	110	70

Compound	**Proposed intervention values for soil [mg/kg$_{dwt}$]**	**Proposed intervention values for groundwater [µg/L]**
Organotin compounds (sum)	-	-
Dibutyltin (DBT)*	30	8.0
Tributyltin (TBT)*	20	0.05
Triphenyltin (TPT)*	60	0.4
1,2-Dichloroethene (sum)*	3.0	940
Free cyanide	0.04	30
Thiocyanate	33	350
Chloride	390	5.7×10^5
Sulphate	-	-

- = not derived
* sum = geometric mean of the SRCs of the individual compounds.

1 Introduction

Within the framework of the Dutch Soil Protection Act Intervention Values are used to discriminate contaminated soil from serious contaminated soil (see also Swartjes et al. 2012). For the 1st series of compounds, Intervention Values were derived in 1991. A 2nd, 3rd and 4th series of compounds followed in respectively 1994, 1995 and 1998 (Van den Berg et al. 1994; Kreule et al. 1995 Kreule and Swartjes 1998). With the implementation of the Intervention Values it was agreed upon that these values would be regularly evaluated. This evaluation is necessary to implement new scientific research data and model improvements to maintain a high quality level of standards. In 2001 the first series of Intervention Values was evaluated. See the report of Lijzen et al. (2001) for detailed information on the procedure.

Within the framework of the project 607711, Risk in relation to Soil Quality, it was decided that an evaluation of the 2nd, 3rd and 4th series of compounds should follow. Selection of relevant compounds to be evaluated, prioritization and planning of activities were discussed with the project group NOBOWA (in Dutch NOrmstelling BOdem en Water. Free translation: Standards for soil and water) and will be discussed more in detail in the next section.

1.1 Selection of compounds

The 2nd - 4th series of compounds consists of 66 chemicals. Due to limits in time a selection was made to determine the most important compounds from this 2nd – 4th series. Furthermore, some compounds from the first series were selected for evaluation as well.

Some of the changes that have been made since the first derivation of Intervention Values in the nineties are:

- changes in exposure modeling (Rikken et al. 2001);
- changes in procedures and assumptions for deriving Intervention Values (Lijzen et al. 2001);
- new methods for deriving environmental Serious Risk Concentration (SRC_{eco}) (Verbruggen 2001).

Furthermore, it is important to use the latest compound specific data, namely toxicological and physico-chemical data.

Based on several selection criteria a selection of priority compounds was made. The first three criteria determine the priority of a compound. The criteria 4-9 are a supplement to this priority. Criteria 10-12 determine the intensity of evaluation of a compound.

Criteria for high priority:

- degree of occurrence at soil contaminations in practice;
- compounds which are considered relevant by the policy advisory group NOBOWA;
- compounds for which the current intervention value does not match with the risk limits in the precautionary framework.

Criteria with a lower priority:

- compounds which are marked as a so-called Indicative level for serious contamination in the current Soil Remediation Circular (2009) have a higher priority than compounds that already have an intervention value.
- the age of the current standard. Compounds with a standard dated before 1998 have a higher priority than more recent derived standards.
- compounds in which new developments in human toxicological risk assessment have occurred (Janssen and Speijers, 1997) need to be evaluated.
- compounds in which new developments in environmental toxicological risk assessment have occurred, need to be evaluated.
- availability of a new European Risk Assessment summary Report (EU-RAR).
- appearance of the compound in the compounds list of the EU Water Framework Directive.

Criteria for determining the intensity of the evaluation:

- mobility of compounds in soil or groundwater. If the mobility of a compound is high, an evaluation of soil standards has a lower priority because the compound will leach from the soil into the groundwater. Priority will be given to the standard for groundwater.
- depending on which risk is critical, human or ecology, the risk for either human health or the ecosystem will be the main focus of attention.
- if one compound within a group of compounds (for example organotin compounds) is evaluated, other compounds from this group will be evaluated as well.

Table 1.1 shows the selected compounds with priority for evaluation of the intervention value.

Table 1.1:Compounds with priority for evaluation of the Intervention Values (1st, 2nd, 3rd and 4th series) and compounds where Intervention Values were not proposed previously (-).

Compound group	**Series of compounds**	**CAS number**
Metals and metalloids		
Antimony	2nd	7440-36-0
Barium	1st	7440-39-3
Boron	2nd	7440-42-8
Selenium	4th	7782-49-2
Thallium	4th	7440-28-0
Tin (inorganic)	4th	7440-31-5
Vanadium	4th	744-62-2
Organotin compounds		
Dibutyltin dichloride (DBT)	-	683-18-1
Tributyltin-cation (TBT)	2nd	36643-28-4
Triphenyltin-cation (TPT)	2nd	892-20-6
Chlorinated hydrocarbons		
Cis-1,2-dichloroethene	3rd	156-59-2

Compound group	Series of compounds	CAS number
Trans-1,2-dichloroethene	3rd	156-60-5
Inorganic compounds		
Free cyanide	1st	57-12-5[1]
Thiocyanate	1st	540-72-7[2]
Chloride	-	16887-00-6
Sulphate	-	7757-82-6[3]

[1]CAS number of the cyanide anion.[2]CAS number of NA-SCN. [3]CAS number of NA_2SO_4

1.2 Components of evaluation

The evaluation of the 2nd – 4th series of compounds as described in this report has been done in accordance with the procedure as described by Lijzen et al. (2001). Therefore, this report will not go into detail on the procedure used, except if it deviates from Lijzen et al. (2001). The evaluation in this report consists of the following aspects:

- evaluation of the MPR for humans (chapter 2);
- evaluation of the ecotoxicological SRC_{eco} (chapter 3);
- evaluation of underlying input data for the human exposure models. Only the accumulation of metals in crops (Bioconcentration factor or BCF) and the partition coefficient are modified (if possible) for deriving risk limits in this study (chapter 4);
- evaluation of secondary poisoning in the terrestrial environment (chapter 5);
- discussion and conclusion of some specific compounds and choices made (chapter 6).

2 Human-toxicological risk limits

2.1 Introduction

Intervention Values are derived based on human-toxicological Maximal Permissible Risk (MPR_{human}) values and Ecotoxicological Maximal Permissible Risks (MPR_{eco}). This chapter comprises the revision of the MPR_{human} for the selected compounds. For several compounds the MPR_{human} was evaluated by Tiesjema and Baars (2009), the remaining compounds have been evaluated for the purpose of this report. Detailed information is presented in annex 1. The MPR_{eco} will follow in chapter 3.

2.1.1 Definitions and approach

The MPR_{human} is defined as the amount of a substance that any human individual can be exposed to daily during a full lifetime without significant health risk. It covers both oral, dermal and inhalation exposure, through both threshold compounds and carcinogenic compounds. The MPR_{human} is generally expressed as either a tolerable daily intake (TDI) or an excess carcinogenic risk via intake (CR_{oral}), both covering exposure by oral ingestion, a tolerable concentration in air (TCA) or an excess carcinogenic risk via air (CR_{inhal}), both covering exposure by inhalation. The procedure to derive MPR_{human} is outlined in detail by Janssen and Speijers (1997).

The approach of the present evaluation is a pragmatic one: use has been made of existing toxicological evaluations by national and international bodies in an attempt to avoid unwanted duplication of work. Existing evaluations were used in a critical fashion: on a case-by-case basis their adequacy for use in the present scope was judged and from that the need to search additional literature was determined. Throughout this chapter the abbreviation 'MPR' is used to indicate the MPR_{human} (Lijzen et al., 2001).

For a more elaborate explanation of the general procedure to derive MPR's we refer to chapter 4 of the report of Lijzen et al. (2001). Hereafter a summary of the revised MPRs are given. Annex 1 presents the derivation more in detail.

Table 2.2 in section 2.15 shows a summary of the current (old) and the evaluated (new) human-toxicological risk limits.

2.2 Antimony

Antimony is a metalloid that occurs mainly in a trivalent or pentavalent state. Natural levels in the environment (soil, water) are at the ppm (parts per million) or ppb (part per billion) level. Antimony trioxide, the antimony compound which is commercially most significant, is used as a flame retardant synergist, as a fining agent in glass manufacture and as a catalyst in plastics. Antimony potassium tartrate has a historical use as anti-schistosomal drug or, in the case of poisoning, to induce vomiting. Antimony is released into the environment (predominantly in the form of antimony trioxide) mostly as a result of coal burning or with fly ash when antimony-containing cores are smelted. Antimony is a non-essential element.

Food, including vegetables grown on antimony-contaminated soils, is the most important source of antimony exposure for the general population. Oral uptake of antimony via food and drinking water is low.

Average daily intake (background exposure) is estimated to be 0.4 µg antimony/kg bw/day. A TDI (Tolerable Daily Intake) of 6 µg antimony/kg

bw/day (oral exposure) could be derived. A TCA (Tolerable Concentration in Air) for inhalation exposure of antimony is not derived because inhalation exposure of antimony is considered not relevant (Tiesjema and Baars, 2009).

2.3 Barium

Barium belongs to the alkali-earth metals. The soluble barium chloride is used in pigments, ceramics, glass and paper products. Besides, it is used in various industrial activities such as tanning and magnesium production. These activities may lead to diffuse contamination of the top soil. Contamination of groundwater is usually the result of naturally occurring barium. The insoluble barium sulphate ('barite') is used in large quantities as weighting agent in oil well drilling mud. These drilling activities may lead to local contamination of deep soil with very high concentrations. Groundwater will then be contaminated if the concentrations of barium sulphate are very high. There is no indication that barium can be considered as an essential element for humans. The major source of exposure is food.

The average daily intake (background exposure) in the Netherlands amounts to 9 µg/kg bw/day. A TDI of 20 µg/kg bw/day (oral exposure) was maintained and a TCA of 1.0 µg/m^3 was derived (inhalation exposure) by Baars et al. (2001). There is still some discussion about the difference in bioavailability of different barium salts. A evaluation of the human-toxicological values is not necessary. In the evaluation of 2001 a difference was made between the soluble and insoluble barium salts.

2.4 Boron

Boron is a metalloid that is widespread in nature at relatively low concentrations. In the environment boron is always found chemically bound to oxygen, usually as alkali or alkaline earth borates, or as boric acid. Boron is not present in the atmosphere at significant levels. Boric acid, borax and other borates are used in a wide range of consumer products, like glass, soaps, detergents, preservatives, fertilisers, insecticides and herbicides (ATSDR, 2007; EFSA, 2004; US-EPA, 2004; WHO, 2009). It is a trace element, however it's essentiality to humans has not yet been directly proven (US-EPA, 2004). The essentiality of boron has been established for most plants and some animals (ATSDR, 2007). Food is the main source of exposure for most populations but exposure via water, especially bottled mineral water, can be substantial as well. Data on dietary intakes of boron are limited. Food rich in boron includes fruits, leafy vegetables, mushrooms, nuts and legumes as well as wine, cider and beer.

Maximum daily intake is estimated to be 5 mg/day which equals 80 µg/kg bw for a 70 kg adult (Van Engelen, 2008). A TDI of 200 µg/kg bw/day is derived and a PTCA (Provisional Tolerable Concentration in Air) of 10 µg/m^3 is proposed. The derivation of the TDI and TCA for Boron is described in Annex 1.

2.5 Selenium

Selenium is a metalloid. It is present in the earth's crust, often in association with sulfur-containing minerals. It has four oxidation states (-2, 0, +4, +6) and occurs in many forms, including elemental selenium, selenites and selenates. Selenium is used in electronics, due to its semi-conductor and photoelectric properties. In addition, it is used for instance in the glass industry, in pigments, as a catalyst in the preparation of pharmaceuticals and as a constituent of fungicides. The principal releases of selenium into the environment result from the combustion of coal and crude oil processing.

For the general population food is the primary exposure route followed by water and air (ATSDR 2003). The main inorganic sources of selenium in the diet are selenate and selenite (ATSDR, 2003).

Being part of several enzymes, selenium is an essential element for humans and animals. Estimated daily requirements as summarised in SCF (2000) range from 40 to about 50 µg/day for adults with a lower limit of 20 µg/day.

EFSA (2000) gives an overview of daily intake levels in European countries. These data indicate daily intake levels up to about 1 µg/kg bw/day (Van Engelen, 2008). Also, European data as summarised by SCF (2000) indicate a mean adult daily intake of 1 µg/kg bw/day. For children twice this figure should be a reasonable estimate, i.e. 2 µg/kg bw/day. Based on the NOAEL of 0.85 mg Se/day from the study by Yang et al. (1989) and an uncertainty factor of 3 to account for sensitive individuals, the TDI is maintained at 5 µg Se/kg bw/day. A TCA for inhalation exposure to selenium is not derived, due to the lack of quantitative data concerning both human and animal exposures. The derivation of the TDI and TCA for Selenium is described in Annex 1.

2.6 Thallium

Thallium is a soft and pliable metal. Thallium exists in a monovalent thallo- and a trivalent thalli-state. It tends to form stable complexes such as sulphur-containing compounds.

In the past, thallium was extensively used for medicinal purposes (treatment of ringworm of the scalp, venereal diseases, tuberculosis and malaria). It was also used as a rodenticide and insecticide. Currently, the main uses of thallium are in the electrical and electronics industries, as in electronic devices for semi-conductors, scintillation counters, low temperature thermometers, in mixed crystals for infrared instruments and laser equipment.

Thallium does not have a known biological use and is a non-essential element for life (Kazantzis, 2000; Cvjetko, 2010). Thallium is highly toxic. In adults, the average lethal oral dose has been estimated to range from 10 to 15 mg/kg. The greatest exposure occurs by eating food, mostly homegrown fruits and green vegetables contaminated by thallium. The average dietary intake of thallium (background exposure) is less than 5 µg/day or 0.07 µg/kg bw/day (assuming an average body weight of 70 kg) (Kazantzis, 2000; Peter and Viraraghavan, 2005).

A TDI of 0.04 µg thallium/kg bw/day was derived. Because of the limitations in the dataset and key study the TDI is provisional. A TCA is not derived since thallium is considered not volatile. The derivation of the TDI and TCA for Thallium is described in Annex 1.

2.7 Tin and inorganic tin compounds

Tin is a silver-white metal that can occur in a divalent (Sn(II)) and tetravalent (Sn(IV)) oxidation state. Natural occurrence of tin in the metallic state is rare. Tin is thought to be relatively immobile in soil. Concentrations in soil vary between 2 and 200 mg/kg, but may be higher in areas of high tin deposits. Background concentrations in soils of Dutch nature reserves range from 0.6-28.1 mg/kg. Tin is used in the lineage of cans and containers. It is also present in tin alloys and some soldering materials. Inorganic tin compounds are used in the glass industry, as catalysts, in food additives and as stabilizers in perfumes and soaps. Contamination will occur mostly from production and use of tin and tin compounds. There is no evidence that tin is nutritionally essential for humans.

The most important source for exposure to tin is from canned food products. According to Vermeire et al. (1991), the maximum daily intake (background exposure) in the Netherlands is approximately 140 µg/kg bw/day. Data from Total Diet Studies in the UK in 1997 mentioned by EFSA (2005) are in the same range of this value, which is therefore maintained.

It is recommended that the TDI as derived by Vermeire et al. (1991) is replaced by a new TDI of 200 µg tin/kg bw/day. Due to a lack of reliable data a TCA for inhalation exposure to tin is not derived (Tiesjema and Baars, 2009).

2.8 Vanadium

Vanadium is a widely distributed transition metal in the earth's crust. In the environment it occurs in varying oxidation states (3^+, 4^+ and 5^+ being the most common) but not as elemental vanadium. In tissues of organism, vanadium predominantly occurs in 3^+ and 4^+ states, due to reduction, while in plasma the 5^+ state is most common. Vanadium is used in alloys with steel and as an oxidation catalyst in chemical industries. The estimated total global emission of vanadium into the atmosphere ranges from 71,000 to 210,000 tons per year. Although vanadium has been considered an essential element for chickens and rats, there is no evidence that vanadium is an essential element for humans.

In 1998, a background exposure of 0.3 µg/kg bw/day was estimated by Janssen et al. (1998).

The provisional TDI of 2 µg vanadium/kg bw/day and the provisional TCA of 1 µg vanadium/m^3 are maintained (Tiesjema and Baars, 2009).

2.9 Organotin compounds

Organotin compounds do not naturally occur in the environment. They are used as polyvinyl chloride heat stabilizers, biocides, catalysts, agrochemicals and glass coatings. Also products like shoes and clothing may contain organic tin. Organotin compounds have the tendency to accumulate in the environment and are highly toxic for aquatic organisms. They are released into the environment either directly (from pesticides and antifouling paints) or by leaching from, or disposal of consumer products.

In particular fish and other seafood are important sources of exposure of the general population to organic tin compounds. According to EFSA (2004), calculations based on the Norwegian consumption patterns of fish and seafood provided dietary exposure levels of 0.078, 0.046 and 0.047 µg/kg bw/day for tributyltin (TBT), dibutyltin (DBT) and triphenyltin (TPT), respectively. These values were based on the 95th percentile of dietary exposure to the mentioned organotin compounds. Since the Norwegian population consumes on average higher amounts of seafood than the Dutch population, we can assume that the dietary exposure in The Netherlands is lower.

The provisional TCA of 0.02 µg/m^3 for tributyltin oxide (TBTO), derived by Janssen et al. (1995), is retained due to the lack of new relevant data concerning the effects of inhalation exposure to TBT compounds. Due to a lack of available information, it is not possible to derive a TCA for other organotin compounds.

A group TDI was established for DBT, TBT and TPT, because the immunotoxic effects caused by these compounds are elicited by a similar mode of action and potency. Di-n-octyltin DOT was not included in the group TDI, because DOT has shown to have a significantly lower toxic potential than TBT (Tiesjema and Baars, 2009).

Table 2.1: Human risk limits for organotin compounds.

Compound	TDI [µg/kg bw/day]	PTCA [µg/m^3]	Background exposure [µg/kg bw/day]
Dibutyltin (DBT)	0.25*	-	0.05
Di-n-octyltin (DOT)	2.3	-	-
Tributyltin (TBT)	0.25*	0.02	0.08
Triphenyltin (TPT)	0.25*	-	0.05

*Group TDI for TBT, DBT and TPT.

2.10 *Cis*- and *trans* 1,2-dichloroethene

Cis- and *trans* 1,2-dichloroethene can be summarized under the common name 1,2-dichloroethene. These are synthetic chemicals which are used as chemical intermediates and industrial solvents. At room temperature they are highly flammable, colourless liquids with an ethereal, slightly acrid odour.

Cis- and *trans*-1,2-dichloroethene were firstly evaluated in 1995 (Janssen et al. 1995) and again in 2001 (Baars et al. 2001). In 2008 they were evaluated (Janssen, 2008) within the framework of the programme 'International normalisation of substances' of the Ministry of Infrastructure and the Environment. This last evaluation has been reported by Janssen, 2008, who concluded that on the basis of new toxicological data the difference between *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene was not longer warranted.

A TDI of 30 µg/kg bw/day and a PTCA of 60 µg/m^3 was derived. The author did not report about the background exposure level, but since there are no data justifying a change in the earlier estimate, the background exposure value as reported in the evaluation of 2001 (0.13 µg/kg bw/day) is maintained (Tiesjema and Baars, 2009).

2.11 Free cyanide

Cyanide occurs in various organic and inorganic compounds containing a cyano-group as part of their molecule. Cyanide is produced and used in various occupational settings where activities include electroplating, some metal mining processes, metallurgy, metal cleaning, certain pesticide applications, tanning, photography and gas work operations. Cyanide is also used in some dye and pharmaceutical industries. Cyanide is not persistent or accumulates in soils under natural conditions. Many species of bacteria, fungi algae and also plants are able to utilise cyanide and degrade it to less toxic substances like CO_2, NO_3^-, SCN^- and OCN^-.

Free cyanide (as CN^-) can be defined as the sum of cyanide in the form of HCN (aq, g) and CN^--ion; the toxicity data on HCN and on CN^--ion and its salts (i.e. calcium ($Ca(CN)_2$), potassium (KCN) and sodium (NaCN) are considered.

Relevant exposure routes are oral uptake and inhalation. A TDI of 50 µg/kg bw/day and a TCA of 25 µg/m^3 (odour threshold) was derived. The background exposure is not quantifiable according to Baars et al. (2001).

2.12 Thiocyanate

The exposure to thiocyanate (SCN^-) is in the same way as to free cyanide, because thiocyanate is the main metabolite of cyanide in the body. Thiocyanate is also present in food and as a soil contaminant. The relevant exposure route is oral ingestion.

A TDI of 11 µg/kg bw/day was derived. The TCA is not applicable because thiocyante is not volatile. The background exposure is 74 µg/kg bw/day according to Baars et al. (2001).

Thiocyanate is considered less toxic than free cyanide. Therefore, it is curious that the TDI for thiocyanate is lower than for free cyanide. This has to do with the available information on toxicity of both compounds. Less information is available for thiocyanate, therefore a larger uncertainty factor is applied resulting in a higher TDI for thiocyanate.

2.13 Chloride

Chloride salts are vital for human metabolic processes and essential for maintaining electrical neutrality in the body. Chloride is the main electrolyte in the mammalian body: it represents 70% of the body's total negative ion content. The suggested amount of chloride intake for an adult is 750-900 mg/day.

For the purpose of human-toxicological MPR's in the framework of soil contamination, chloride can be considered non-toxic (Tiesjema and Baars, 2009).

2.14 Sulphate

Sulphates occur naturally in numerous minerals and are extensively used commercially. The highest levels occur in groundwater and are from natural origin. The average daily intake of sulphate from water, air and food is approximately 500 mg, food being the major source. Sulphates can give a bad taste to drinking water.

For the purpose of human-toxicological MPRs in the framework of soil contamination, sulphate can be considered non-toxic (Brand, 2008 and Tiesjema and Baars, 2009).

2.15 Summary of the human-toxicological risk limits

Table 2.2 presents a summary of the current (old) and the evaluated (new) human-toxicological risk limits.

Table 2.2: Summary of old and new human-toxicological risk limits.

Compound	Old TDI [µg/kg bw/day]	Old PTCA or or TCA [µg/m^3]	New TDI [µg/kg bw/day]	New PTCA or TCA [µg/m^3]
Metals and metalloids				
Antimony	0.86	-	6	-
Barium	20	-	20	1.0
Boron	90	-	200	10
Selenium	5	-	5	[a]
Thallium	200	-	0.04	-
Tin (inorganic)	2000	[a]	200	-
Vanadium	2	1	2	1
Organotin compounds				
Dibutyltin (DBT)	-	-	0.25[b]	-
Di-n-octyltin (DOT)	-	-	2.3	-
Tributyltin (TBT)	0.3	0.02	0.25[b]	0.02
Triphenyltin (TPT)	0.5	-	0.25[b]	-

Compound	Old TDI [µg/kg bw/day]	Old PTCA or or TCA [µg/m^3]	New TDI [µg/kg bw/day]	New PTCA or TCA [µg/m^3]
Chlorinated hydrocarbons				
Cis-1,2-chloroethene	6	30	30	60
Trans-1,2-chloroethene	17	60	30	60
Inorganic compounds				
Free cyanide	50	200	50	25
Thiocyanate	11	-	11	-
Chloride	-	-	-	-
Sulphate	-	-	-	-

TDI: Tolerable Daily Intake (oral exposure).
(P)TCA: (Provisional) Tolerable Concentration in Air (inhalation exposure).
[a] Due to a lack of reliable data a TCA for inhalation exposure is not derived.
[b] Group TDI for TBT, DBT and TPT.

3 Environmental risk limits

3.1 Introduction

Besides human risk limits, Environmental Risk Limits (ERLs) for soil and groundwater (gw) are required as well. The environmental risk limits (SRC_{eco} or HC50) are based on the hazardous concentration where 50% of the test species and/or processes in an ecosystem may encounter adverse effects, based on single species laboratory studies. To present a complete overview of environmental risk limits, the ERLs for fresh surface water (fw) and sediment (sed) are also presented, when available. The evaluation also includes the saltwater compartment and the Maximal Acceptable Concentration (MAC_{eco}) for surface water. The ERLs for surface water, sediment and the saltwater compartment have not been used for the derivation of Intervention Values.

A full description of the methodology used for the derivation of ERLs will not be described in this report. For each compound reference will be made to the report describing the method of derivation used. For the derivation of ERLs for metals the added risk approach is followed. A short description of the added risk approach and the background concentrations used is given in the next section.

For the environmental risk limits presented in the following sections, it has not been investigated if additional data on physico-chemical properties or (eco) toxicity is available since the derivation of the cited ERLs.

Currently, ERLs for soil and sediment are expressed for Dutch standard soil or sediment with 10% organic matter. In some cases, normalisation to Dutch standard soil or sediment was considered not realistic because sorption was influenced by more than organic matter only. Therefore, in every case is indicated if ERLs are normalised or not.

Table 3.1 in section 3.18 shows a summary of the Serious Risk Concentrations (SRCs) for soil and groundwater. An overview of all derived ERLs is given in Annex 2.

3.2 Added risk approach

To derive the SRC_{eco} for metals, the added risk approach ihas been used (Crommentuijn et al., 2000). The background concentration (Cb) can contribute significantly to the SRC_{eco}. In the experiments used to derive the HC50, the background concentration in the test soils are neglected and do not contribute to toxicity. The HC50 is therefore derived based on added amounts of compound to the test soil. This added amount is referred to as the Serious Risk Addition (SRA). For the purpose of Intervention Values, the SRC is compared with a concentration in the field which is not a measure of the added (anthropogenic) concentration but a total concentration (Lijzen et al., 2001). Therefore the SRC_{eco} is equal to the SRA and the backgroundconcentration (SRC_{eco} = SRA + Cb). A generic Cb is used to derive a general SRC_{eco}.

Currently, there are two accepted references available for (natural) background values. These are the background values as determined by the steering Group Integral Standard Setting (INS) (Crommentuijn et al., 1997) and the background values as derived in the project AW2000 (Background values 2000) (Spijker and Van Vlaardingen, 2007; VROM, 2008). The INS- as well as the AW2000 background values are based on soil concentrations from relatively unpolluted natural areas in the Netherlands. However, both are based on a different dataset of measurements at different locations. Also, the AW2000 background values are based on the 95% percentile of the soil concentrations

where as the INS values are based on the 90% percentile. Furthermore, different methodologies were applied in deriving background concentrations from the datasets. From some points of view, the AW2000 values are considered to include anthropogenic influence therefore the INS values should better estimate natural background concentrations. The latter is however contradicted because some AW2000 values are lower than the equivalent INS values. Without concluding which of the values are the most realistic in representing natural background concentrations, the AW2000 values are used in this report because they are commonly used in the Dutch soil policy for which this report is written. In the last columns of tables 4.1 and 4.2 both background values are presented. In the absence of AW2000 values, INS values are used. For not natural compounds, like organotin compounds, the added risk approach is not followed and background concentrations are not used. For sediment, the AW2000 background values are the same as the INS background values.

It might be possible that the natural background on location differs from this general background. The site-specific information on background concentrations can be used when determining the remediation urgency. To estimate the background concentrations, the fraction of organic matter and lutum of the soil can be used (Lijzen et al. 2001).

3.3 Antimony

The ERLs for water and soil are cited from Van Leeuwen and Aldenberg (2011) and taken over for this report.

3.3.1 Water

The $SRC_{fw, eco}$ for antimony is 9600 µg antimony/L. This value is not determined by the background concentration (Cb) of 0.29 µg antimony/L. The Serious Risk Addition ($SRA_{fw, eco}$) is also 9600 µg antimony/L. With the reported Cb for groundwater of 0.09 µg antimony /L, the $SRC_{gw, eco}$ is also 9600 µg antimony /L. For the saltwater environment there were not enough data available to derive a $SRA_{sw, eco}$.

3.3.2 Soil

The $SRC_{soil, eco}$ for antimony is 1400 mg antimony/kg_{dwt}; this value is not influenced by a Cb of 4 mg antimony/kg_{dwt}. The $SRA_{soil, eco}$ is also 1400 mg antimony/kg_{dwt}. Based on the characteristics of the substance, normalisation for binding to organic matter is not applicable. Therefore these values have not been normalised to Dutch standard soil with 10% organic matter.

3.3.3 Sediment

The $SRC_{sediment, fw, eco}$ for antimony is 110 mg/kg_{dwt}. This value is not influenced by a Cb for freshwater sediment of 3 mg/kg_{dwt}. The $SRA_{sediment, fw, eco}$ is 110 mg/kg_{dwt}. Based on the characteristics of the substance, normalization for binding to organic matter is not applicable. Therefore normalization to Dutch standard sediment with 10% organic matter is not performed. For the saltwater environment there were not enough data available to derive a $SRA_{sediment, sw, eco}$.

3.4 Barium

The ERLs for water and soil are cited from Van Vlaardingen et al. (2005) and taken over for this report.

3.4.1 Water

The $SRC_{fw, eco}$ for barium derived by Van Vlaardingen et al. (2005) is

1.67 x 10^4 µg/L. This value is slightly determined by a Cb of 73 µg/L. The $SRA_{fw, eco}$ is 1.65 x 10^4 µg/L. Rounded, the $SRC_{fw, eco}$ and $SRC_{fw, eco}$ are both 1.7 x 10^4 µg/L. With the reported Cb for groundwater of 200 µg/L, the $SRC_{gw, eco}$ is 1.7 x 10^4 µg/L. For the saltwater environment there were not enough data available to derive a $SRA_{sw, eco}$.

3.4.2 Soil

The $SRC_{soil, eco}$ for barium is 400 mg/kg_{dwt} soil, which is composed of a Cb of 190 mg/kg_{dwt} and a $SRA_{soil, eco}$ of 206 mg/kg_{dwt}. The $SRA_{soil, eco}$ based on microbial processes and enzymatic reactions was 990 mg/kg_{dwt}, while the $SRA_{soil, eco}$ based on toxicity data for other terrestrial species was 206 mg/kg_{dwt}. The lowest $SRA_{soil, eco}$, rounded as 210 mg/kg_{dwt}, was selected. Based on the characteristics of the substance, these values have not been normalised to Dutch standard soil with 10% organic matter.

3.4.3 Sediment

The $SRC_{sediment, fw, eco}$ for barium is 2.52 x 10^4 mg/kg_{dwt}. This value is slightly influenced by a Cb for freshwater sediment of 155 mg/kg_{dwt}. The $SRA_{sediment, fw, eco}$ is 2.5 x 10^4 mg/kg_{dwt}, calculated from the $SRA_{fw, eco}$ through equilibrium partitioning with a log $K_{p, susp\text{-}water}$ of 3.18. In the risk model CSOIL 2000 (Brand et al. 2007) a more actual Kd of 2500 L/kg is available. With this Kd the $SRA_{sediment, fw, eco}$ is 4.15 x 10^4 mg/kg_{dwt} giving a $SRC_{sediment, fw, eco}$ of 4.17 x 10^4 mg/kg. The latter value is considered more appropriate. Based on the characteristics of the substance, these values have not been normalised to Dutch standard sediment with 10% organic matter.

These values have been calculated with equilibrium partitioning and are very high. This indicates that the application of equilibrium partitioning is probably not justified in this case, or that the risk for an ecosystem is low due to high sorption of barium to sediment. For clarification, additional research with benthic organisms is suggested. For the saltwater environment, there were not enough data available to derive an $SRA_{sediment, sw, eco}$.

3.5 Boron

Boric acid is one of the natural forms of boron. The boron ion itself does not exist in the environment and all naturally occurring forms of boron are present as boric acid species. Boric acid is used as a model substance for risk assessment. The ERLs are cited from Van Herwijnen and Smit (2010).

ERL derivation was performed following Van Vlaardingen and Verbruggen (2007), based on the European assessment of boron in the context of biocide authorisation under Directive 98/8/EC.

Background concentrations for soil are currently not available for boron; therefore ERLs for boron in soil are expressed in terms of added concentrations for elemental boron.

3.5.1 Water

The $SRC_{aquatic, eco}$ for boron is 6900 µg boron/L. This value is slightly determined by a Cb of 62 µg boron/L. The $SRA_{aquatic, eco}$ is 6800 µg boron/L. The $SRC_{aquatic, eco}$ is only valid for the fresh surface water environment. With the Cb of 262 µg Boron/L for fresh groundwater the $SRC_{fgw, eco}$ is 7100 µg boron/L. With the Cb of 3454 µg boron/L for salt and brackish groundwater the $SRC_{sgw, eco}$ is 1 x 10^4 µg boron/L.

3.5.2 Soil

The $SRA_{soil, eco}$ for boron is 10.9 mg boron/kg_{dwt}, rounded this value is 11 mg boron/kg_{dwt}. The $SRC_{soil, eco}$ for boron can be calculated by SRC = SRA + Cb. The $SRA_{soil, eco}$ is based on the geometric mean of all chronic toxicity values. The $SRA_{soil, eco}$ is not converted to Dutch standard soil because sorption to soil is determined by more parameters than organic carbon and a reference line for boron is not available.

3.5.3 Sediment

In Van Herwijnen and Smit (2010) ERLs for sediment are not derived since the log $K_{p, susp-water}$ of boron is below the trigger value of 3.

3.6 Selenium

The ERLs for water are cited from Van Vlaardingen and Verbruggen (2009) and taken over for this report. The ERLs for soil are cited from Van Vlaardingen et al. (2005).

3.6.1 Water

The $SRC_{fw, eco}$ for selenium is 130 µg/L. The Cb of 0.041 µg/L is negligible as compared to the $SRA_{fw\ eco}$ of 130 µg/L. With the reported Cb for groundwater of 0.024 µg/L, the $SRC_{gw, eco}$ is also 130 µg/L. For the saltwater environment, the $SRA_{sw, eco}$ is 582 µg/L. No background concentration is available for the marine environment, therefore a $SRC_{sw, eco}$ cannot be given. The SRC for selenium can be calculated by SRC = SRA + $Cb_{saltwater}$.

3.6.2 Soil

The $SRC_{soil, eco}$ for selenium is 1.9 mg/kg_{dwt} soil, which is composed of a Cb of 0.7 mg/kg_{dwt} and a $SRA_{soil, eco}$ of 1.2 mg/kg_{dwt}. The $SRA_{soil, eco}$ based on microbial processes and enzymatic reactions was 130 mg/kg. The $SRA_{soil, eco}$ based on toxicity data for other terrestrial species was 1.2 mg/kg_{dwt}. The latter value was based on chronic toxicity data for plants. Based on the characteristics of the substance, these values have not been normalised to Dutch standard soil with 10% organic matter.

3.6.3 Sediment

In Van Vlaardingen and Verbruggen (2009) ERLs for sediment are not derived since the log Kp, susp-water of selenium is below the trigger value of 3.

3.7 Thallium

The ERLs for water are cited from Van Vlaardingen and Verbruggen (2009) and taken over for this report. The ERLs for soil are cited from Van Vlaardingen et al. (2005).

3.7.1 Water

The $SRC_{fw, eco}$ for thallium is 6.5 µg/L. This value is not influenced by a Cb of 0.038 µg/L. The $SRA_{fw, eco}$ is also 6.5 µg/L.

No $SRC_{gw, eco}$ has been derived in Van Vlaardingen and Verbruggen (2009) because no realistic Cb for groundwater was available. For the saltwater environment the $SRA_{sw, eco}$ is 616 µg/L. No background concentration is available for the marine environment, therefore a $SRC_{sw, eco}$ cannot be given. The SRC for thallium can be calculated by SRC = SRA + $Cb_{saltwater}$.

3.7.2 Soil

The $SRC_{soil, eco}$ for thallium is 2.0 mg/kg soil, which is composed of a Cb of 1.0 mg/kg and a $SRA_{soil, eco}$ of 1.0 mg/kg. The $SRA_{soil, eco}$ is based on aquatic toxicity data using equilibrium partitioning; this value was lower than the $SRA_{soil, eco}$ based on toxicity data from soil species. Based on the characteristics of the substance, these values have not been normalised to Dutch standard soil with 10% organic matter.

3.7.3 Sediment

The $SRC_{sediment, fw, eco}$ for thallium is 11 mg/kg_{dwt}. This value is slightly influenced by a Cb for freshwater sediment of 1.0 mg/kg_{dwt}. The $SRA_{sediment, fw, eco}$ is 9.8 mg/kg_{dwt}.

For the saltwater environment the $SRA_{sediment, sw, eco}$ is 935 mg/kg_{dwt}. No background concentration is available for marine sediment, therefore a $SRC_{sw, eco}$ cannot be given. The SRC for thallium can be calculated by SRC = SRA + $Cb_{saltwater, sediment}$. Based on the characteristics of the substance, these values have not been normalised to Dutch standard sediment with 10% organic matter.

3.8 Tin (inorganic)

The ERLs for water are cited from Van Vlaardingen and Verbruggen (2009) and taken over for this report. The ERLs for soil are cited from Van Vlaardingen et al. (2005).

3.8.1 Water

The $SRC_{fw, eco}$ for tin is 400 µg/L. The Cb of 0.0082 µg/L is negligible as compared to the $SRA_{fw, eco}$ is 400 µg/L. No $SRC_{gw, eco}$ has been derived in Van Vlaardingen and Verbruggen (2009) because no realistic Cb for groundwater was available. For the saltwater environment were not enough data available to derive a $SRA_{sw, eco}$.

3.8.2 Soil

The $SRC_{soil, eco}$ for tin is 260 mg/kg_{dwt}, which is composed by rounding a Cb of 6.5 mg/kg_{dwt} and a $SRA_{soil, eco}$ of 249 mg/kg_{dwt}. The $SRA_{soil, eco}$ is based on seven results for microbial processes and/or enzymatic reactions. No acute or chronic toxicity data of tin for terrestrial species were available. Based on the characteristics of the substance, these values have not been normalised to Dutch standard soil with 10% organic matter.

3.8.3 Sediment

The $SRC_{sediment, fw, eco}$ reported in Van Vlaardingen and Verbruggen (2009) for tin is 1.49 x 10^5 mg/kg_{dwt}. This value is slightly influenced by a Cb for freshwater sediment of 19 mg/kg_{dwt}. The $SRA_{sediment, fw, eco}$ is 1.48 x10^5 mg/kg_{dwt}, calculated from the $SRA_{fw, eco}$ through equilibrium partitioning with a log $K_{p, susp-water}$ of 5.57. In the risk model CSOIL 2000 (Brand et al., 2001) a more actual Kd of 1905 L/kg (log = 3.28) is available. With this Kd the $SRA_{sediment, fw, eco}$ is 760 mg/kg_{dwt} giving a rounded $SRC_{sediment, fw, eco}$ of 780 mg/kg. The latter value is considered more appropriate. Based on the characteristics of the substance, these values have not been normalised to Dutch standard sediment with 10% organic matter. For the saltwater environment there were not enough data available to derive a $SRA_{sediment, sw, eco}$.

3.9 Vanadium

3.9.1 Water

The ERLS for vanadium in water have been revised in 2012 (Smit, 2012). In this report no SRC values have been derived. However, the $MPC_{fw, eco}$ and $MAC_{fw, eco}$ are based on a species sensitivity distribution from which also HC50 values were calculated. From the HC50 values a $SRC_{fw, eco}$ can be derived. The HC50 from chronic freshwater data is 62 µg vanadium/L (range 12-315) and from acute freshwater data is 2722 µg vanadium/L (range 1542-4806). The acute HC50 divided by 10 is higher than the chronic HC50. Therefore, the $SRA_{fw, eco}$ is based on the chronic HC50: 62 µg vanadium/L. With the background concentration of 0.82 µg vanadium/L, the $SRC_{fw, eco}$ is 63 µg vanadium/L. For groundwater the background concentration in groundwater of 1.2 µg vanadium/L does also give a $SRC_{gw, eco}$ of 63 µg vanadium/L. For saltwater no ERLs have been derived.

3.9.2 Soil

The ERLs for soil are cited from Van Vlaardingen et al. (2005).

The $SRC_{soil, eco}$ for vanadium is 105 mg/kg_{dwt}, which is composed of a Cb of 80 mg/kg_{dwt} and a $SRA_{soil, eco}$ of 25 mg/kg_{dwt}. The $SRA_{soil, eco}$ based on microbial processes and enzymatic reactions was 84 mg/kg_{dwt}, while the $SRA_{soil, eco}$ based on toxicity data for other terrestrial species was 25 mg/kg_{dwt}. It should be noted that only one toxicity endpoint was available for 'other terrestrial species'. The resulting $SRC_{soil, eco}$ is based on the result of this toxicity study. Based on the characteristics of the substance, these values have not been normalised to Dutch standard soil with 10% organic matter.

3.9.3 Sediment

In the report of Smit (2012) no ERLs for sediment have been derived. A $SRC_{sediment, fw, eco}$ can be derived through equilibrium partitioning with the $SRA_{fw, eco}$ given above and the Kp value of 309 given in Annex 3. The resulting $SRA_{sediment, fw, eco}$ is 96 mg/kg_{dwt}. With a background concentration of 42 mg/kg_{dwt} as reported by Van Vlaardingen and Verbruggen (2009), the $SRC_{sediment, fw, eco}$ is 138 mg/kg. Based on the characteristics of the substance, these values have not been normalised to Dutch standard sediment with 10% organic matter.

3.10 Dibutyltin (DBT)

The ERLs for water, soil and sediment for the dibutyltin-ion are cited from Van Herwijnen (2012).

3.10.1 Water

The $SRC_{aquatic, eco}$ for the dibutyltin-ion is 16 µg/L. This value is valid for the freshwater and the saltwater environment. For groundwater the $SRC_{gw, eco}$ is 50 µg/L.

3.10.2 Soil

The $SRC_{soil, eco}$ for the dibutyltin-ion is 28 mg/kg_{dwt} for Dutch standard soil with 10% organic matter.

3.10.3 Sediment

The $SRC_{sediment, fw, eco}$ for dibutyltin is 123 mg/kg_{dwt} for Dutch standard sediment with 10% organic matter.

3.11 Tributyltin (TBT)

The ERLs for tributyltin, expressed for the TBT-cation, are adopted as the ERLs for all tributyltin compounds. The ERLs for tributyltin are cited from Van Herwijnen (2012).

3.11.1 Water

The $SRC_{aquatic, eco}$ for the tributyltin-cation is 0.026 µg/L. This value is valid for the freshwater and the saltwater environment. For groundwater the $SRC_{gw, eco}$ is 0.046 µg/L.

3.11.2 Soil

The $SRC_{soil, eco}$ is calculated as the geometric mean of the chronic toxicity data as described by Van Herwijnen (2012). The $SRC_{soil, eco}$ is 13 mg/kg_{dwt} for Dutch standard soil with 10% organic matter.

3.11.3 Sediment

The $SRC_{sediment, fw, eco}$ for tributyltin is 0.027 mg/kg_{dwt} for Dutch standard sediment with 10% organic matter.

3.12 Triphenyltin (TPT)

The ERLs for triphenyltin, expressed for the TPT-cation, are adopted as the ERLs for all triphenyltin compounds. The ERLs for triphenyltin are cited from Van Herwijnen (2012) and Van Herwijnen et al. (2012).

3.12.1 Water

The $SRC_{aquatic, eco}$ for the triphenyltin-cation is 0.10 µg/L. This value is valid for the freshwater and the saltwater environment. For groundwater the $SRC_{gw, eco}$ is 0.40 µg/L.

3.12.2 Soil

The $SRC_{soil, eco}$ is calculated as the geometric mean of the chronic toxicity data as described by Van Herwijnen (2012). The $SRC_{soil, eco}$ is 68 mg/kg_{dwt} for Dutch standard soil with 10% organic matter.

3.12.3 Sediment

The $SRC_{sediment, fw, eco}$ for triphenyltin is 0.0022 mg/kg_{dwt} for Dutch standard sediment with 10% organic matter.

3.13 *Cis*- and *trans*- 1,2-dichloroethene

The ERLs are cited from Fleuren et al. (2009) and from De Jong et al. (2007). In De Jong et al. (2007) ERLs are reported the *cis* and *trans* isomer separately as well as for 1,2-dichloroethene in general. In Fleuren et al. (2009) only ERLs for 1,2-dichloroethene in general are reported. The ERLs for 1,2-dichloroethene in general are sum ERLs, which are valid for the sum of the concentrations of the two isomers. All ERLs have been derived on the basis of the same water ERLs using equilibrium partitioning. The soil ERLs for the *cis* and *trans* isomer differ from each other because toxicity studies were available for the different isomers.

Since *cis*- and *trans*-1,2-dichloroethene are not naturally occurring compounds, a background concentration is not relevant for these substances.

3.13.1 Water

The sum $SRC_{fw, eco}$ for *cis*- and *trans*-1,2-dichloroethene is 1.07×10^4 µg/L, rounded this value is 1.1×10^4 µg/L. This value is valid for the freshwater and the saltwater environment and is also valid for groundwater.

3.13.2 Soil

The $SRC_{soil, eco}$ for *cis*-1,2-dichloroethene is 31 mg/kg_{dwt}. The $SRC_{soil, eco}$ for *trans*-1,2-dichloroethene is 44 mg/kg_{dwt}. The sum $SRC_{soil, eco}$ for 1,2- dichloroethene is 37 mg/kg_{dwt} (sum = geometric mean of SRCs of the individual compounds). These values are expressed for Dutch standard soil with 10% organic matter.

3.13.3 Sediment

In De Jong et al. (2007) ERLs for sediment were derived. However, Fleuren et al. (2009) concluded that derivation of ERLs for sediment are not required since the log $K_{p, susp\text{-}water}$ of 1,2-dichloroethene is below the trigger value of 3. Therefore, the sediment ERLs were not taken over by Fleuren et al. (2009) and are not taken over in this report as well.

3.14 Free cyanide

3.14.1 Water

The ERLs for free cyanide in water are cited from Verbruggen et al. (2001). The method of ERL derivation (NC, MPC and SRC_{eco}) is also described in the cited report. Free cyanide is also a priority substance under the Water Framework Directive (WFD). For this framework aquatic quality standards are being derived (EC, in preparation). This derivation is however not finalised, awaiting the results from some additional tests on ecotoxicity. The preliminary quality standards are given below as additional value. The $SRC_{fw, eco}$ for free cyanide is 31 µg/L, this value is also valid for groundwater. For the saltwater environment no $SRC_{sw, eco}$ has been derived.

3.14.2 Soil

No ERLs for free cyanide in soil have been derived by Verbruggen et al. (2001) since no suitable terrestrial toxicity data were available. An update of the ecotoxicological literature has not been performed. The $SRC_{soil, eco}$ for free cyanide, calculated using equilibrium partitioning and the log Koc for HCN is 0.04 mg/kg_{dwt}. This value is expressed for Dutch standard soil with 10% organic matter.

3.14.3 Sediment

In Verbruggen et al. (2001) no ERLs for free cyanide in sediment have been derived. In the derivation for the WFD, derivation of quality standard for sediment is not considered relevant because the log Koc is smaller than 3.

3.15 Thiocyanate

The ERLs are cited from Verbruggen et al. (2001). The method of ERL derivation (NC, MPC and SRC_{eco}) is also described in the cited report. An update of the toxicological literature was not performed. No experimental data for BCFs are found and BCF estimates using EPISuite (US EPA 2009) range from 1.1 to 3.2 L/kg.

3.15.1 Water

The $SRC_{fw, eco}$ for thiocyanate is 1.07 x 10^4 µg/L, this value is also valid for groundwater. For the saltwater environment no SRC_{eco} has been derived.

3.15.2 Soil

The $SRC_{soil, eco}$ for thiocyanate is 620 mg/kg_{dwt}. The $SRC_{soil, eco}$ is based on the result of a single acute toxicity study with a terrestrial species (AF = 10). A comparison with the $SRC_{soil, eco}$ derived for freshwater organisms could not be performed due to the lack of distribution coefficients. Based on the characteristics of the substance, this value has not been normalised to Dutch standard soil with 10% organic matter.

3.15.3 Sediment

In Verbruggen et al. (2001) no ERLs for thiocyanide in sediment have been derived.

3.16 Chloride

The ERLs for chloride are cited from Verbruggen et al. (2008). MPC derivation was performed following Van Vlaardingen and Verbruggen (2007). The added risk approach, generally used for naturally occurring compounds, is not applicable to chloride.

3.16.1 Water

The $SRC_{fw, eco}$ for chloride is 570 mg Cl^-/L. This value includes the natural background concentration and is valid for fresh surface water and groundwater.

3.16.2 Soil

The $SRC_{soil, eco}$ for chloride is 390 mg Cl^-/kg soil. This value is only valid for soils that have not been influenced by brackish or salt waters. Based on the characteristics of the substance, this value has not been normalised to Dutch standard soil with 10% organic matter.

3.16.3 Sediment

The $SRC_{sediment, fw, eco}$ for chloride is 2100 mg Cl^-/kg_{dwt}. Based on the characteristics of the substance, this value has not been normalised to Dutch standard sediment with 10% organic matter.
For the saltwater environment, no $SRC_{sediment, sw, eco}$ has been derived.

3.17 Sulphate

ERLs for soil, groundwater and sediment are not available for sulphate (Brand, 2008). See also section 6.6 for additional information.

3.18 Summary of the environmental risk limits

Table 3.1: Environmental Serious Risk Concentrations (SRC_{eco}) for soil and groundwater.

Compound	$SRC_{soil, eco}$ [mg/kg_{dwt}]	$SRC_{gw, eco}$ [µg/L]
Metals and metalloids		
Antimony	1400	9600
Barium	400[a]	1.7×10^4
Boron	-[b]	7100[c]
Selenium	1.9[a]	130
Thallium	2.0	-
Tin (inorganic)	260[a]	-
Vanadium	105[a]	63
Organotin compounds		
Dibutyltin (DBT) - cation	28[d]	50
Tributyltin (TBT) - cation	13[d]	0.046
Triphenyltin (TPT) - cation	68[d]	0.40
Chlorinated hydrocarbons		
1,2-dichloroethene (sum)*	37	1.1×10^4
Cis-1,2-dichloroethene	31	-
Trans-1,2-dichloroethene	44	-
Inorganic compounds		
Free cyanide	0.04	31
Thiocyanate	620	1.1×10^4
Chloride	390[e]	5.7×10^5
Sulphate	-	-

- = not derived

[a] ERLs are possibly not protecting for secondary poisoning, although the current datasets are too small to draw conclusions.
[b] Not deterived since C_b for Boron is not available.
[c] This value is for fresh groundwater only. For brackish and salt groundwater the $MPC_{sgw, eco}$ is 3.6 mg/L and the SRC_{sgw}, eco is 10 mg/L.
[d] ERLs are not protecting for secondary poisoning.
[e] This value is only valid for soils that have not been influenced by brackish or salt waters
* sum = geometric mean of the SRCs of the individual compounds.

4 Integration of human and environmental risk assessment

4.1 Introduction

In this chapter the integrated risk limits (integrated SRCs) for soil and groundwater are presented. These values are proposed for Intervention Values. The presented risk limits in sections 2.15 and 3.18 are used for this purpose.

4.1.1 Integration

The evaluation as described in this report is done in accordance with the procedure as described by Lijzen et al. (2001). Figure 4.1 shows an overview of the method to derive an integrated risk limit for soil and groundwater.

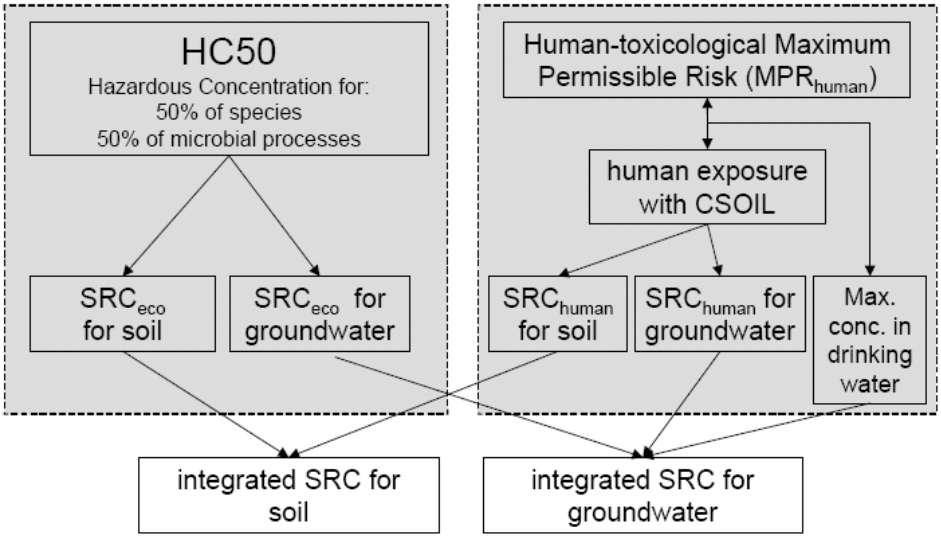

Figure 4.1: Diagram of the derivation of risk limits (integrated SRC) for soil and groundwater (Lijzen et al. 2001).

To summarize, for both groundwater and soil an integrated SRC is derived based on human-toxicological and ecotoxicological risk assessment. For the SRC_{eco} the HC50 (chapter 3) is the basis. To derive the SRC_{human}, the MPR_{human} (chapter 2) together with the exposure model CSOIL2000 is used (Brand et al., 2007. Basically, the lower value of both the SRC_{eco} and SRC_{human} is chosen as the integrated SRC. However, occurrence in soil and groudwater and reliability of the derived value are considered as well. In these cases expert judgement was used to make a definitive proposal. This was for example done with the proposals for tributyltin and triphenyltin (see also chapter 6).

4.2 Evaluated input CSOIL 2000

The SRC_{human} is calculated with the exposure model CSOIL 2000. It is worth mentioning that in contrast to the evaluation of the Intervention Values in 2001, for the current evaluation of the Intervention Values a new CSOIL 2000 model (updated in 2006) is used. An elaborate description of this new model is given in report by Brand et al. (2007). Relevant exposure routes that are included in the risk assessment are:

- soil ingestion;
- dermal contact indoors and outdoors;

- soil inhalation;
- crop consumption;
- inhalation of vapours indoors and outdoors;
- inhalation of vapours during showering/bathing;
- dermal contact during showering or bathing.

For the derivation of the SRC_{human} the default user scenario 'Residential with garden' is used.

As part of the evaluation, literature was reviewed for the compound specific parameters values for BCF and Kp. The review only focussed on these critical parameters. These parameter values are relevant as input for the CSOIL 2000 model.

4.2.1 Bioconcentration Factor for plants (BCF_{crop})

The BCF_{crop} as used in the integration process for Intervention Values is defined as 'the ratio of the metal in the edible part of the crop and the total metal concentration in the soil' (Otte et al., 2001). BCFs for larger animals or soil organisms are not used for this purpose.

The BCFs for uptake in crops currently in use date back to 1994-1998. A minor literature review was performed, prioritizing the compounds for which uptake by crops is a major exposure pathway. Besides a literature review, information was gathered from other institutes (Alterra in Wageningen and Vito in Belgium).

Only new data were found for barium to derive a new BCF for leafy vegetables. The new BCF is based on a research of Römkens and Rietra (2011). Table A3.2 in Annex 3 presents an overview of the BCFs as used in this evaluation.

4.2.2 Soil-water partition coefficient for metals (Kp)

The soil water partition coefficient (Kp) describes the partitioning of a compound over two phases. The current Kp values date back to 1994-1998. Just as the BCFs, a minor literature review was performed.

In 2006 Verschoor et al. performed a major literature review including several compounds relevant in this report. Data from this review were used for the current evaluation. Furthermore, Sauve et al. (2000) reported several average Kd values in mineral soils. Finally, Herwijnen en Smit reported a new Kp value for Boron in 2010. If no new data were present, the current Kp was used. Table A3.8 in Annex 3 presents an overview of the Kp values as used in this evaluation.

4.3 Summary of the derivation of proposed Intervention Values

Table 4.1 presents the SRCs for soil based on ecotoxicological and human-toxicological risk assessment, together with the current and the new proposed Intervention Values or indicative values for soil. Table 4.2 presents the same information for groundwater.

Table 4.1: Current intervention values and indicative values for soil (VROM, 2009), new derived ecotoxicological serious risk concentrations (SRC_{eco}), human toxicological serious risk concentrations and the proposed intervention values soil in mg/kg_{dwt}. The lowest SRC-values are underlined.

Compound	**Current Intervention Values[a] or indicative values[b] for soil [mg/kg_{dwt}]**	**Proposed intervention values for soil [mg/kg_{dwt}]**	**SRC_{human} for soil [mg/kg_{dwt}]**	**SRC_{eco} for soil incl. background concentration [mg/kg_{dwt}]**	**Background concentration standard soil [mg/kg_{dwt}] (INS**/AW 2000)**
Metals and metalloids					
Antimony	22[a]	**150**	152	1400	3/4
Barium	-	**400**	9235	400[e]	155/190
Boron	-	**3100**	3085	- [c]	n.a.
Selenium	100[b]	**5.9**	323	5.9[e]	0.7/ 4
Thallium	15[b]	**2.5**	8.7	2.5	1.0/ 1.5
Tin (inorganic)	900[b]	**260**	1.5×10^5	260[e]	19/6.5
Vanadium	250[b]	**110**	1245	105[e]	42/80
Organotin compounds					
Organotin compounds (sum)*	2.5[a]	**-**	-	-	n.a./0.15
Dibutyltin (DBT)	-	**28**	150	28[j]	n.a.
Tributyltin (TBT)	-	**13**	54[f]	13[j]	n.a./0.065
Tributyltin chloride	-	**-**	0.5[h]	-	n.a.
Tributyltin hydride	-	**-**	0.003[g]	-	n.a.
Tributyltin oxide	-	**-**	*54*	-	n.a.
Triphenyltin (TPT)	-	**57[i]**	-	68[j]	n.a.
Triphenyltin acetate	-	**-**	*9.3*	-	n.a.
Triphenyltin chloride	-	**-**	95	-	n.a.
Triphenyltin hydroxide	-	**-**	57	-	n.a.
Chlorinated hydrocarbons					
1,2-Dichloroethene (sum)*	1.0[a]	**3.0**	3.0	37	n.a./0.3
Cis-1,2-dichloroethene	-	-	2.7	31	n.a.

Compound	Current Intervention Values[a] or indicative values[b] for soil [mg/kg$_{dwt}$]	Proposed intervention values for soil [mg/kg$_{dwt}$]	SRC$_{human}$ for soil [mg/kg$_{dwt}$]	SRC$_{eco}$ for soil incl. background concentration [mg/kg$_{dwt}$]	Background concentration standard soil [mg/kg$_{dwt}$] (INS**/AW 2000)
Trans-1,2-dichloroethene	-	-	3.4	44	n.a.
Inorganic compounds					
Free cyanide	20[a]	**0.04**	70/43[k]	0.04	n.a./3.0
Thiocyanate	20[a]	**33[k]**	8914/33[k]	620	n.a./6.0
Chloride	-	**390**	-	390[d]	n.a.
Sulphate	-	**-**	-	-	n.a.

n.a. = not available
- = not derived.
a Current Intervention Values (in 'Soil remediation Circulair 2009').
b Current indicative values (in 'Soil remediation Circulair 2009').
c Not derived since Cb for Boron is not available.
d This value is only valid for soils that have not been influenced by brackish or salt waters.
e ERLs are possibly not protecting for secondary poisoning, although the current datasets are too small to draw conclusions.
f This value is based on the TBTO because this compound is mostly found in the environment.
g Although TBT hydride is the most toxic compound, this compound is rarely found in the environment.
h This compound is rarely found in the environment.
i This value is based on the TPTOH because the other derivatives are hydrolised into TPTOH if they come into contact with water; it is therefore assumed that TPTOH is mostly found in the environment.
j ERLs are not protecting for secondary poisoning.
k In case of acute exposure to a child with 5 g soil ingestion and without crop consumption.
* sum = geometric mean of the SRCs of the individual compounds.
** Crommentuijn et al. 1997.

Table 4.2: Current intervention values and indicative values for groundwater (VROM, 2009), new derived ecotoxicological serious risk concentrations (SRC$_{eco}$), human toxicological serious risk concentrations and the propsed intervention values groundwater in µg/L. The lowest SRC-values are underlined.

Compound	Current Intervention Values[a] or indicative values[b] for groundwater [µg/L]	Proposed Interven-tion Values for ground-water [µg/L]	SRC$_{human}$ for ground water [µg/L]	SRC$_{human}$ for ground water used as drinking water [µg/L]	SRC$_{eco}$ for groundwater (incl. background concentra-tion) [µg/L]	Background concentration groundwater deep (> 10m) [µg/L] (INS**)
Metals and metalloids						
Antimony	20[a]	**190**	1780	188	9600	0.09
Barium	625[a]	**630**	3690	628	1.7×10^4	200
Boron	-	**6300**	3.0×10^5	6280	7100[c]	262

Compound	Current Intervention Values[a] or indicative values[b] for groundwater [µg/L]	Proposed Intervention Values for groundwater [µg/L]	SRC_{human} for groundwater [µg/L]	SRC_{human} for groundwater used as drinking water [µg/L]	SRC_{eco} for groundwater (incl. background concentration) [µg/L]	Background concentration groundwater deep (> 10m) [µg/L] (INS**)
Selenium	160[b]	**130**	2.1×10^4	157	130	0.02
Thallium	7[b]	**1.3**	55	1.3	-	<2
Tin (inorganic)	50[b]	**6300**	7.8×10^4	6280	-	<2
Vanadium	70[b]	**70**	4020	63	63	1.2
Organotin compounds						
Organotin compounds (sum)*	0.7[a]	**-**	**-**	-	-	n.a.
Dibutyltin (DBT)	-	**8.0**	62	7.9	50	n.a.
Tributyltin (TBT)	-	**0.05**	45[f]	7.9[f]	0.046	n.a.
Tributyltin chloride	-	-	0.23[e]	7.9	-	n.a.
Tributyltin hydride	-	-	0.005[d]	7.9	-	n.a.
Tributyltin oxide	-	-	45	7.9	-	n.a.
Triphenyltin (TPT)	-	**0.4**	98[g]	7.9	0.40	n.a.
Triphenyltin acetate	-	-	80	7.9	-	n.a.
Triphenyltin chloride	-	-	26	7.9	-	n.a.
Triphenyltin hydroxide	-	-	98	7.9	-	n.a.
Chlorinated hydrocarbons						
1,2-dichloro-ethene (sum)*	20[a]	**940**	963	942	1.1×10^4	n.a.
Cis-1,2-dichloro-ethene	-	-	1040	942	-	n.a.
Trans-1,2-dichloro-ethene	-	-	892	942	-	n.a.
Inorganic compounds						
Free cyanide	1500[a]	**31**	6.1×10^4/ 3.9×10^4 [h]	1570/1570[h]	31	n.a.

Compound	**Current Intervention Values[a] or indicative values[b] for groundwater [µg/L]**	**Proposed Intervention Values for groundwater [µg/L]**	**SRC_{human} for groundwater [µg/L]**	**SRC_{human} for groundwater used as drinking water [µg/L]**	**SRC_{eco} for groundwater (incl. background concentration) [µg/L]**	**Background concentration groundwater deep (> 10m) [µg/L] (INS**)**
Thiocyanate	1500[a]	**350**	3.6×10^7/ 1.3×10^5 [h]	346/346[h]	1.1×10^4	n.a.
Chloride	-	**5.7×10^5**	-	-	5.7×10^5	n.a.
Sulphate	-	-	-	-	-	n.a.

n.a. = not applicable
- = not derived.
a Current Intervention Values (in 'Soil remediation Circulair 2009').
b Current indicative values (in 'Soil remediation Circulair 2009').
c This value is for fresh groundwater only. For brackish and salt groundwater the $MPC_{gw, eco}$ is 3.6 mg/L and the SRCgw,eco is 10 mg/L.
d Although TBT hydride is the most toxic compound, this compound is rarely found in the environment.
e This compound is rarely found in the environment.
f This value is based on the TBTO because this compound is mostly found in the environment.
g This value is based on the TPTOH because the other derivatives are hydrolised into TPTOH in water. Therefore is assumed that TPTOH is mostly found in the environment.
h In case of acute exposure to a child with 5 g soil ingestion and without crop consumption.
* sum = geometric mean of the SRCs of the individual compounds.
** Crommentuijn et al. 1997.

5 Secondary poisoning in the terrestrial compartment

5.1 Introduction

Besides direct toxicity, secondary poisoning is a relevant subject in risk assessment. However, in the procedure followed in chapter 3 for the derivation of soil quality standards, secondary poisoning is not included. Therefore, the soil quality standards derived in this report are based on direct toxicity only. For deriving soil quality standards for soil management, secondary poisoning is described in this chapter. This information can be used for derivation of maximal values in the precautionary framework if necessary. In section 5.2 the methodology followed for the metals is reported. In section 5.3 the results for the metals are reported. In section 5.4 the relevance of secondary poisoning for the non-metal compounds is reported.

5.2 Model and method for metals

5.2.1 Introduction

Current INS methodology for ERL derivation for the soil compartment includes derivation of a standard considering secondary poisoning if this is deemed relevant. In the ERL derivation in chapter 3 the need to include bioconcentration for derivation of terrestrial ERLs was not investigated. In this chapter for all compounds is reported whether secondary poisoning is relevant.

For most compounds the necessary information could be cited from the report on which the original ERLs derivation was reported. For the metals vanadium, tin, barium and the metalloid selenium however, a literature search was performed to collect information on bioaccumulation of these metals. The scientific literature was searched by means of the databases TOXLINE period 1985-2001 and CURRENT CONTENTS period 1997-2007. In addition, for these compounds the internet was screened for information on this topic.

The outcome of the literature search shows that experimental data on bioaccumulation in terrestrial organisms of the metals regarded in this report are very scarce. It was not possible to assess whether bioaccumulation and secondary poisoning is relevant. A different procedure was therefore followed, based on the methodology described in Van Vlaardingen and Verbruggen (2007). This methodology, which determines if secondary poisoning for the metals could be more critical than direct toxicity, is described in the following sections. See also Figure 5.1 for a schematic overview of the assessment of secondary poisoning.

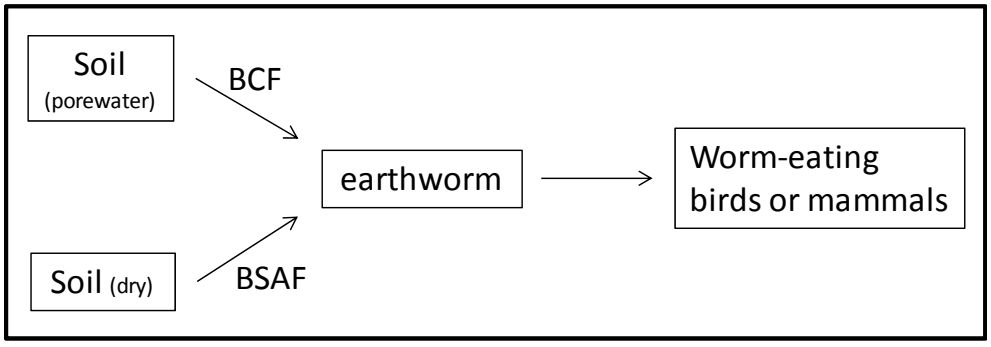

Figure 5.1: Assessment of secondary poisoning.

5.2.2 *Model and equations*

In the EU technical guidance document (EU-TGD; EC (JRC), 2003), secondary poisoning in the terrestrial ecosystem is modelled as: soil → earthworm → mammal/bird. This methodology is also implemented in the REACH risk assessment framework. The model and equations from the EU-TGD have been implemented in INS guidance (Van Vlaardingen and Verbruggen, 2007); EU-TGD equations were rearranged and calculation of both a $BCF_{earthworm}$ and a $BSAF_{earthworm}$ is possible. The equations used are displayed below. Note that the BCF concept as included in the EU-TGD has been developed for (neutral) hydrophobic organic substances (McGeer et al., 2003; Jager, 2003) which may limit the applicability of this earthworm bioaccumulation model to metals. First, equation nr. 82c (page 132) from the EU-TGD is cited:

$$C_{\text{earthworm}} = \frac{BCF_{\text{earthworm}} \cdot C_{\text{porewater}} + C_{\text{soil}} \cdot F_{\text{gut}} \cdot CONV_{\text{soil}}}{1 + F_{\text{gut}} \cdot CONV_{\text{soil}}} \qquad \textit{Equation 1}$$

An explanation of the parameters and units in this and following equations is given in Table 5.1. In EU-TGD modelling, $C_{porewater}$ is related to C_{soil} via equilibrium partitioning. The relationship between these two variables is given in Equation 67 from the EU-TGD (EU-TGD Equation. 67, page 85):

$$C_{\text{porewater}} = C_{\text{soil}} \cdot \frac{RHO_{\text{soil}}}{K_{\text{soil-water}} \cdot 1000} \qquad \textit{Equation 2}$$

Note that C_{soil} in these equations is expressed in mg/kg_{ww}, i.e. per kilograms of wet weight soil. The conversion factor $CONV_{soil}$ (in kg_{ww}/kg_{dwt}) is used to convert concentrations in wet weight soil to dry weight soil.

Several parameters (e.g. $CONV_{soil}$, RHO_{soil}) have default values which are listed in Table 5.1. The derivation of these parameters is based on other equations. These equations will not be cited here, but the reader is referred to the EU-TGD for more detail.

Equation 2 was substituted in equation 1 and the resulting equation rearranged to give equation 3. This equation is cited from the INS guidance (Van Vlaardingen and Verbruggen, 2007). Note that some variables have been renamed to be consistent with INS terminology:

C_{soil} has been replaced by $MPC_{sp,\ soil,\ EU\text{-}TGD}$ (mg/kg_{dwt}). The latter variable denotes the 'maximum permissible concentration in soil (MPC_{soil}) derived via secondary poisoning based on the EU-TGD model'. In the case of metals, as discussed here, when the added risk approach is followed, MPC should be replaced by MPA (maximum permissible addition).

$C_{earthworm}$ has been replaced by $MPC_{oral,\ min}$ (mg/kg_{food}). The latter variable denotes the lowest available concentration in food of the predator that does not exert negative effects. In practice, the $MPC_{oral,\ min}$ is a NOAEL expressed in mg/kg_{fd}, derived from toxicity studies with mammals or birds. In the model used here, the predator's food is represented by the earthworm.

$$MPC_{\text{sp, soil,TGD}} = \frac{MPC_{\text{oral, min}} \cdot (1 + F_{\text{gut}} \cdot CONV_{\text{soil}})}{BCF_{\text{earthworm}} \cdot \frac{RHO_{\text{soil}}}{K_{\text{soil-water}} \cdot CONV_{\text{soil}} \cdot 1000} + F_{\text{gut}}} \qquad \textit{Equation 3}$$

$K_{soil\text{-}water}$ is calculated according to:

$$K_{\text{soil}-\text{water}} = F\text{air}_{\text{soil}} \times K_{\text{air}-\text{water}} + F\text{water}_{\text{soil}} + F\text{solid}_{\text{soil}} \times \frac{K\text{p}_{\text{soil}}}{1000} \times RHO\text{solid}$$

Equation 4

This is cited from the EU-TGD (equation 24, page 47). For the metals considered here, $K_{air\text{-}water}$ was set to zero and for $K_{p,\ soil}$, values used in the ERL derivation of the metals considered will be used (Van Vlaardingen et al. 2005).

Experimentally determined bioaccumulation factors in earthworms may be expressed as a BCF.
The BCF is the ratio of concentration in earthworm over the concentration in pore water in units of $L/kg_{wet\ earthworm}$.

$$BCF_{\text{earthworm}} = \frac{C_{\text{earthworm}}}{C_{\text{porewater}}}$$

Equation 5

Biota to soil accumulation factors (BSAF or BAF) are also regularly reported. The BSAF is the ratio of the concentration in wet earthworm over the concentration in dry soil in units of $kg_{dw\ soil}/kg_{ww\ earthworm}$. Note that experimentally determined BSAFs may also be expressed in $kg_{dw\ soil}/kg_{dw\ earthworm}$.

$$BSAF_{\text{earthworm}} = \frac{C_{\text{earthworm}}}{C_{\text{soil}} \cdot CONV_{\text{soil}}}$$

Equation 6

Combining Equation 2, 5 and 6 gives:

$$BSAF_{\text{earthworm}} = BCF_{\text{earthworm}} \cdot \frac{RHO_{\text{soil}}}{K_{\text{soil}-\text{water}}.CONV_{\text{soil}} \cdot 1000}$$

Equation 7

This can be substituted in Equation 3 to give:

$$MPC_{\text{sp, soil, TGD}} = \frac{MPC_{\text{oral, min}} \cdot (1 + F_{\text{gut}} \cdot CONV_{\text{soil}})}{BSAF_{\text{earthworm}} + F_{\text{gut}}}$$

Equation 8

Table 5.1: List of defaults and variables used in Equations.

Symbol	Description of variable	Unit	Default value
-	Conversion factor from m^3 to litre	L/m^3	1000
$BSAF_{earthworm}$	Biota (earthworm) to soil accumulation factor	kg_{dwt}/kg_{ww}	
$BCF_{earthworm}$	Bioconcentration factor for earthworm on wet weight basis	L/kg_{ww}	
$C_{earthworm}$	Concentration in porewater corresponding with C_{soil} via EqP	mg/L	
$CONV_{soil}$	Conversion factor for soil concentration wet-dry weight soil	kg_{ww}/kg_{dwt}	1.13
$C_{porewater}$	Concentration in porewater corresponding with C_{soil} via EqP	mg/L	
C_{soil}	Concentration in soil corresponding with $C_{porewater}$ via EqP	mg/kg_{ww}	
$Fair_{soil}$	Fraction air in soil	m^3/m^3	0.2

Symbol	Description of variable	Unit	Default value
F_{gut}	Fraction of gut loading in earthworm	kg_{dwt}/kg_{ww}	0.1
$Fsolid_{soil}$	Fraction solids in soil	m^3/m^3	0.6
$Fwater_{soil}$	Fraction water in compartment soil	m^3/m^3	0.2
$K_{air\text{-}water}$	Air-water partition coefficient	m^3/m^3	
Kp_{soil}	Solids-water partition coefficient in soil	m^3/kg^1	
$K_{soil\text{-}water}$	Soil-water partition coefficient	mg/m^3	
$MPC_{oral,\ min}$	Lowest MPC_{oral} derived from toxicity studies with mammals and/or birds	mg/kg_{food}	
$MPC_{sp,\ soil,\ EU\text{-}TGD}$	Maximum permissible concentration in soil based on secondary poisoning using EU-TGD soil characteristics	mg/kg	
RHO_{soil}	Bulk density of wet soil	kg_{ww}/m^3	1700
RHOsolid	Density of the solid phase	kg_{solid}/m_{solid}^3	2500

5.2.3 *Calculation of criticalBCF and criticalBSAF for metals*

Departing from a non-observed adverse effect concentration (NOAEL) for metal X in a mammal, it can be estimated which bioconcentration factor in the food of the mammal would be necessary in order to obtain a concentration in soil below the current environmental risk level (MPC_{soil} or $SRC_{soil,\ eco}$). Again, note that we use the model (Equations 3 and 8) to calculate BCF and BSAF. The equations were programmed in Microsoft Excel® and a build-in iteration routine was used to estimate BCF and BSAF which would result in a MPA_{soil} or SRA_{soil} lower than the values based on direct toxicity. To emphasise that the BCF and BSAF values calculated here serve indicative purposes only, they will be expressed as $criticalBCF^{est}$ and $criticalBSAF^{est}$.

In order to calculate bioaccumulation factors, the following parameters are needed:

- NOAEL for birds or mammals. Searching and evaluating these data is laborious and has not been performed for the exploratory investigation carried out here. Instead, the NOAEL underlying the human-toxicological risk limit has been used. Because these data are well evaluated, for protecting human health, they can serve as screening values to protect terrestrial mammals living in and on the soil. The NOAELs are generally conservative estimates, since for the protection of human health, sublethal effects are often considered. These effects are not deemed relevant for the assessment of secondary poisoning where effects like mortality, reproduction and growth are considered key endpoints. Since threshold limits for humans are generally based on mammalian data, it should be kept in mind that toxicity data on birds are not taken into consideration using this approach. As stated above, complete retrieval and evaluation of mammalian and avian toxicity data was not part of this project. The NOAEL, expressed in $mg/kg_{bw}/d$, will be recalculated into a concentration in food using the methodology from the EU-TGD (EC (JRC), 2003) which is also used within the Dutch INS framework. A daily food conversion factor is applied to convert the NOAEL from $mg/kg_{bw}/d$ to mg/kg_{food} and an assessment factor is applied (its height depending on study type) to cover for extrapolation from lab to field, interspecies variation and subacute to chronic effects. The conversion factors and the assessment can be found in the INS guidance. The MPA_{soil} and SRA_{soil} are based on direct ecotoxicological data. These values are reported in the ERL derivation in RIVM Report 601501029 (Van Vlaardingen et al., 2005).

- For critical BCF^{est} calculation a soil-water partition coefficient (K_p) is needed. The K_p values used in the ERL derivation (Van Vlaardingen et al. 2005) has been used here.

5.2.4 Correction for the use of laboratory feed in bird and mammal test

In the assessment factors that are applied to use toxicity data for birds and mammals for the assessment of secondary poisoning, a generic factor of three is used. This correction factor is applied to correct for the difference in calorific value of the feed used in the laboratory trials in comparison to the feed consumed by wild animals in the field. This value is based on the consumption of fish for the assessment in aquatic ecosystems. This value is however also used for the assessment of worms in soil ecosystems and is currently under discussion, since the calorific value of earthworms is lower than that for fish. Based on this, the exposure through secondary poisoning in soil ecosystems might be underestimated using the factor of three. The factor of three is used for as long as no alternative value is decided upon; the assessments for secondary poisoning in soil ecosystems should be evaluated when a new value becomes available. Due to the linear relationship evaluation of secondary poisoning in soil ecosystems this can be done in a short time frame. The above applies to all organotin compounds.

5.2.5 Gut content in earthworm model

In the EU-TGD earthworm bioaccumulation model the concentration in the entire earthworm ($C_{earthworm}$) is composed of a concentration in the tissue and in the gut of the earthworm; the latter of which is assumed to be filled with soil. This seems reasonable since the predator eating the earthworm will ingest the worm plus its gut content. The calculated BCF or BSAF applies however to the earthworm tissue, i.e. the entire worm minus its gut content. This is in line with experimentally determined BSAFs where earthworms are allowed to excrete their gut content before their metal contents are analysed (e.g.Vijver et al. 2007; Hobbelen et al. 2006).

Note that the model BCF or BSAF are whole body values, irrespective of the uptake route, which is not included in the model. Model calculations used here are thus not influenced by uptake route. Information on the importance of some uptake routes of metals in earthworm species is available, although not for the metals investigated here. Vijver et al. (2003) and Saxe et al. (2001) have reported that copper, lead, cadmium and zinc uptake occurs predominantly via the dermal route. Gut uptake was maximally 30% for zinc and 17% for cadmium, but marginally for the other two metals. However, Hobbelen et al. (2006) pointed towards information (Morgan et al. 2004) that suggests the gut uptake to be important for cadmium.

5.2.6 Limitations

It should be noted that calculated bioaccumulation factors for metals should be used with care. Bioconcentration of metals by organisms is dependent on the external metal concentration; the bioconcentration factor often decreases with increasing external concentration.This was observed for several metals and for aquatic and terrestrial organisms (e.g. McGeer et al. 2003; Saxe et al. 2001; Veltman et al. 2007). Although this relationship is regularly observed, it is not easily explained. Many factors can influence metal uptake, internal 'handling' and excretion processes performed by organisms; in addition, these factors may also differ per metal depending on e.g. its physical and chemical properties (speciation, charge, ionic radius, etc.), the metal being essential or non-essential, etc. Various environmental parameters influence metal availability for

uptake, which in turn may differ for the gut environment compared to the external environment. Schlekat et al. (2007) is cited here as a reference addressing many of these topics. Consequently, BCFs and BSAFs (BAFs) for metals cannot be treated as point estimates (Smolders et al. 2007), as is usually done for organic compounds. This knowledge, combined with a lack of data on the metals investigated here when focussing on terrestrial ecosystems, seriously hampers straightforward interpretation of estimated BCFs of BSAFs.

A conservative approximation in the model calculations is that total concentrations accumulated in the earthworm are (bio)available to its predator and that the total concentration will contribute to toxicity, irrespective of the form in which the metal is present. This is the intrinsic assumption that is made when a concentration in earthworm is compared to a NOAEL for a wildlife species (mammal or bird), which is determined using laboratory studies, where animals are exposed to metals via the diet or by oral gavage dosage. Although this assumption is conservative and thereby provides an extra safety in the risk assessment, its magnitude cannot be estimated for the various metals.
The mentioned limitations should be kept in mind when evaluating the potential for bioaccumulation of these metals.

5.2.7 Interpretation

In the following sections criticalBCF^{est} and criticalBSAF^{est} will be calculated as indication for each metal. The level of these indicative values (criticalBCF^{est} and criticalBSAF^{est}) corresponds with the height of the MPA_{soil} based on direct ecotoxicity. CriticalBCF^{est} and criticalBSAF^{est} values corresponding with the SRA_{eco} has also been calculated. Exceeding this calculated, indicative BCF (or BSAF) means that the modelled secondary poisoning route will lead to a lower MPA_{soil} value (or $SRA_{soil, eco}$) than the MPA (SRA) value based on direct ecotoxicity. Please note that the presented criticalBCF^{est} and criticalBSAF^{est} values are calculated. These values should not be interpreted as experimental estimates of bioaccumulation. In view of the limitations mentioned in the previous section, we regard the outcomes as a rough screening only. With the information available here, a more accurate estimation is not possible. Very low indicative criticalBCF^{est} or criticalBSAF^{est}, which are likely to be reached in field situations, may be interpreted as 'there is a high possibility that an experimental BCF or BSAF will exceed this indicative value and therefore the possibility of secondary poisoning cannot be ruled out, further investigation is needed'.

Extremely high indicative criticalBCF^{est} or criticalBSAF^{est}, which are unlikely to occur in the field, may be interpreted as 'it is unlikely that an experimental BCF or BSAF will exceed this indicative value and therefore secondary poisoning in the field seems unlikely'.

5.3 Estimation of critical bioaccumulation levels for metals

5.3.1 Antimony

Bioaccumulation at the level of the MPA_{soil} and SRA_{soil}.
Van Leeuwen and Aldenberg (2011) have derived an MPC for secondary poisoning for soil of 1600 mg Sb/kg_{dwt}. Since this value is higher than the $MPA_{soil, eco}$, $MPC_{soil, eco}$, $SRA_{soil, eco}$ and $SRC_{soil, eco}$, secondary poisoning is not considered relevant for antimony.

5.3.2 Barium

Introduction
Virtually no data on bioaccumulation of barium in the terrestrial environment were found. Hope et al. (1996) determined BAF values from measured barium concentrations in soil, plants, insects, mice and rats, all collected at one location. BAFs (dry weight ratios of kg_{soil} over $kg_{organism}$) were 0.4, 0.2, 0.02 and 0.02 kg/kg for plants, terrestrial insects, mice and rats, respectively. Data for earthworms were not reported. Data for barium were not included in the work of Sample et al. (1998). A recent study on accumulation in earthworms from soil was retrieved (Nahmani et al., 2007). The study is summarised in the introduction on thallium. The BCF and BSAF values calculated from this study for Ba are 14.8±236 L/kg_{ww} (n = 10) and 0.036±0.049 kg_{dw}/kg_{ww} (n = 10).

Bioaccumulation at the level of the MPA_{soil}
The current MPC_{human} for Ba is based on human-toxicological data (Baars et al., 2001, Van Engelen et al. 2008). The endpoints of the NOAELs for mammalian species reported in Baars et al. are not closely related to population effects and are not useful for estimation of secondary poisoning. Hope et al. (1996) and Sample et al. (1996) both refer to the same study on rats, that gave the lowest NOAEL of 5.1 $mg/kg_{bw}/d$ for the endpoint growth in a chronic (>1 year) study. The K_p soil:water for barium is 2512 L/kg (log K_p =3.40). The NOAEL of 5.1 $mg/kg_{bw}/d$ is recalculated to an MPC_{oral} of 3.4 mg/kg_{food}, using a conversion factor of 20 g_{bw} d/g_{food} and an assessment factor of 30 (EU-TGD). With this MPC_{oral} a $criticalBCF^{est}$ of 909 L/kg is calculated as critical value. This means that when barium exhibits BCF values higher than this value, secondary poisoning may result in an MPA_{soil} value which is lower than the MPA_{soil} based on direct toxicity (8.2 mg/kg). This $criticalBCF^{est}$ can be recalculated to a $criticalBSAF^{est}$ of 0.36 $kg_{dwt\ soil}/kg_{ww\ worm}$ using the equations from the TGD. Assuming a fraction water of 0.84 for earthworms (Jager, 2003) or a correction factor of 0.16 $kg_{dwt\ worm}/kg_{ww\ worm}$, the $criticalBSAF^{est}$ becomes 2.25 kg/kg based on dry weight ratios.

Both the BCF (14.8 L/kg) and BSAF (0.036 kg_{dw}/kg_{ww}) values of Nahmani et al. (2007) are lower than the respective $criticalBCF^{est}$ of 909 L/kg and $criticalBSAF^{est}$ of 0.36 kg/kg. It should be kept in mind that the experimental data are based on a single study with an earthworm species that is not a soil representative species. The few BAF values reported for Barium (see the introduction) confirm this observation as they are also lower than the $criticalBSAF^{est}$. Based on the present, a limited data set, it can be tentatively excluded that secondary poisoning of earthworms will lead to a lower MPC_{soil} than the MPC_{soil} for direct toxicity.

Bioaccumulation at the level of the SRA_{soil}

Calculation of a $SRC_{eco,\ oral}$ (average estimate for mammalian or avian toxicity at NOAEL level) is hardly possible. Sample et al. (1996) have reported estimated NOAEL values for several wildlife species which were however all based on the rat NOAEL of 5.1 $mg/kg_{bw}/d$ used to derive the MPC_{oral}. These data are therefore not used. The endpoint of 5.1 $mg/kg_{bw}/d$ is recalculated to an MPC_{oral} applying a conversion factor of 20 to convert to a concentration in food, an assessment factor of 3 to correct for laboratory feed. The MPC_{oral} from this study is 34 mg/kg_{food} A second study with rats reported by Sample et al. (1996) gives a NOAEL of 209 $mg/kg_{bw}/d$ of $BaCl_2$ (endpoint mortality), which is 138 $mg/kg_{bw}/d$ of barium. Conversion of this value using the same assessment factors and an additional assessment factor to correct from subchronic to chronic gives an MPC_{oral} of 307 mg/kg_{food}. Sample et al. (1996) also summarized a study with 1-day old chicks exposed for 4 weeks. The NOAEL from this study is 2000 mg/kg_{food}. Correction for the use of laboratory feed and correction from subchronic to chronic gives an MPC_{oral} for chicks of 222 mg/kg_{food}. In addition to Sample et al. (1996), ATSDR (2007) gives a comprehensive overview of available NOAELs from toxicity studied with mammals. The most sensitive chronic endpoints given are a rat NOAEL of 15 mg/kg_{bw}/day for reproduction from a 68 week study and a mouse NOAEL of 160 mg/kg_{bw}/day for reproduction from a 2 year study. Conversion to a concentration in food (factor 20 or 8.3 for rats and mice respectively) and correction for laboratory feed (assessment factor 3) gives NOAELS of 100 mg/kg_{food} and 443 mg/kg_{food} for rats and mice respectively. The geometric mean of the most sensitive endpoints for rats (34 mg/kg_{food}), mice (443 mg/kg_{food}) and chicks (222 mg/kg_{food}) is 150 mg/kg_{food}. This value is used as $SRC_{eco,\ oral}$.

With these data a $criticalBCF^{est}$ of 1750 L/kg or a $criticalBSAF^{est}$ of 0.7 kg_{dwt}/kg_{ww} are calculated. This means that when barium exhibits higher BCF or BSAF values in the field, secondary poisoning may result in a SRC_{soil} value which is lower than the SRA_{soil} based on direct toxicity (210 mg/kg_{soil}). The BCF (14.8 L/kg) and BSAF (0.036 kg_{dw}/kg_{ww}) values of Nahmani et al. (2007) are lower than these values. And as concluded above for the MPC, it can also be tentatively excluded that secondary poisoning of earthworms will lead to a lower SRC_{soil} than the SRC_{soil} for direct toxicity.

5.3.3 Boron

For boron the risk of secondary poisoning has been assessed in Van Herwijnen and Smit (2010) and therefore the procedure as described above has not been followed. Boron has a BCF < 100 L/kg, therefore an environmental risk through secondary exposure is not considered relevant.

5.3.4 Selenium

Introduction

Information on bioconcentration of selenium in the terrestrial environment is scarce. Several indications were found that effects of selenium on birds are observed. However, since these data were not reviewed for this project, it cannot be stated if the observed effects are the result of secondary poisoning and whether these effects occur in birds that obtain their feed from the terrestrial compartment. Data on uptake of selenium in higher (terrestrial) plants are also found. E.g. Efroymson et al. (2001) report a mean uptake factor (UF) (in $kg_{dwt\ soil}/kg_{dwt\ plant}$) of 2.25 (s.d. = 8.7). Estimates of bioaccumulation factors for earthworms are scarce. Sample et al. (1998) report a mean UF ($kg_{dwt\ soil}/kg_{dwt\ worm}$) of 1.8 (s.d. = 3.3).

Note that this value was based on data from a single study with 14 observations. UF's used by Efroymson et al. (2001) and Sample et al. (1996) are identical to BSAFs according to the definition: earthworm concentration/soil concentration. Since both these factors are determined under field conditions, they likely fulfil steady state conditions.

Bioaccumulation at the level of the MPA_{soil}

The current MPC_{human} for Selenium is based on human-epidemiological data. The endpoints of the NOAELs for mammalian species are not closely related to population effects and are not useful for estimation of secondary poisoning. Sample et al. (1996) summarised six studies, all with the endpoint reproduction. All studies have some drawbacks. One study is rejected since effects are noted not to be related to Se. The lowest NOAEL is 0.055 $mg/kg_{bw}/d$ (mouse) but this is based on a single concentration; consequently, the NOAEL may also be higher. This study will be used as additional information. A NOAEL of 0.076 $mg/kg_{bw}/d$ (3 generation mouse test) is based on one test concentration (LOAEL of 0.76 $mg/kg_{bw}/d$), to which an assessment factor of 10 was applied to derive the NOAEL.

Other studies resulted in higher NOAELs: 0.2 (rat, 2 generation), 0.21 (mouse, 48 day) and 0.46 (mouse 4 days during critical life stage) $mg/kg_{bw}/d$. In all studies, selenium was dosed via water, apart from the NOAEL of 0.46 $mg/kg_{bw}/d$ where oral gavage was used. However, the latter study is again based on a single concentration.

Summarising, the 3 generation mouse study giving the NOAEL 0.076 $mg/kg_{bw}/d$ will be used for an estimation of the critical BCF, because it is the lowest value. It is recognised that there are serious shortcomings to the test underlying this NOAEL (one concentration, exposure via water, NOAEL estimated from LOAEL by a factor of 10).

The K_p soil:water for selenium is 15.1 L/kg ($\log K_p$ =1.18). The NOAEL of 0.076 $mg/kg_{bw}/d$ is recalculated to an MPC_{oral} of 0.021 mg/kg_{food}, using a conversion factor of 8.3 $g_{bw}\ d/g_{food}$ and an assessment factor of 30 (EU-TGD). With this MPC_{oral}, a $criticalBCF^{est}$ of 60 L/kg or a $criticalBSAF^{est}$ of 3.9 kg_{dwt}/kg_{ww} are calculated. This means that when selenium exhibits higher BCF or BSAF values, secondary poisoning may result in an MPC_{soil} value which is lower than the MPA_{soil} based on direct toxicity (0.0058 mg/kg). The $criticalBSAF^{est}$ is based on a concentration in wet weight earthworm. Assuming a fraction water of 0.84 for earthworms (Jager, 2003) or a correction factor of 0.16 $kg_{dwt\ worm}/kg_{ww\ worm}$, the $criticalBSAF^{est}$ becomes 24 kg/kg based on dry weight ratios. This $criticalBSAF^{est}$ is higher than the uptake factor (UF) reported by Sample et al. (1998). This means that accumulation of selenium in earthworms using the UF of 1.8 kg/kg of Sample et al. (1996) based on dry weight ratios would not result in a slightly lower MPC than the MPC based on direct toxicity.

Although bioaccumulation data for selenium in terrestrial organisms are scarce, we tentatively conclude that the secondary poisoning of selenium will not lead to an MPC below the concentration of the MPA_{soil}. In addition, it must be stated that selenium is one of the few elements that is known to biomagnify, at least in some aquatic food chains (e.g. Stewart et al. 2004; Jarman et al. 1996; Ikemoto et al., 2008 but not in Campbell et al. 2005 and Anderson et al. 2010). This biomagnification has not been taken into account in the current exercise.

Bioaccumulation at the level of the SRA_{soil}

The information from the studies summarised by Sample et al. (1996) (see above) is used to calculate an $SRC_{eco,\ oral}$ based on the endpoint reproduction. Although all mouse endpoints are for reproduction and considered to be chronic because of exposure through gestation, preference is given to the endpoint of

the 3 generation study in which exposure was performed over a period of more than one year. The endpoints for mice and rats were recalculated to a NOAEL in mg/kg_{food} using conversion factors from the EU-TGD: 8.3 and 20 g_{bw} d/g_{food} for mice and rats respectively. An assessment factor of 3 was applied to each NOAEL for correction for laboratory feed; since both endpoints were chronic, no additional assessment factor is necessary. Resulting NOAELs are 0.21 and 1.3 mg/kg_{food}, with a geometric mean $SRC_{eco,\ oral}$ of 0.52 mg/kg_{food}.

With these data a $criticalBCF^{est}$ of 6 L/kg or a $criticalBSAF^{est}$ of 0.4 kg_{dwt}/kg_{ww} is calculated. This means that when selenium exhibits higher BCF or BSAF values in the field, secondary poisoning may result in a SRC_{soil} value which is lower than the SRA_{soil} based on direct toxicity (1.2 mg/kg_{soil}). As stated above, the $criticalBSAF^{est}$ is based on a concentration in wet weight earthworm. Assuming a fraction water of 0.84 for earthworms (Jager, 2003) or a correction factor of 0.16 $kg_{dwt\ worm}/kg_{ww\ worm}$, the $criticalBSAF^{est}$ becomes 2.5 kg/kg based on dry weight ratios. This $criticalBSAF^{est}$ is similar to the uptake factor (UF) reported by Sample et al. (1998), based on dry weight. This means that accumulation of Se in earthworms using the UF of 1.8 kg/kg of Sample et al. would not result in a lower MPC than the MPC based on direct toxicity.

Although bioaccumulation data for selenium in terrestrial organisms are scarce, we tentatively conclude that the secondary poisoning of Se will not lead to a SRC below the concentration of the SRC_{soil}.

5.3.5 Thallium

Introduction

A literature search on bioaccumulation factors (or data) of thallium in terrestrial organisms (preferably earthworms) yielded few results. However, a useful study on earthworm accumulation was retrieved (Nahmani et al. 2007); the set-up is described in short below.

Measurements of thallium (or a range of trace metals, including Tl) are regularly reported, but in most cases this concerns measurements in organisms which are not directly relevant or not directly useful to the present study, e.g. lichens, moss, mushrooms, bird feathers, fox, lizards, toads, etc. The vast majority of these monitoring studies report values in the organism without reporting the concentration in the environmental compartment, if applicable. Thallium concentrations in plants are also reported regularly. This is remarkable considering the uptake route soil → plant → plant eater is not incorporated as a 'secondary poisoning' route.

Soils from eight metalliferous sites and two control sites were collected. Earthworms *Eisenia fetida*, acclimatized in metal free soil, were placed onto the soils (12 per soil), kept at a constant temperature (20°C) with soils moistened up to 60% of MWHC without additional food. Each soil/worm combination had 18 replicates. Worms were removed from the soils at regular intervals up to 42 days. At day 42, soil, pore water and worms were analysed (ICP-OES) for 17 metals, including barium and thallium. Soils were aqua regia digested. Pore water was collected by centrifugation followed by 0.45 µm cellulose nitrate filtration. Worms were washed (deionised water) and allowed to empty their guts for 48 h in the dark on filter paper which was replaced every 12 hours to prevent coprophagy. Worms were dried at 60°C before digestion in nitric acid and subsequent analysed (Nahmani et al. 2007). Since metal concentrations were provided in dry weight worm only, concentrations were recalculated to wet weight using the dry:wet weight conversion factor of 0.16 kg_{dry}/kg_{wet} from Jager et al. (2003).

The resulting average BCF is 5.5 ± 2.7 L/kg_{wwt} (n = 8) and the average BSAF 0.018 ± 0.014 kg_{dw}/kg_{wwt} (n = 9).

It should be noted that *Eisenia fetida* is rather a compost-worm than a soil inhabiting species. Moreover, since the BCFs derived here are only based on one study, with worms that were not field collected, the results should be used with caution.

Bioaccumulation at the level of the MPA_{soil}

The current MPC_{human} for thallium is based on a 90 days drinking water study in rats (see Annex 1, chapter 3). The endpoint of the NOAEL used for derivation of the TDI for thallium is hair follicle atrophy. Although hair loss (alopecia) and related effects are characteristic for thallium toxicity, this endpoint is not relevant for secondary poisoning. A data search on metal toxicity studies with birds and mammals and endpoints relevant to secondary poisoning was not performed for the present study, as explained in the introduction. Therefore it is not possible to relate the height of the NOAEL to effect levels for population relevant endpoints for birds and/or mammals. This seriously hampers the predictive value of the method employed here for thallium. It should be noted that thallium has been used as rodenticide, but since unintended death of e.g. dogs, cats, badgers, martens, and foxes occurred, this use has been banned in many countries (Peter and Viraraghavan, 2005).

Although a comprehensive overview of effect levels on relevant endpoints for birds and mammals are missing, there are indications that thallium has a significant impact on small mammals in regions with elevated thallium soil concentrations, e.g. around zinc smelters (Dmowski et al. 2004, Wierzbicka et al. 2004).

The K_p soil:water for thallium is 464 L/kg (log K_p =2.20). The NOAEL of 0.04 $mg/kg_{bw}/d$ is recalculated to a MPC_{oral} of 0.0089 mg/kg_{food} using a conversion factor of 20 g_{bw} d/g_{food} and an assessment factor of 90 (EU-TGD). Entering this MPC_{oral} and the known MPA soil for direct toxicity of 0.1 mg/kg in the EU-TGD model for terrestrial bioaccumulation, any $BCF_{earthworm}$ will always lead to an MPC that is below 0.1 mg/kg. The terrestrial model for bioaccumulation (taken from the EU-TGD, part II, equation 82c (EC, 2003)) assumes that the gut content of an earthworm is filled with soil. This means that with a known threshold concentration in the worm of 0.0089 mg/kg_{food} (MPC_{oral}) and when there is no bioaccumulation (or even when biodilution occurs) of thallium, the concentration in soil cannot be higher than $MPC_{oral}/0.01015$ = 0.0990 mg/kg. As this is lower than the MPA for direct toxicity, this would mean that secondary poisoning would always result in a more critical MPC_{soil}.

From the study of Nahmani et al. (2007) both BCF and BSAF values were derived (see previous section). Calculating the concentration in soil ($MPC_{soil, sec\ pois}$) using these values and departing from the MPC_{oral} of 0.0089 mg/kg_{food} results in values of 0.089 mg/kg and 0.084 mg/kg. Both values are slightly lower than the current MPA_{soil} of 0.10 mg/kg, which is based on direct toxicity. This means that the MPA_{soil} based on direct toxicity would not be protective for secondary poisoning.

Since the MPC_{oral} for thallium is not based on a population relevant endpoint for predators, a reliable estimation of the MPC_{soil} based on secondary poisoning can not be made. However, it should be realised that although thallium does not bioaccumulate in earthworms to a great extent (based on the *E. fetida* - Nahmani data), the level of accumulation observed would be expected to lead to a small exceeding of the MPC_{oral} if population relevant endpoints would be of the same order of magnitude (or lower) as those used for the TDI. Based on the current data, secondary poisoning of thallium in the terrestrial compartment cannot be excluded, but is unlikely to be the critical route.

Bioaccumulation at the level of the SRA_{soil}
Since only one MPC_{oral} value is available, this aspect cannot be assessed.

5.3.6 Tin

Introduction
Selected literature was screened for data on elemental tin, but not on organotin compounds. Many studies report on trace metal concentrations in freshwater and marine animals and sediments collected in surveys. No publications were retrieved in which bioaccumulation of tin in terrestrial organisms was investigated.

WHO (1980) reports 'little reliable information exists on the bioaccumulation of tin and its derivatives'. No information is given on the bioaccumulation of tin in the terrestrial environment. A more recent WHO document (WHO, 2005) reports a few bioaccumulation factors for aquatic organisms, demonstrating accumulation, but no information is given for the terrestrial compartment.

Citations from Eisler (1989)
'Inorganic tin compounds are of low toxicologic risk due largely to their low solubility, poor absorption, low accumulations in tissues, and rapid excretion. Inorganic tin compounds and some heterocyclic organic tin compounds are of low toxicologic risk to mammals, due largely to their low solubility, poor absorption, low tissue accumulations, and rapid tissue excretion. Inorganic tin compounds accumulate mostly in liver and kidney, rarely in brain, in proportion to dose and regardless of the exposure route.'

Citation from ATSDR (2005):
'Inorganic tin compounds may also be bioconcentrated, but data are limited. There is no information available on the potential transfer of inorganic tin or organotin compounds from lower trophic levels to higher levels.'

Bioaccumulation at the level of the MPA_{soil}
A human-toxicological risk limit for tin is available: an MPC_{human} of 0.2 $mg/kg_{bw}/d$ (Tiesjema and Baars, 2009). This MPCs is based on a 13 weeks study with rats with a no observed adverse effect concentration (NOAEL) of 32 $mg/kg_{bw}/d$. The endpoint of this NOAEL is 'haematological effects'. Using a diet conversion factor of 20 g_{bw} d/g_{food} and an assessment factor of 90, the $MPC_{oral,\ mammal}$ is 7.1 mg/kg_{food}. A K_p soil:water for tin of 1905 L/kg (log K_p =3.28) has been used.

The outcome of the bioaccumulation model calculation is the need for a $criticalBCF^{est}$ of >200,000 L/kg or a $criticalBSAF^{est}$ of 116 kg_{dwt}/kg_{ww} to obtain a MPA which is lower than the MPA_{soil} based on direct toxicity (0.068 mg/kg). Both bioconcentration estimates are very high. However, since no experimental data on bioaccumulation of tin in terrestrial organisms were found, no meaning can be assigned to the height of these estimates. We cannot postulate with certainty whether the current MPA_{soil} for tin will be protective against possible effects of secondary poisoning, although effects due to secondary poisoning of tin at the level of the MPA_{soil} are deemed unlikely.

Bioaccumulation at the level of the SRA_{soil}
An estimate of the $SRC_{eco,\ oral}$ cannot be made easily. The $SRC_{eco,\ oral}$ should be calculated as the geometric mean of the distribution of MPC_{oral} values for mammals (and birds). Available is a NOAEL of 32 mg/kg_{food} as given above for haematological effects. With an assessment factor of 3 to correct for laboratory

feed and an assessment factor of 10 to correct from subacute to chronic, the MPC_{oral} is 1.1 mg/kg_{food}. In addition, WHO (2005) reports on two2-year studies with mice and rats. Both resulted in a NOAEL of 1000 mg/kg_{diet} for the endpoint survival (only two concentrations were tested). Applying an assessment factor of 3 to both NOAELs to correct for laboratory feed this results in an MPC_{oral} value of 333 mg/kg_{food}. No further information was available. When the $SRC_{eco, oral}$ is calculated as the geometric mean of the most sensitive MPC_{oral} values for rats and mice, a value of 19 mg/kg_{food} results.

The $SRA_{soil, eco}$ is 250 mg/kg_{soil} based on direct ecotoxicological data. Since the model assumption is that gut content filled with soil makes up 10% of an earthworm, the concentration in earthworms is already 25 mg/kg based on soil alone. This exceeds the $SRC_{eco, oral}$ of 19 mg/kg_{food}. Consequently, at the SRC level, even a low bioaccumulation of tin would lead to a concentration in soil below the current $SRC_{soil, eco}$ based on direct toxicity. At the level of the $SRA_{soil, eco}$ no accumulation of tin in earthworms can be allowed.

5.3.7 Vanadium

Introduction

From several publications bioaccumulation potential of vanadium emerges. The majority of possibly relevant studies focus on (species from the) aquatic compartment: mussels, toads, crustaceans, fish, marine mammals (whales), 'aquatic birds'. Publications in which bioaccumulation is quantified and specifically those focussed on species relevant to the terrestrial compartment, are very limited. Only one study described a higher accumulation of vanadium in above ground plant parts. The following citation confirms the general picture from the above findings.

Citation from Irwin (1997):
'Preliminary data suggests the potential for bioaccumulation or bioconcentration of vanadium is low or limited for the following biota: mammals, birds, and fish. It appears to be high to very high for mollusks, crustacea, and lower animals and moderate for higher plants, mosses, lichens, and algae. It has a much higher bioconcentration potential in mollusks than in fish. The best potential mediums for biological monitoring (including gradient monitoring) appear to include higher plants, clams, mosses, and lichens. Plants take up vanadium from soil, groundwater, surface water, and air pollution. Animals take up vanadium from contaminated air, contaminated water, and contaminated food. Presumably, man & animals do not store or accumulate vanadium in hazardous amount.'

The scientific information collected with the literature search performed in this project is too few to conclude whether bioaccumulation of vanadium in the terrestrial environment occurs and if so, to which extent (quantification of BCF or BSAF). Therefore, with the current information we have to conclude that the importance of bioaccumulation of vanadium in the terrestrial environment cannot be assessed, although the overview of Irwin suggests it to be low.

Bioaccumulation at the level of the MPA_{soil}

The MPC_{human} for vanadium is based on a 60 d oral gavage, reproduction study with rats, which resulted in a LOAEL of 2.1 mg/kg_{bw}/d for the endpoint 'development of offspring' (Tiesjema and Baars, 2009). This endpoint is considered relevant to secondary poisoning as well. The LOAEL is recalculated to an MPC_{oral} of 0.047 mg/kg_{food} using a conversion factor of 20 g_{bw} d/g_{food}, an assessment factor of 90 (EU-TGD) and an extra factor of 10 to convert the

LOAEL to a NOAEL. The latter factor was also used in the derivation of the MPC_{human}. A K_p soil:water for vanadium of 309 L/kg (log K_p = 2.49) has been used.

With these data a criticalBCF^{est} of 487 L/kg or a criticalBSAF^{est} of 1.5 kg_{dwt}/kg_{ww} is calculated. This means that when vanadium exhibits higher BCF or BSAF values in the field, secondary poisoning may result in an MPC_{soil} value which is lower than the MPA_{soil} based on direct toxicity (0.032 mg/kg_{dwt}).

Because no bioaccumulation data for vanadium in terrestrial organisms were found, we cannot exclude that these accumulation levels occur at the concentration level of the MPA_{soil}.

Bioaccumulation at the level of the SRA_{soil}

For calculation of the SRC_{oral} the LOAEL of 2.1 $mg/kg_{bw}/d$ for rat given above is converted to food with a value of 2.1 x 20 = 42 mg/kg_{food}. After application of an assessment factor of 3 to correct for laboratory feed, an assessment factor of 3 for conversion from subchronic to chronic and a factor 10 to convert from LOAEL to a NOAEL, the MPC_{oral} value for rats is 0.47 mg/kg_{food}. In Tiesjema and Baars (2009) from which the MPC_{human} was cited, a subchronic mice NOAEL of 2.1 $mg/kg_{bw}/d$ and a subchronic mice LOAEL of 7.5 $mg/kg_{bw}/d$ were reported for maternal toxicity. Additionally, in an earlier evaluation (Janssen et al. 1998), a NOAEL from a subchronic reproduction study with mice was obtained of 40 $mg/kg_{bw}/d$ for decreased fertility. These NOAEL values are 17.4 and 332 mg/kg_{food} respectively when converted to food concentrations. After application of an assessment factor of 3 to correct for laboratory feed and an assessment factor to correct for subchronic to chronic, the MPC_{oral} values for mice are 1.9 and 36.9 mg/kg_{food} respectively. The maternal toxicity endpoint is most sensitive and therefore the MPC_{oral} for mice is 1.9 mg/kg_{food}. When the $SRC_{eco, oral}$ is calculated as the geometric mean of the values for mice (1.9 mg/kg_{food}) and rats (0.47 mg/kg_{food}) a $SRC_{eco, oral}$ value of 0.94 mg/kg_{food} results.

The $SRA_{soil, eco}$ is 25 mg/kg_{soil} based on direct ecotoxicological data. Since the model assumption is that gut content filled with soil makes up 10% of an earthworm, the concentration in earthworms is already 2.5 mg/kg based on soil alone. This exceeds the $SRC_{eco, oral}$ of 0.94 mg/kg_{food}. Consequently, at the SRC level, even a low bioaccumulation of vanadium would lead to a concentration in soil below the current $SRC_{soil, eco}$ based on direct toxicity. At the level of the $SRA_{soil, eco}$ no accumulation of vanadium in earthworms can be allowed (Tiesjema and Baars, 2009).

5.4 Relevance of secondary poisoning for the 'non-metal' compounds

5.4.1 Organotin compounds

Bioaccumulation at the level of the MPA_{soil} has been assessed in Van Herwijnen (2012) for dibutyltin, tributyltin and triphenyltin. Only for DBT was concluded that the $MPC_{soil, eco}$ is lower than the $MPC_{soil, secpois}$. Therefore, there is a risk for secondary poisoning at the level of the $MPC_{soil, eco}$ of TBT and TPT. Secondary poisoning at the level of the $SRC_{soil, eco}$ has also been assessed; for all three compounds can be concluded that at the level of the $SRC_{soil, eco}$ accumulation of DBT, TBT and TPT in earthworms is expected.

The $MPC_{soil, secpois}$ values derived in Van Herwijnen (2012) are 0.37 mg/kg_{dwt}, 2.3 ng/kg_{dwt} and 4.0 $\mu g/kg_{dwt}$ for DBT, TBT and TPT respectively. The $SRC_{soil, secpois}$ values are 28 mg/kg_{dwt}, 52 $\mu g/kg_{dwt}$ and 0.24 mg/kg_{dwt} for DBT, TBT and TPT respectively.

5.4.2 *Chlorinated hydrocarbons*

Cis- and *trans*-1,2-dichloroethene have a BCF < 100 L/kg; therefore an environmental risk through secondary exposure is not considered relevant (Fleuren et al. 2009).

5.4.3 *Inorganic compounds*

Secondary poisoning is considered not relevant for free cyanide (EC, in preparation).

For thiocyanate, bioaccumulation and hence secondary poisoning of thiocyanate is not relevant due to the hydrophilic and ionised (log K_{ow} ~ -0.39) nature of the thiocyanate molecule. No experimental data for BCFs are found and BCF estimates using EPISuite (US EPA, 2009) range from 1.1 to 3.2 L/kg.

Secondary poisoning is not considered relevant for chloride and sulphate because these compounds have no accumulation tendencies.

5.5 Summary of secondary poisoning

Table 5.2 Overview of the possible relevance of secondary poisoning for the derived ERLs for soil.

Compound	Is secondary poisoning deemed relevant?	Are predators at risk at MPC level?	Are predators at risk at SRC level?
Metals and metalloids			
Antimony	yes	no	no
Barium	yes	unlikely	unlikely
Boron	no	-	-
Selenium	yes	unlikely	unlikely
Thallium	yes	unlikely	not assessed *
Tin (inorganic)	yes	unlikely	possibly
Vanadium	yes	possibly	possibly
Organotin compounds			
Dibutyltin (DBT) - kation	yes	no	yes
Tributyltin (TBT) - kation	yes	yes	yes
Triphenyltin (TPT) - kation	yes	yes	yes
Chlorinated hydrocarbons			
1,2-dichloroethene (sum)*	no	no	no
Cis-1,2-dichloroethene	no	no	no
Trans-1,2-dichloroethene	no	no	no
Inorganic compounds			
Free cyanides	no	no	no
Thiocyanate	no	no	no
Chloride	no	no	no
Sulphate	no	-	-

* Toxicity studies for birds and mammals with population relevant endpoints were not available.

6 Discussion and conclusions

In this chapter the proposed Intervention Values as presented in tables 4.1 en 4.2 will be discussed. Furthermore, attention is given to some of the choices made along the process, such as the use of sum values.

6.1 Barium

Barium was also evaluated in 2001 by Lijzen et al. The value derived at that time is considerably higher (890 mg/kg) than the value derived in this report (400 mg/kg). For barium, ecosystems are the relevant receptor. The value used in this report was derived by Van Vlaardingen (2005). An explanation why the current value is relatively low is because the value from 2001 was only based on soil processes. When the value of 2005 was derived, chronic data for annelids and insects were also available. These indicated a higher sensitivity for those species which resulted in a lower $SRC_{soil.\ eco}$.

Currently, the intervention value for barium is temporarily withdrawn due to the fact that the natural background concentration for barium frequently exceeds the intervention value. If increased concentrations are found from an anthropogenic origin, these concentrations are currently assessed based on the previous intervention value of 920 mg/kg. This value is based on the same information as the value of 890 mg/kg derived in 2001; however, the background concentration changed since 2001 from 160 mg/kg to 190 mg/kg. The change in intervention value is directly related to the change in background concentration.

Part of the problem with barium is caused by the background value of 190 mg/kg, which is based on the 95-percentile of concentrations in relatively unpolluted areas. Natural background concentrations for barium can be a factor 3 higher based on the 95-percentile of the most recent dataset (Mol et al. 2012; Van der Veer, 2006). The background value of 190 mg/kg corresponds with the 95-percentile of the reactive fractions of barium in soils from the same dataset. In practice, measured total barium is often bound in barium sulfates and therefore not available to excert negative effects. The new proposed intervention value therefore refers only to the reactive fractions of barium. A solution for this problem is beyond the scope of this report, but might be found in using extraction techniques that only determine the reactive fractions, like 0.43 HNO_3 extraction.

6.2 Organotins

6.2.1 Tributyltin

Organotin compounds can be present in different derivatives. For tributyltin, SRCs were derived based on the chemical properties of tributyltin chloride, -hydride and - oxide. Of these three compounds, tributyltin hydride is the most toxic for human exposure, but this compound is rarely found in contaminated soils. Tributyltin oxide is less toxic than hydride but is commonly found in contaminated soils. It was therefore decided to select the SRC_{human} for tributyltin oxide as a sum SRC_{human} for all tributyltins.

6.2.2 Triphenyltin

For triphenyltin also different derivatives exist. These derivatives are triphenyltin chloride, -hydroxide and -acetate. For triphenyltin no information was found as to which of these derivatives can be found most often at contaminanted soils. It

is however known that triphenyltin acetate and -chloride are hydrolised into triphenyltin hydroxide in water. When Van der Berg et al. in 1994 derived the first intervention value for triphenyltin, they used the chemical properties of the hydroxide derivative. Based on this information, it was therefore decided that the SRC_{human} for triphenyltin hydroxide will be used as the sum SRC_{human} for triphenyltin in soil and groundwater.

6.3 Dichloroethene

It was decided to derive a sum value for *cis* 1,2-dichlorethene and *trans* 1,2-dichloroethene. This choice was made because Janssen (2008) concluded that on the basis of new toxicological data the difference between *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene was not longer warranted. Furthermore, the differences between the individual SRCs are relatively small due to compound behaviour.

6.4 Free cyanide and thiocyanate

Free cyanide and thiocyanate were evaluated in 2001 by Lijzen et al. However at that time no proposals for an intervention value were done. Köster (2001) proved that exposure via crop consumption is considered negligible because uptake by crops is overestimated and e.g. cyanide is easily converted into non-toxic compounds. Human exposure to soil contamination could mainly occur via inhalation of free cyanide in ambient air or via soil ingestion of ferrohexacyanide or thiocyanate dissolved in groundwater.

In 2006, RIVM performed specific calculations concerning the risks due to the exposure by inhalation of HCN. The results were discussed by the working group NOBOWA. It was decided that, given the uncertainties, no risk levels for HCN could be legally established. However, in case of the presence of free HCN, additional measurements in soil air and/or indoor air should be performed.

Moreover, it was decided that the exposure through crop consumption can be excluded from the risk assessment procedure. Thiocyanate was not considered in 2006.

6.4.1 Free cyanide

Concerning the toxic properties of cyanide no new toxicological data (since the evaluation of 2001) have become available. Therefore, the SRC_{eco} and MPR_{human} proposed in this report are equal to those proposed in 2001.
Concerning the physical chemical properties of free cyanides (HCN), the evaluation resulted in the determination of new values for the SRC_{human} in soil.

It is likely that HCN degradates in the soil-water system during transport from e.g. groundwater to indoor air. However, the COIL model does not account for biodegradation or chemical conversions when exposure of compounds is assessed. Moreover, given the nature of HCN it is difficult to predict the chemical form of the occurrence of cyanide. Cyanide can be present as HCN (volatile), CN- (solluble) of complexed (immobile).

Therefore, besides the standard approach for the derivation of $SRC_{human,}$ two alternative exposure scenarios were considered to assess the risks due to the exposure by free cyanide. For exposure calculations in the second scenario HCN was considered as an organic compound. In an aqueous environment HCN is in equilibrium with CN^- as follows:

$$CN^- + H_2O \rightarrow HCN\ (g) + OH^-$$

Moreover, given the results of Köster (2001), exposure through crop consumption was not included in the assessment. This scenario is in line with the additional calculations in 2006.

The third scenario was defined to include the risk of acute exposure of children through the single ingesting of 5 grams soil and no crop consumption.

These alternative scenarios were chosen because cyanide has an acute human toxicity and this is neglected in the lifelong exposure scenario. It should be noted that the standard approach resulted in unrealistic high SRC_{human} values which also included uptake via vegetable consumption. Therefore, these values were discarded.

6.4.2 Thiocyanate

Concerning the toxic properties of thiocyanate no new toxicological data (since the evaluation of 2001) have become available. Thiocyanate is considered less toxic than free cyanide. It is therefore curious that the TDI for thiocyanate is lower than for free cyanide. This has to do with the available information on toxicity of both compounds. Less information is available for thiocyanate, therefore a larger uncertainty factor is applied resulting in a higher TDI for thiocyanate.

Although no new toxicity data for human or ecosystems were found in the review in this report, new physical chemical properties for thiocyanate resulted in new SRC proposals in this report.

As with free cyanide two alternative scenarios were used when deriving new proposals for Intervention Values. The first alternative scenario is equal to the default scenario, but without vegetable consumption. The second alternative scenario corresponds with the risk of acute exposure of children through the single ingesting of 5 grams soil.

6.5 Chloride

Currently there is no intervention value for chloride in soil or groundwater. Furthermore, chloride is considered non-toxic to humans and the proposed value in this report is only based on the SRC_{eco} for soils and groundwater that are not influenced by brackish or salt waters. Chloride is very mobile, so an intervention value for soil is less relevant.

It is recommended to assess the need for an intervention value for chloride in a broader perspective together with policy makers. Intervention Values are necessary to take decisions concerning historical soil contamination. As chloride is completely soluble and very mobile it is hardly possible that a chloride contamination will be traceable after 25 years ore more. New cases of soil contaminations should be restored independent of found concentrations.

6.6 Overall changes per compound group

6.6.1 Metals and metalloids

Soil

The proposed SRCs for metals and metalloids for soil are generally substantially lower than the current SRCs for selenium, thallium, tin and vanadium. For these compounds the ecotoxicological risk criteria are decisive. The changes originate mostly from additional ecotoxicological information that has become available for these compounds since the first study.

For antimony the proposed human based SRC is significantly higher than the current intervention value. For barium and boron there are currently no Intervention Values available.

Groundwater
The SRCs for antimony and tin in groundwater are higher than the current intervention value (antimony) and indicative level (tin). Both SRCs are based on human exposure via groundwater used as drinking water. The proposed SRCs for barium, selenium and vanadium are not significantly different from the current Intervention Values for soil and groundwater. The proposed SRC for thallium is lower than the current indicative level. For boron currently no SRC is available.

6.6.2 Organotins

Soil
In contrast to the current intervention value for organotins which is a sum value for DBT, TBT and TPT, no sum value for the organotins was derived in this report. For each organotin a separate SRC was derived. The SRC for DBT, TBT and TPT are all above the current sum value of 2,5 mg/kg for organotins.

Groundwater
Also for groundwater no sum value for organotins was derived. The new derived SRC for tributyltin and triphenyltin are both lower than the current sum value of all organotins. The value for dibutyltin is higher than the current sum value.

6.6.3 Cis- and trans 1,2 dichloroethene

Soil
For *cis*- and *trans* dichloroethene it was decided to maintain a sum value. The new SRC is a factor 3 higher than the current SRC and is based on the lowest SRC_{human}.

Groundwater
For groundwater, the new SRC, which is based on the exposure via groundwater consumption, is substantially higher than the current SRC. This change can be explained by new toxicological data in human risk assessment.

6.6.4 Inorganic compounds

Free cyanide and thiocyanate

Soil
The new SRC_{soil} for free cyanide is considerably lower than the current intervention value. The difference can be explained because the new SRC is based on ecosystems whilst the current value is based on human exposure including crop consumption, which is no longer a relevant exposure route.

For thiocyanate the new human based SRC is slightly higher than the current value, which is based on ecology. The exposure pathway vegetable consumption is however not included in the new proposal and furthermore this value is based on an acute exposure for children with a single soil ingestion of 5 grams at once.

Groundwater
The new SRC for free cyanide in groundwater is considerably lower than the current value. The new value is based on risks for ecosystems, whilst the current value is probably based on the human consumption of groundwater.

For thiocyanate the new SRC in groundwater is also lower than the current value. The new value is based on human consumption of groundwater. The difference between the new and the current value can be explained by new physical/chemical data.

Chloride and Sulphate

Soil

For chloride there are currently no Intervention Values for soil. The newly derived SRC is based on risk for ecosystems only because no adverse effects for humans are expected. For sulphate no new SRCs could be derived because the compound does not have any toxicological effects on humans or ecosystems. For other secondary effects of sulphate, is referred to Brand et al. (2008).

Groundwater

Also for groundwater there are currently no Intervention Values. The newly derived SRC for groundwater is also based on risk for ecosystems. For sulphate no new SRCs could be derived because the compound does not have any toxicological effects on humans or ecosystems. However, sulphate can cause problems via secondary effects like eutrophication if the groundwater stands in direct contact with surface water (Brand et al. 2008).

Abbreviations

AF	Assessment Factor
ATSDR	Agency for Toxic Substances and Disease Registry
B	Boron
Ba	Barium
BAF	Bio Accumulation Factor
BCF	Bio Concentration Factor
BSAF	Biota to Soil (Sediment) Accumulation Factor
CAR	Competent Authority Report
C_b	Background concentration
DBT	Dibutyltin
DOT	Di-n-octyltin
TBT	Tributyltin
TPT	Triphenyltin
EC	European Commission
ECB	European Chemicals Bureau
EFSA	European Food Safety Authority
ERL	Environmental Risk Limit
EU-RAR	European Union - Risk Assessment Report
INS	Acronym of the project 'International and national environmental quality standards for substances' in the Netherlands
JRC	Joint Research Centre
JECFA	Joint FAO/WHO Expert Committee on Food
LOAEL	Lowest Observed Adverse Effect Level
MAA	Maximum Acceptable Addition
MAC	Maximum Acceptable Concentration
MPA	Maximum Permissible Addition
MPC	Maximum Permissible Concentrations
MPR	Maximal Permissible Risk
NOAEL	No Observed Adverse Effect Level
NC	Negligible Concentration
PNEC	Predicted No Effect Concentration
PTWI	Provisional Tolerable Weekly Intake
Sb	Antimony (Stibium)
Se	Selenium
Sn	Tin (Stannum)
SRA_{eco}	Serious Risk Addition for ecosystems
SRC_{eco}	Serious Risk Concentration for ecosystems
TCA	Tolerable Concentration in Air
TDI	Tolerable Daily Intake
EU-TGD	European Technical Guidance Document
UF	Uptake Factor
V	Vanadium

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Annex 1 Evaluation of human-toxicological maximum permissible risk levels for boron, selenium and thallium

A1.1 Boron

A1.1.1 Introduction

Boron was evaluated within the scope of this project by Janssen et al. in 1995. They derived a TDI of 90 µg/kg bw/day for oral intake. This TDI was based on a NOAEL of 350 mg B/kg diet (~ 8.8 mg/kg bw/day, LOAEL 1170 mg B/kg diet) in a 2-year study with dogs and a multigeneration study in rats with the same dose and an application of an uncertainty factor of 100. A TCA was not proposed.

For the present update additional literature was reviewed (published since 1995). This included evaluations by EFSA (2004 and 2005), IPCS (1998), ATSDR (2007), WHO (2009), US-EPA (2004) and RIVM (Van Engelen et al. 2008).

Boron is a naturally-occurring non-metal element that is widespread in nature at relatively low concentrations. In the environment boron is always found chemically bound to oxygen, usually as alkali or alkaline earth borates or as boric acid. The overall average concentration in the earth's crust has been estimated to be 8-10 ppm. Concentrations reported in sea water range from 0.5-9.6 ppm with an average of 4.6 ppm. (ATSDR, 2007; EFSA, 2004; US-EPA, 2004).

Boric acid, borax and other borates are used in a wide range of consumer products, including glass, fiberglass insulation, soaps, detergents, preservatives, adhesives, porcelain, mild antiseptics, cosmetics, pharmaceuticals (as pH buffers), enamel, leathers, carpets, artificial gemstones, high-contrast photographic material, wicks, electric condensers, metal alloys, fire retardants, fertilisers, insecticides and herbicides (ATSDR, 2007; EFSA, 2004; US-EPA, 2004; WHO, 2009).

A1.2 Toxicology

A1.2.1 Toxicokinetics

Absorption

Both borates and boric acid are well absorbed from the gastrointestinal tract. In several studies in human volunteers absorption percentages of 84% and higher were found. Also in animal studies boron is readily absorbed from the gastrointestinal tract (> 90% within 3 hours) (US-EPA 2004).

Boron can also be absorbed in humans and animals during inhalation exposure, but quantitative data are not available. Boron is not absorbed across intact skin, although it is readily absorbed across severely damaged skin in rabbits in proportion to the exposure concentration (US-EPA,2004).

Distribution

Boron is distributed fairly uniformly outside the blood compartment across various tissues (liver, kidney, muscle, large intestine, brain hypothalamus, testis, epididymis, seminal vesicles, seminal vesicle fluid, adrenals, and prostate). However, consistently lower concentrations of boron were found in fat (20% of plasma levels after day 7) and consistently higher concentrations were observed in bone (2- to 3-fold over plasma levels after day 7) (US-EPA, 2004).

Metabolism
There is no evidence that boric acid is degraded in the body. The lack of metabolism is probably due to the large amount of energy (523 kJ/Mol) required to break the boron-oxygen bond. Boric acid can form complexes with various biomolecules (US-EPA, 2004).

Excretion
Boron is excreted quickly and primarily via urine: more than 90% of an orally administered dose of boric acid is excreted unchanged in the urine over a short time after treatment. Renal clearance of boron from female rats is greater than in human females; pregnant rats and pregnant women clear boron slightly more efficiently than nonpregnant rats and women. The magnitude of the difference (rat:human) between average clearance values was respectively approximately 3.6-fold and 4.9-fold for pregnant and nonpregnant individuals, in close agreement with differences in kinetic parameters predicted by allometric scaling (approximately 4-fold) (US-EPA, 2004).

A1.2.2 *Toxicity*
Because boric acid exists primarily as the undissociated acid (H_3BO_3) in aqueous solution at physiological pH, as do the borate salts, the toxicity associated with these compounds is expected to be similar, based on boron equivalents. Also boron oxide will produce similar effects because it is an anhydride that reacts exothermically with water in the body to form boric acid (US-EPA, 2004; WHO, 2009).

Essentiality
Boron is a trace element which essentiality is suspected but has not been directly proven in humans (US-EPA, 2004). However, the essentiality of boron has been established for most plants and some animals (ATSDR, 2007).

Irritation
The primary health effects associated with inhalation exposure of humans to boron are acute respiratory and ocular irritation. Acute-duration exposures of mining and processing workers to 0.44–3.1 mg boron/m^3 (5.7–14.6 mg particulates/m^3) as sodium borate dusts has been associated with mild irritation of the eyes, throat and nose, as well as cough and breathlessness. Similar symptoms and signs of upper respiratory tract irritation have been observed in exercising volunteers exposed for short durations (<1 hour) to 1.5 mg boron/m^3 as sodium borate dusts. In rats exposed to 73 mg boron/m^3 for 10 weeks, some indication of local irritation of the external nares (nostrils) was reported (ATSDR, 2007).

Limited data indicate that 5 or 10% aqueous solutions of boric acid and borates are mild skin irritants. An NOAEL for this effect is unknown (Van Engelen 2008).

Acute and subacute toxicity
The primary health effects associated with dermal exposure are irritation of the eyes and reversible skin changes. Boron can be lethal following short-term oral exposure at high doses, although the variability in human responses to acute exposure is quite large. The minimal lethal dose of ingested boron (as boric acid) was reported to be 2–3 g in infants, 5–6 g in children and 15–20 g in adults. However, a review of 784 human poisonings with boric acid (10–88 g) reported no fatalities, with 88% of cases being asymptomatic. Liver, kidney, central nervous system, gastrointestinal effects and skin lesions have been

found in lethal cases following ingestion of boron, but death has been attributed to respiratory failure (ATSDR, 2007).

The oral LD_{50} values for boric acid or borax in mice and rats are in the range of about 400–700 mg of boron per kg of body weight. Oral LD_{50} values in the range of 250–350 mg of boron per kg of body weight for boric acid or borax exposure have been reported for guinea pigs, dogs, rabbits, and cats. Signs of acute toxicity for both borax and boric acid in animals given single large doses orally include depression, ataxia, convulsions and death; kidney degeneration and testicular atrophy are also observed (WHO, 2009).

Subchronic and chronic toxicity

Consistently observed effects following intermediate and chronic exposure in animals include hematological alterations (decreases in hemoglobin levels and splenic hematopoeisis) and desquamated skin on the paw; these effects have been observed at doses of ≥60 mg boron/kg/day. Chronic inflammation and coagulative necrosis have also been observed in the livers of mice exposed to 79 mg boron/kg/day for 2 years (ATSDR, 2007).

Genotoxicity and carcinogenicity

No epidemiological studies have identified an association between boron exposure and development of cancer. Chronic-duration oral studies in rats, mice and dogs involving dietary exposure to boric acid or borax have not found significant increases in neoplastic lesions. In vitro genotoxicity assays with boric acid (Ames test, L5178Y mouse lymphoma tk assay, unscheduled DNA synthesis, chromosomal aberrations or sister chromatid exchanges) have given predominantly negative results. In addition, no induction of chromosomal aberrations or mitotic spindle abnormalities in bone marrow erythrocytes was observed in an in vivo study in mice. IARC, NTP and EPA have not classified boron for human carcinogenicity (EFSA, 2004; ATSDR, 2007).

Reproduction toxicity

Oral exposure animal studies have clearly identified the reproductive system and developing fetus as the most sensitive targets of boron toxicity. Reproductive effects were observed both in repeated dose toxicity studies and reproduction studies. In a 2-year toxicity study in rats by Weir and Fisher (1972) reproductive effects (atrophy of seminiferous epithelium and decreased size of testicular tubules) were observed at 58.5 mg boron/kg bw/day but not at the lower dose level of 17.5 mg boron/kg bw/day (NOAEL) (EFSA, 2004). Decreases in the number of live fetuses and litters, decreases in body weight and increases in the occurrence of external, visceral, and cardiovascular malformations were observed in the fetuses of rabbits administered 44 mg boron/kg/day on gestation days 6–19; no developmental effects were observed at 22 mg boron/kg/day (ATSDR, 2007).

Following exposure of rats to 13.3 mg boron/kg/day on gestation days 0–20 (Price et al. 1996), decreases in body weight and increases in the occurrence of skeletal malformations have been observed in the fetuses. A NOAEL of 9.6 mg boron/kg/day was identified (EFSA 2004; ATSDR, 2007).

Alterations in the testes and sperm effects have been observed at higher doses (NOAEL in a 2-week study in rats was 44–53 mg boron/kg/day and 24 or 79 mg boron/kg/day in 2-year studies in rats and mice, respectively) (ATSDR, 2007).

A1.3 Evaluation by other organisations

US-EPA derived an RfD based on the 95% lower confidence limit of the benchmark dose associated with a 5% reduction in fetal body weight (BMDL05) in a benchmark dose analysis of the studies by Price et al. (1996) and Heindel et al. (1992). The combined data gave a $BMDL_{05}$ of 59 mg boric acid/kg-day (10.3 mg boron/kg-day). Application of a chemical-specific uncertainty factor of 66 (3.3 for toxicokinetic extrapolation from animals to humans, 3.16 for toxicodynamic extrapolation from animals to humans, 2.0 for variability in human toxicokinetics, and 3.16 for variability in human toxicodynamics) resulted in an RfD of 0.2 mg boron/kg bw/day. US-EPA did not derive an RfC, based on inadequate data (US-EPA, 2004).

In 2004 EFSA derived a tolerable upper intake level of 0.16 mg/kg bw/day, based on a NOAEL of 9.6 mg boron/kg bw/day for decreased foetal body weight in rats following maternal exposure during pregnancy (Price et al., 1996) and an uncertainty factor of 60 (10 for interspecies and 6 for intraspecies variation) (EFSA, 2004).

ATSDR has derived an MRL of 0.01 mg/m^3 for acute-duration inhalation exposure (14 days or less) to boron, based on symptoms of acute eye and respiratory irritation (nose, throat, cough, breathlessness) in volunteers after exposure to a 6-hour time-weighted average (TWA) concentration of 0.44 mg boron/m^3 (Wegman et al., 1994) and an uncertainty factor of 30 (3 for use of a minimally adverse LOAEL and 10 for human variability). In addition, an acute-duration oral MRL of 0.2 mg boron/kg/day was derived using the NOAEL of 22 mg boron/kg/day in the study of Price et al. (1996), based on increased incidence of external, visceral, and cardiovascular malformations and reduced body weight in the fetuses of rabbits administered boric acid via gavage on gestation days 6–19 and an uncertainty factor of 100 (10 for interspecies extrapolation and 10 for human variability). An MRL of 0.2 mg/kg/day has been derived for intermediate-duration oral exposure (15–364 days) to boron, based on the same approach as US-EPA has been used for the derivation of the RfD (ATSDR, 2007).

Also WHO considered the critical effect to be decreased fetal body weight in rats, for which the NOAEL was 9.6 mg/kg bw/day (Price et al., 1996). A benchmark dose, defined as the 95% lower bound on the dose corresponding to a 5% decrease in the mean fetal weight (BMDL05), which was used by the US-EPA in 2004 (also based amongst others on the study by Price et al., 1996), was 10.3 mg/kg bw/day, which is close to the Price et al. (1996a) NOAEL of 9.6 mg/kg bw/day. WHO applied an uncertainty factor of 60 to the BMDL05 of 10.3 mg/kg bw, which results in a TDI of 0.17 mg/kg body weight, rounded to 0.2 mg/kg body weight (WHO, 2003).

A1.4 Evaluation

In 1995, RIVM derived a TDI of 90 µg/kg bw/day for oral intake. This TDI was based on testicular effects observed in rats and dogs after administration of 1170 mg boron/kg diet. A NOAEL of 350 mg boron/kg diet (~ 8.8 mg/kg bw/day, LOAEL at 1170 mg/kg diet) was observed. Since then, several studies in rats and rabbits indicate that developmental effects are the most critical effects. The developmental studies by Price et al. (1990, 1994, 1996), in which decreased fetal weight was observed as well as an increased incidence of external, visceral and cardiovascular malformations, may serve as critical studies. Based on a NOAEL of 9.6 mg boron/kg bw/day for decreased foetal body weight in rats following maternal exposure during pregnancy (Price et al., 1996) and an uncertainty factor of 60 (10 for interspecies and 6 for intraspecies variation, adopted from WHO, 2009 and EFSA, 2004), a TDI of 0.20 mg/kg bw (rounded) is derived.

Exposure to concentrations of 0.44-3.1 mg boron/m^3 (6-hour time-weighted average) in workers resulted in mild irritation of the eyes, throat and nose, as well as in cough and breathlessness. Similar symptoms and signs of upper respiratory tract irritation have been observed in exercising volunteers exposed for short durations (<1 hour) to 1.5 mg boron/m^3 as sodium borate dusts. The available inhalation data indicate the respiratory tract as the sensitive target. Limited inhalation animal studies show absence of adverse systemic effects after inhalation exposure. Based on mild irritation in the respiratory tract in humans as the critical effect, a provisional TCA of 0.01 mg/m^3 is proposed (equal to the ATSDR acute MRL). In the derivation an uncertainty factor of 30 (rounded) is used (3 for use of a minimally adverse LOAEL, 3 for human variability, 3 for limited study duration).

A1.5 Background exposure

Boron is not present in the atmosphere at significant levels (WHO, 2009). Food is the main source of exposure for most populations but exposure via water, especially bottled mineral water, can be substantial as well. Data on dietary intakes of boron are limited. Foods rich in boron include fruits, leafy vegetables, mushrooms, nuts and legumes as well as wine, cider and beer. Meat, fish and dairy products are poor sources. For the UK total dietary intake for adults has been estimated at 1.5 mg/day (mean) and 2.6 mg/day (97.5 percentile) for the year 1994. Also for the UK a 2003 estimate for adults indicates a mean intake via water of 0.2-0.6 mg/day, via supplements up to 2.0 mg/day, and via cosmetics and consumer products of up to 0.47 mg/day. Maximum total daily intake was estimated at 5.67 mg/day. As stated, intake via bottled water may be high. EFSA (2005) indicates concentrations as high as 4.3 mg B/L have been measured in bottled mineral water. Based on these data, normal maximum daily intake is estimated to be 5 mg/day which equals 0.08 mg/kg bw for a 70 kg adult (Van Engelen, 2008).

A1.6 Conclusion

Table A1.1: Derived TDI and TCA for Boron.

Compound	TDI	PTCA	Background exposure
Boron	0.2	0.01	0.08

TDI: tolerable daily intake (oral exposure); mg/kg bw/day

PTCA: provisional tolerable concentration in air (inhalation exposure); mg/m^3

Background exposure; mg/kg bw/day (maximum of given range, rounded)

Profile compilation: B. Tiesjema (23-09-2010)
Profile review: P. Janssen, C. de Heer

A2.1 Selenium

A2.1.1 Introduction

Selenium was evaluated within the scope of this project by Janssen et al. (1998). They derived a TDI of 5 µg/kg bw/day for oral intake. This TDI was based on an NOAEL of 0.85 mg selenium/day for clinical selenose in a Chinese epidemiological study to which an uncertainty factor of 3 was applied. A TCA was not proposed.

For the present update, additional literature was reviewed (published since 1995). This included evaluations by IARC (updated in 1999), SCF (2000), ATSDR (2003), WHO (2004) and RIVM (Van Engelen et al. 2008).
Selenium is present in the earth's crust, often in association with sulfur-containing minerals. It has four oxidation states (2^-, 0, 4^+, 6^+) and occurs in many forms, including elemental selenium, selenites and selenates. Selenium is used in electronics, due to its semi-conductor and photoelectric properties. In addition, it is used in the glass industry, in pigments that are used in plastics, paints, enamels, inks and rubber, as a catalyst in the preparation of pharmaceuticals and as a constituent of fungicides. The principal releases of selenium into the environment as a consequence of human activities result from the combustion of coal. For the general population food is the primary exposure route followed by water and air (ATSDR, 2003).

A2.2 Toxicology

The toxicological data available for selenium and its compounds do not indicate the need for separate evaluation of the toxicological effects of the different oxidation states in which selenium occurs. Therefore, the different ionic forms of selenium and metallic selenium will be treated as equipotent in this evaluation (RIVM, 1998).

A2.2.1 Toxicokinetics

Absorption

Selenium compounds are generally very efficiently absorbed from the gastrointestinal tract by humans. For example, absorption of the selenite form of selenium is greater than 80% whereas that of selenium as selenomethionine or as selenate may be greater than 90%. The rate-limiting step determining the overall availability of dietary selenium is not likely to be its absorption, but rather its conversion within tissues to its metabolically active forms (WHO, 2004). Absorption and bioavailability of selenium compounds highly depend on the matrix (SCF, 2002; WHO, 2004). In addition, the physical state of the compound (e.g. solid or solution), the chemical form of selenium (e.g. organic, inorganic) and the dosing regimen, but not the exposure level, are factors influencing absorption. The main inorganic sources of selenium, selenate and selenite, are absorbed to a lesser degree than the organic forms (ATSDR, 2003).

Occupational studies indicate that humans absorb elemental selenium dusts and other selenium compounds, but quantitative inhalation toxicokinetic studies in humans have not been done. Studies in dogs and rats indicate that following inhalation exposure absorption is also high, although the rate and extent of absorption vary with the chemical form of selenium (ATSDR, 2003).

Distribution

Most studies report similar distribution patterns for both organic and inorganic selenium compounds tested. In plasma, selenium mainly distributes into three plasma proteins, namely selenoprotein P, glutathione peroxidase and albumin. In addition, approximately 3% of total plasma selenium is bound to lipoproteins. Selenium accumulates in many organ systems in the body; in general, the

highest concentrations are found in the liver and kidney. Blood, hair and nails also contain selenium. After a single oral dose of 2 mg selenium/kg bw (as sodium selenite) selenium concentration was highest in the kidney and liver, followed by the heart, lung and spleen followed by plasma and brain (ATSDR, 2003). Both inorganic and organic forms of selenium cross the placenta in humans and experimental animals (SCF, 2000). In addition, it is found in human milk (ATSDR, 2003).

Metabolism

The available data indicate that selenium-containing aminoacids and probably other selenium forms, such as selenite and selenate, can be converted into selenide in mammals. Selenide is a central metabolic form of selenium, which is utilised for the formation of selenocysteine, incorporated into specific selenoproteins, and in case of high exposure, into excretory products such as dimethyl selenide (which is exhaled) and trimethylselenonium ions (which are excreted into urine). Selenomethionine and selenocysteine formed by transsulfuration of selenomethionine can be non-specifically incorporated into protein as analogues to methionine and cysteine (SCF, 2000).

Excretion

Selenium is primarily eliminated in the urine and feces in both humans and laboratory animals. The distribution of selenium between the two routes seems to vary with the level of exposure and time after exposure. The form of selenium excreted is dependent on the form of selenium that was ingested. In cases of acute exposure to toxic concentrations of selenium or selenium compounds, significant amounts of selenium can be eliminated in the breath, causing the characteristic 'garlic breath'. Some researchers have found that urinary excretion and fecal excretion of selenium are similar, with each route contributing approximately 50% of the total output. Nevertheless, the proportion excreted via each route depends on several factors, including the level of exposure, the time since exposure, and the level of exercise (ATSDR, 2003).

A2.2.2 Toxicity

Limited data in humans suggest that children may be less sensitive for selenium toxicity than adults (ATSDR, 2003).

Essentiality

Being part of several enzymes, selenium is an essential element in humans and animals. Selenium has been implicated in the protection of body tissues against oxidative stress, maintenance of defenses against infection and modulation of growth and development (WHO, 2004).

Estimated daily requirements as summarised in SCF (2000) range from 40 to about 50 µg/day for adults with a lower limit of 20 µg/day.

Acute and subacute toxicity

Acute oral exposure to extremely high levels of selenium (several thousand times more than normal daily intake) produces nausea, vomiting, and diarrhea in both humans and laboratory animals. Acute oral exposure of humans to selenium has occasionally caused cardiovascular symptoms, such as tachycardia (dose not reported), but no electrocardiographic abnormalities were found in individuals from a human population chronically exposed to selenium (ATSDR, 2003). In a case report cardiovascular symptoms were also reported, eventually resulting in the death of two cases (Spiller and Pfiefer, 2007). In laboratory animals, acute- and intermediate-duration oral exposure to very large amounts

of selenium (approximately 100 times normal human intake) has produced myocardial degeneration (ATSDR, 2003).

Selenite, selenate and selenomethionine are among the most acutely toxic selenium compounds (Högberg and Alexander, 1986). Intake of 250 mg selenium as a single dose or multiple doses of 27-31 mg resulted in acute toxicity with nausea, vomiting, nail changes, dryness of hair, hair loss, tenderness and swelling of fingertips, fatigue, irritability and garlicky breath (SCF, 2000). In addition, diarrhea, fatigue and joint pain are reported following oral intake of a high dose of selenium (41 mg) (Macfarquhar et al. 2010).

The primary target organ in humans and laboratory animals in cases of acute, high-level inhalation exposure to selenium dusts or fumes is the lung, with cardiovascular, hepatic, nervous, and renal involvement as well. Workers acutely exposed to high concentrations of elemental selenium dust (dose not reported) have reported stomach pain and headaches, whereas workers briefly exposed to high levels of selenium dioxide dust (dose not reported) experienced respiratory symptoms such as pulmonary edema, bronchial spasms, symptoms of asphyxiation and persistent bronchitis, elevated pulse rates, lowered blood pressure, vomiting, nausea and irritability (ATSDR, 2003).

Subchronic and chronic toxicity

Animals show growth reduction, liver changes, anaemia, pancreatic enlargement and some domestic animals also exhibit neurotoxicity following selenium exposure above 0.03-0.4 mg/kg bw (Alexander and Meltzer, 1995).

Chronic oral intake of very high levels of selenium (10–20 times more than normal) can produce selenosis in humans (e.g. recently reported in Sutter et al., 2008). As shown by affected populations in China, chronic dietary exposure to these excess levels of selenium has caused diseased nails and skin and hair loss as well as neurological problems, including unsteady gait and paralysis. A LOAEL of 1.26 mg selenium/day and a NOAEL of 0.85 mg selenium/day can be derived for these effects (Yang et al. 1989). In another study in volunteers no signs of selenosis were observed with doses up to 724 µg/day (Longnecker et al. 1991). Slight increases in prothrombin-time and in the liver enzyme ALAT, indicating liver damage, have also been observed following chronic (18 months) intake of 0.35-0.6 mg selenium/day; but the clinical significance of these slight increases remains unclear, SCF (2000) concludes.

Several occupational studies describe respiratory effects such as irritation of the nose, respiratory tract and lungs, bronchial spasms and coughing following exposure to selenium dioxide or elemental selenium as dust. Similar effects have been seen in animals inhaling high doses of elemental selenium fumes or dust (ATSDR).

Genotoxicity and carcinogenicity

As to its carcinogenic potential IARC concluded the available data are insufficient to allow an evaluation of the carcinogenicity of selenium compounds (IARC, 1999). Evidence suggests that some forms of selenium exert an anti-tumorigenic action in animals and humans. Selenium sulfide however appears an exception, producing increased tumor incidences after oral administration (RIVM, 1998). There are no epidemiologic data that support a causal association between the inhalation of elemental selenium dusts or selenium compounds and the induction of cancer in humans (ATSDR, 2003).

A moderate genotoxic activity of selenium compounds (i.e. selenite, selenate, selenide, selenocysteine and selenosulphide) has been found in several *in vitro* systems (SCF, 2002). Organoselenium compounds in the range of 10–

100 µM were genotoxic and cytotoxic to human leukocytes cells *in vitro* (Santos et al. 2009).

It is suggested that selenite acts as an oxidizing agent in S. cerevisiae, producing DNA double strand breaks as well as frame shift mutations (Letavayová et al. 2008). There is one in vivo study showing chromosomal aberrations and increased SCE in hamster bone marrow cells after selenite treatment. This occurred only at doses of 3, 4, and 6 mg Se/kg bw i.p. that were associated with severe systemic toxicity, including lethality. *In vitro* studies indicate that the mutagenic effects of selenium salts are associated with production of reactive oxygen radicals and glutathione promotes these reactions (SCF, 2002). Selenium compounds have also shown anti-genotoxic effects (Valdiglesias et al. 2009). Generally, the genotoxic effects were observed at high dosages and the anti-genotoxic at low dosages (RIVM, 1998; Moreira Rosa et al. 2007).

Reproduction toxicity

In animals, oral exposure to high doses of sodium selenate or selenite (at least 8 times greater than those normally supplied by an adequate diet) caused increased numbers of abnormal sperm as well as testicular hypertrophy, degeneration and atrophy in male rats, and affected the estrous cycle in female rats and mice. No effects on fertility have been reported (ATSDR, 2003). Effects of selenium compounds on reproduction and offspring in rodents have usually been associated with overt maternal poisoning and nutritional deprivation (SCF, 2000). Studies on macaques fed selenomethionine (25, 150 and 300 µg/kg bw/day) during organogenesis showed no signs of terata, whereas a dose-dependent maternal toxicity was observed in this study (Tarantal et al., 1991 SCF). No indication of teratogenicity of selenium has been shown in humans, even in the areas of high selenium intake in China (Yang et al. 1989b).

A2.3 Evaluation by other organisations

SCF (2000) used the NOAEL of 0.85 mg/day for clinical selenosis as derived from the Chinese epidemiology studies (Yang et al., 1989). It was pointed out that several other studies further support this NOAEL. Application of an uncertainty factor of 3 to allow for the remaining uncertainties of the studies used, led to Tolerable Upper Intake Level (UL) of 300 µg/day. No specific UL for children was derived by lack of appropriate data. In addition, there are no reports indicating that children are more susceptible to adverse effects from selenium. Hence it seems appropriate to extrapolate the UL from adults to children on a body weight basis (SCF, 2000).

ATSDR (2003) did not derive MRLs for inhalation exposure to selenium or acute or intermediate oral exposure to selenium because of insufficient quantitative data concerning both human and animal exposures. Based on a NOAEL of 819 µg/day (absence of clinical selenosis in the Chinese epidemiology studies (Yang et al., 1989) and an uncertainty factor of 3 for human variability, an MRL of 0.005 mg/kg/day (5 µg/kg/day) has been derived for chronic oral exposure (>365 days) to selenium. An uncertainty factor of 3 was considered appropriate because the individuals in this study were sensitive individuals drawn from a larger population and were supported by other studies (ATSDR, 2003).

A2.4 Evaluation

Since the evaluation of selenium in 1998 by Janssen et al. no new data have become available that indicate a change in TDI for selenium is necessary. Therefore, based on the NOAEL of 0.85 mg Se/day from the study by Yang et al.

(1989) and an uncertainty factor of 3 to account for sensitive individuals, the TDI is maintained at 5 µg Se/kg bw/day.

A TCA for inhalation exposure to selenium is not derived, due to the lack of quantitative data concerning both human and animal exposures.

A2.5 Background exposure

The main inorganic sources of selenium in the diet are selenate and selenite (ATSDR, 2003). EFSA (2000) gives an overview of daily intake levels in European countries. These data indicate daily intake levels up to about 1 µg/kg bw/day (van Engelen, 2008). European data as summarised by SCF (2000) also indicate a mean adult daily intake of 1 µg/kg bw/day. For children twice this figure should be a reasonable estimate, i.e. 2 µg/kg bw/day. General exposure via air is estimated to be low compared to the amounts ingested via food (concentrations in air are below 10 ng/m^3) (RIVM, 1998).

A2.6 Conclusion

Table A1.2: Derived TDI and TCA for Selenium.

Compound	TDI	TCA	Background exposure
Selenium	5	-	1-2

TDI: tolerable daily intake (oral exposure); µg/kg bw/day.

TCA: tolerable concentration in air (inhalation exposure); µg/m^3.

Background exposure; µg/kg bw/day (maximum of given range, rounded).

A TCA for inhalation exposure to selenium is not derived.

Profile compilation: B. Tiesjema (23-09-2010)
Profile review: P. Janssen, C. de Heer

A3.1 Thallium

A3.1.1 Introduction

Thallium was evaluated within the scope of this project by Janssen et al. in 1998. They derived a provisional TDI of 0.2 µg/kg bw/day for oral intake. This TDI was based on a NOAEL of 0.20 mg thallium/kg bw/day in a 90 day gavage study in rats, and application of an uncertainty factor of 1000. The TDI was considered provisional, since only a very limited database was available and genetic risks could not be excluded. A TCA was considered not needed.
For the present update additional literature was reviewed (published since 1998). This included an evaluation by US-EPA (2009).

Thallium belongs to the heavy metals (density 11.83 g/cm^3). Thallium is a soft and pliable metal. It melts at 303.5°C and boils at 1482°C. It is colorless, odorless and tasteless (US-EPA, 2009; Cvjetko, 2010). Thallium exists in a monovalent thallo- and a trivalent thalli-state. It tends to form stable complexes such as sulphur-containing compounds. The chemical properties of the monovalent compound are similar to alkali metals whereas the trivalent compound behaves more like aluminium (US-EPA, 2009; Cvjetko, 2010). Most thallium salts are soluble in water except thallium(III)oxide (US-EPA, 2009). Mean concentrations of thallium in the earth's crust are in the order of 0.1–1.7 mg/kg, thallium concentrations in soil are in the order of 0.1 to about 1.0 mg/kg (US-EPA, 2009; Kazantzis, 2000).

In the past thallium was extensively used for medicinal purposes (treatment of ringworm of the scalp, venereal diseases, tuberculosis and malaria). It was also used as a rodenticide and insecticide. Currently, the main uses of thallium are in the electrical and electronics industries as in electronic devices for semi-conductors, scintillation counters and low temperature thermometers, in mixed crystals for infrared instruments and laser equipment. In addition, it is used for gamma radiation detection equipment, fireworks and imitation jewelry. Trace amounts of thallium are used as a contrast agent in the visualization of cardiac function and tumors (US-EPA, 2009; Kazantzis, 2000; Daubert, 2008).

A3.2 Toxicology

A3.2.1 Toxicokinetics

Absorption

In humans and animals thallium compounds are well absorbed by various routes, including skin. No information was found regarding the absorption of thallium salts via inhalation (US-EPA, 2009; Saha, 2004).

Distribution

Thallium ions are rapidly distributed throughout the body in both experimental animals and humans, regardless of the route of exposure, dose, and length of exposure (US-EPA, 2009). In the first 4 hours following exposure, thallium is rapidly distributed into the blood and well-perfused organs such as the kidney, liver and muscle. Over the next 4-48 hours thallium is distributed into the CNS (Health Council of the Netherlands, 2002). In blood about 70% is bound to erythrocytes (Saha, 2004). In humans, kidneys display the highest concentrations; followed, in a decreasing order, by the bones, stomach, intestines, spleen, liver, muscle, lung and brain, with none being detected in fat tissue (US-EPA, 2009; Cvjetka, 2010).

Metabolism
Because thallium is an element, it is not metabolized. It is not known if thallium is transformed from one valence state to another in vivo (US-EPA, 2009).

Excretion
Thallium is primarily eliminated through excretion into the feces and the urine, but the amount excreted via each route varies depending on the animal species. In humans, excretion occurs mostly via urine, whereas in rats and rabbits excretion via feces is predominant. Thallium is also excreted through breast milk, sweat, saliva, and tears. Deposition in the hair and nails is also an important route of thallium elimination (US-EPA, 2009; Cvjetka, 2010). The elimination phase begins about 24 hours after ingestion. Elimination is slow with an elimination half-life of 3-30 days, varying with the dose and duration of the exposure (Daubert, 2008).

A3.2.2 *Toxicity*
The toxicological data available for thallium and its compounds do not allow differential evaluation of the toxicological effects of the different oxidation states in which thallium occurs. Thus, following the approach taken in the existing toxicity evaluations for thallium, the different forms of thallium and metallic thallium in the present evaluation are treated as being equipotent indicators of thallium toxicity (Janssen, 1998).

Essentiality
Thallium does not have a known biological use and is a non-essential element for life (Kazantzis, 2000; Cvjetko, 2010).

Irritation
No effects on irritation could be found in literature.

Acute and subacute toxicity
In adults the average lethal oral dose has been estimated to range from 10 to 15 mg/kg. Acute toxicity of thallium in humans is characterized by alopecia, severe pain in the extremities, lethargy, ataxia, abdominal pain or vomiting, back pain, abnormal reflexes, neuropathy, muscle weakness, coma, convulsion, other neurological symptoms (i.e. mental abnormalities, tremors, abnormal movements, abnormal vision and headache) and death. The lowest known single dose of thallium associated with adverse effects was reported to be 0.31 g of thallium (I) acetate (3.4 mg/kg thallium, assuming a 70 kg body weight). This dose caused paresthesia, pain, weakness, vomiting and alopecia (US-EPA, 2009).

Subchronic and chronic toxicity
In a 90-day gavage study with thallium(I)sulphate in rats (0.008, 0.04 or 0.20 mg thallium/kg bw/day), lacrimation, exophthalmos and miosis were observed at higher incidences in the treated male and female rats (all dose groups) compared with controls. Incidence of rough coat, piloerection, shedding, alopecia, incidence of aggression, tension/agitation, hyperactivity, vocalization and self-mutilation was also elevated. Dose-related increases in AST, LDH and sodium levels and decreases in blood sugar levels were detected in male and female rats after 30 and 90 days of exposure (MRI, 1988).

A dose of 1.4 mg thallium/kg/day (as thallium(I)sulphate) in drinking water of rats resulted in mortality (15 and 21% after 40 and 240 days of

treatment), hair loss and functional and histopathologic changes in the peripheral nerves (US-EPA, 2009).

Thallous acetate, provided via the diet of rats for 15 weeks (0, 0.0005, 0.0015, 0.003 and 0.005%) resulted in increased mortality at doses ≥ 0.003%. Alopecia was observed at doses of ≥ 0.0015% (Health Council of the Netherlands, 2002).

Genotoxicity and carcinogenicity

Positive results were obtained for thallium (I) nitrate (1 mM) in the recombination-repair (Rec) assay using Bacillus subtilis strains H17 and M45, with and without metabolic activation. Thallium (I) nitrate was not mutagenic in reverse mutation assays using several strains of Salmonella typhimurium and Escherichia coli or in assays for mitogenic gene conversion and reverse mutation in yeast. Single-strand DNA breaks occurred in cell cultures of C57BL/6 mouse and rat embryo fibroblasts exposed to thallium (I) carbonate, but not in CBA mouse fibroblasts. A dominant lethal test with thallium (I) carbonate on male rats resulted in an increase in embryonic death, suggestive of a dominant lethal effect. A dose of 200 mg thallium sulfate caused a slight increase in SCEs in peripheral blood lymphocytes taken from a 48-year-old man on day 1 and day 15 postexposure and caused a 3.5-fold increase in binucleated cells with micronuclei (US-EPA, 2009). In a micronucleus assay in human lymphocytes, thallium sulphate did not induce a statistical significant increase of micronuclei frequency (Migliore, 1999).

There are presently no studies that evaluate the carcinogenic potential of thallium or thallium compounds in animals and no adequate studies of humans chronically exposed to thallium (US-EPA, 2009).

Reproduction toxicity

No studies of the potential effects on fertility of thallium in female experimental animals are available. In male rats administration of thallium(I)sulphate in drinking water (approximately 0.7 mg/kg-day thallium) for 60 days resulted in disarrangement of the tubular epithelium, cytoplasmic vacuolation and distention of smooth endoplasmic reticulum of the Sertoli cells, reduced testicular β-glucuronidase activities, high concentrations of thallium in the testes and reduced sperm motility. In mice, administration of thallium(I)carbonate in drinking water for 6 months resulted in affected sperm motility at 0.001 mg/L (~0.3 µg thallium /kg bw/day) and higher; increased number of dead sperm at 0.01 mg/L (~3 µg thallium /kg bw/day) and reduced sperm count as well as an increased percentage of deformed sperm at 0.1 mg/L (~30 µg thallium /kg bw/day) and higher (US-EPA, 2009).

Administration of thallium(I)sulphate in drinking water (1 mg/dL) of female rats from day 1 of gestation to weaning (postnatal day 22) resulted in a delay in the development of the pilus apparatus by 50 days. A reduction of the α- and β-adrenergic and muscarinic vasomotor reactivity was also noted. Similar effects were observed after postnatal treatment of the dams with the same dose. Intraperitoneal studies with thallium exposure during development can also produce abnormalities (increased incidence of hydronephrosis and missing or non-ossified vertebral bodies, delays in ossification) (US-EPA, 2009).

The only consistent finding in several case reports of pregnant women exposed to thallium was a trend toward prematurity and low birth weight. Several children had alopecia, particularly those exposed during the third trimester (Hoffman, 2000).

Mechanism

A well-known mechanism of thallium toxicity is related to the interference with vital potassium-dependent processes, substitution of potassium in the (Na^+/K^+)-ATPase, as well as a high affinity for sulfhydril groups from proteins and other biomolecules (Galván-Arzate, 1998).

Both thallium(I) and thallium(III) cytotoxicities are associated with reactive oxygen species (ROS) formation, lipid peroxidation, collapse of mitochondrial membrane potential, activation of caspases cascade, lysosomal membrane leakiness and cellular proteolysis. It is suggested that thallium(I) is reductively activated by GSH, while GSH only plays an antioxidant role against thallium(III) cytotoxicity (Pourahmad, 2010).

A3.3 Evaluation by other organisations

US-EPA did derive candidate RfDs for soluble thallium salts based on two endpoints: a NOAEL of 0.04 mg/kg-day thallium for hair follicle atrophy and an average BMDL10 of 0.01 mg/kg-day thallium for alopecia (as representative of clinical observations more generally), both based on a 90 day study in rats (MRI, 1988). However, because the available toxicity database for thallium contains studies that are generally of poor quality and particular difficulties in the selection of appropriate endpoints, US-EPA decided not to derive an RfD for soluble thallium salts. In addition, they did not derive an RfD for thallium(III)oxide and thallium(I)selenite due to insufficient toxicity information (US-EPA, 2009).

The Health Council of the Netherlands recommends a health-based occupational exposure limit of 0.02 mg/m^3 for elemental thallium and its water-soluble compounds as an 8-hour time-weighted average (TWA). This value is based on alopecia, induced by ~0.5 mg thallium /kg bw/day in rats. Application of a factor 4 for the allometric scaling from rat to man, based on caloric demand, and an overall factor of 27 covering inter- and intraspecies variation and differences between experimental conditions and the exposure pattern of the worker, results in a NAEL for humans of 0.0046 mg/kg bw/day. Assuming a 70-kg worker inhales 10 m^3 of air during an 8-hour working day and a retention of 100% with applying the preferred value approach, a health-based occupational limit of 0.02 mg/m^3 is recommended for thallium and its water-soluble compounds. Many reports indicate that thallium can pass the skin, so the committee recommends a skin notation (Health Council of the Netherlands, 2002).

OSHA has set an exposure limit of 0.1 milligrams per cubic meter (0.1 mg/m^3) for thallium in workplace air. ACGIH has established the same guidelines as OSHA for the workplace. NIOSH has recommended that 15 mg/m^3 of thallium must be considered immediately dangerous to life and health (Peter and Viraraghavan, 2005).

A3.4 Evaluation

Data on the mutagenic potential of thallium are inconclusive. In addition, no carcinogenicity studies are available. Further toxicology data are also limited. Given the lack of a conclusion with respect to genotoxicity and the lack of data on carcinogenicity, the only feasible option in limit value derivation is the calculation of a (provisional) TDI value using a threshold approach.

Concurrent with US-EPA, a NOAEL of 0.04 mg/kg-day thallium for hair follicle atrophy (as representative of clinical observations more generally) as observed in a 90-day rat drinking-water study can be used as starting point for a TDI. Use of uncertainty factors for interspecies (10) and intraspecies (10) variation and for using a subchronic study instead of a chronic study (10), results in a TDI of 0.04 µg thallium/kg bw/day. Because of the limitations in the

dataset, including the limitations in the key study, the TDI is provisional. A TCA is not derived since it is considered not needed.

A3.5 Background exposure

The greatest exposure occurs by eating food, mostly homegrown fruits and green vegetables contaminated by thallium. The average dietary intake of thallium is less than 5 µg/day or 0.07 µg/kg bw/day (assuming an average body weight of 70 kg) (Kazantzis, 2000; Peter and Viraraghavan, 2005).

A3.6 Conclusion

Table A1.3: Derived TDI and TCA for Thallium.

Compound	pTDI	TCA	Background exposure
Thallium	0.04	-	0.07

TDI: tolerable daily intake (oral exposure); µg/kg bw/day.

TCA: tolerable concentration in air (inhalation exposure); mg/m^3.

Background exposure; µg/kg bw/day (maximum of given range, rounded).

Profile compilation: B. Tiesjema (23-09-2010)
Profile review: P. Janssen, C. de Heer

Annex 2 Overview of the Environmental Risk Levels

This Annex gives an overview of the Environmental Risk Levels (ERLs) for direct ecotoxicity (summarized in chapter 3). For soil was considered whether secondary poisoning would be relevant for the substances. This has not been done for the aquatic environment because this was not necessary for the purpose of this report.

An overview of the relevance of secondary poisoning in the soil compartment for substances is given in Table A2.8. In those cases where the risk of secondary poisoning was considered relevant for metals in soil, ERLs on the basis of secondary poisoning could not be derived because of the absence of sufficient relevant data. Therefore, an assessment of the potential bioaccumulation at the levels of the derived MPCs and SRCs for direct toxicity of metals in soil has been made in chapter 5. In that chapter, the relevance of secondary poisoning for the other compounds is also reported.

Table A2.1: Final freshwater ecotoxicological MPA, MAA, SRA for metals.

Compound	**$MPA_{fw,\ eco}$ [mg/L]**	**$MAA_{fw,\ eco}$ [mg/L]**	**$SRA_{fw,\ eco}$ [mg/L]**	**C_b [mg/L]**
Antimony	0.1	0.2	9.6	0.29×10^{-3}
Barium	29×10^{-3}	0.15	17	73×10^{-3}
Boron	0.18	0.45	6.8	62×10^{-3}
Selenium	1.2×10^{-3}	25×10^{-3}	0.13	0.041×10^{-3}
Thallium	0.16×10^{-3}	0.76×10^{-3}	6.5×10^{-3}	38×10^{-6}
Tin (inorganic)	0.60×10^{-3}	36×10^{-3}	0.40	8.2×10^{-3}
Vanadium	0.4×10^{-3}	2.2×10^{-3}	62×10^{-3}	0.82×10^{-3}

- = not derived.

Table A2.2: Final ecotoxicological ERLs for fresh surface water.

Compound	**$MPC_{fw,\ eco}$ [mg/L]**	**$MAC_{fw,\ eco}$ [mg/L]**	**$SRC_{fw,\ eco}$ [mg/L]**
Antimony	0.10	0.2	9.6
Barium	0.10	0.22	17
Boron	0.24	0.51	6.9
Selenium	1.3×10^{-3}	25×10^{-3}	0.13
Thallium	0.20×10^{-3}	0.80×10^{-3}	6.5×10^{-3}
Tin (inorganic)	0.61×10^{-3}	36×10^{-3}	0.40
Vanadium	1.2×10^{-3}	3.0×10^{-3}	63×10^{-3}
Dibutyltin (DBT)	0.15×10^{-3}	0.30×10^{-3}	1.6×10^{-2}
Di-n-octyltin (DOT)	-	-	-
Tributyltin (TBT)	0.2×10^{-6}	1.5×10^{-6}	2.6×10^{-5}
Triphenyltin (TPT)	0.23×10^{-6}	0.47×10^{-3}	1.0×10^{-4}
Sum 1,2-Dichloroethene	6.8×10^{-3} [a]	-	11 [a]
cis-1,2-Dichloroethene	-	-	-
trans-1,2-Dichloroethene	-	-	-
Free cyanide [c]	0.23×10^{-3}	-	3.1×10^{-2}
Thiocyanate	3.6×10^{-3}	-	10
Chloride	94 [b]	-	570 [b]
Sulphate	-	-	-

- = not derived.

[a] This value is valid for the sum concentration of the *cis*- and *trans*-isomer.

[b] This concentration is expressed as the chloride ion (Cl^-).

[c] Under the WFD new ERLs for free cyanide in surface water are being derived. The report is currently not finished, but preliminary ERLS are 0.1 x 10^{-3} mg/L for the $MPC_{fw, eco}$ and 0.8 x 10^{-3} mg/L for the $SRC_{fw, eco}$.

Since ERLs based on secondary poisoning for soil could not be derived in some cases, all soil ERLs are based only on direct ecotoxicity. Those ERLs for soil that might not be protective for the occurrence of secondary poisoning are indicated in table A2.8. In those cases additional research on the risk of secondary poisoning is recommended.

Table A2.3: Final ecotoxicological ERLs for groundwater.

Compound	**$MPC_{qw, eco}$ [mg/L]**	**$SRC_{qw, eco}$ [mg/L]**
Antimony	0.1	9.6
Barium	0.23	17
Boron	0.44 [a]	7.1 [a]
Selenium	1.3 x 10^{-3}	0.13
Thallium	-	-
Tin (inorganic)	-	-
Vanadium	1.2 x 10^{-3}	63 x 10^{-3}
Dibutyltin (DBT)	0.15 x 10^{-3}	5.0 x 10^{-2}
Di-n-octyltin (DOT)	-	-
Tributyltin (TBT)	0.2 x 10^{-6}	4.6 x 10^{-5}
Triphenyltin (TPT)	0.23 x 10^{-6}	4.0 x 10^{-4}
1,2-Dichloroethene	6.8 x 10^{-3} [b]	11 [b]
cis-1,2-Dichloroethene	-	-
trans-1,2-Dichloroethene	-	-
Free cyanide [c]	0.23 x 10^{-3}	3.1 x 10^{-2}
Thiocyanate	3.6 x 10^{-3}	10
Chloride	94	570
Sulphate	-	-

- = not derived.
[a] These values are for fresh groundwater only. For brackish and salt groundwater the $MPC_{sgw, eco}$ is 3.6 mg/L and the SRC_{sgw}, eco is 10 mg/L.
[b] This value is valid for the sum concentration of the *cis*- and *trans*-isomer.
[c] Under the WFD new ERLs for free cyanide in surface water are being derived. These new ERLs will also be applicable to groundwater.

Table A2.4: Final freshwater sediment ecotoxicological MPA, SRA for metals.

Compound	**$MPA_{sediment. fw. eco}$ [mg/kg_{dwt}]**	**$SRA_{sediment. fw. eco}$ [mg/kg_{dwt}]**	**C_b [mg/kg_{dwt}]**
Antimony	11	110	3
Barium	73	42 x 10^3	155
Boron	-	-	-
Selenium	-	-	-
Thallium	0.24	9.8	1.0
Tin (inorganic)	1	760	19
Vanadium	0.62 [a]	96 [a]	42

- = not derived.
[a] = calculated in this report through equilibrium partitioning.

Table A2.5: Final ecotoxicological ERLs for freshwater sediment.

Compound	**$MPC_{sediment, fw, eco}$ [mg/kg_{dwt}]**	**$SRC_{sediment, fw, eco}$ [mg/kg_{dwt}]**
Antimony	14	110
Barium	230	2.5×10^{4}
Boron	-	-
Selenium	-	-
Thallium	1.2	11
Tin (inorganic)	20	780
Vanadium	43	138
Dibutyltin (DBT)	0.37	123
Di-n-octyltin (DOT)	-	-
Tributyltin (TBT)	10×10^{-6}	2.7×10^{-2}
Triphenyltin (TPT)	2.2×10^{-6}	2.2×10^{-3}
1,2-Dichloroethene	-	-
cis-1,2-Dichloroethene	-	-
trans-1,2-Dichloroethene	-	-
Free cyanide	-	-
Thiocyanate	-	-
Chloride	340	2100
Sulphate	-	-

- = not derived.

Table A2.6: Final soil ecotoxicological MPA, SRA and geomean of of MPA and SRA for metals.

Compound	**$MPA_{soil, eco}$ [mg/kg_{dwt}]**	**$SRA_{soil, eco}$ [mg/kg_{dwt}]**	**Geomean MPA-SRA [mg/kg_{dwt}]**	**C_b [mg/kg_{dwt}] (INS/NOBO)**
Antimony	1.0×10^{2}	1.4×10^{3}	3.7×10^{2}	3/4
Barium	8.2	210	41	155/190
Boron	0.4	11	2.1	n.a./n.a.
Selenium	5.8×10^{-3}	1.2	8.3×10^{-2}	0.7/n.a.
Thallium	0.1	1.0	0.32	1.0/n.a.
Tin (inorganic)	6.8×10^{-2}	250	4.1	19/6.5
Vanadium	3.2×10^{-2}	25	0.89	42/80

n.a. = not available.

Table A2.7: Final ecotoxicological ERLs for soil.

Compound	$MPC_{soil, eco}$ [mg/kg_{dwt}]	$SRC_{soil, eco}$ [mg/kg_{dwt}]	C_b + (geomean MPA-SRA) [mg/kg_{dwt}]	Geomean MPC-SRC [mg/kg_{dwt}]
Antimony	1.0×10^2	1.4×10^3	3.7×10^2	–
Barium	200[c]	400[c]	230	–
Boron	-[a]	-[a]	-[a]	–
Selenium	0.71[c]	1.9[c]	0.78	–
Thallium	1.1	2.0	1.3	–
Tin (inorganic)	6.6	260[c]	11	–
Vanadium	80[c]	105[c]	81	–
Dibutyltin (DBT)	0.37	28[d]	-	3.2
Di-n-octyltin (DOT)	-	-	-	-
Tributyltin (TBT)	0.13[d]	0.052 [d]	-	0.82
Triphenyltin (TPT)	4.0×10^{-3} [d]	0.24 [d]	-	0.031
1,2-Dichloroethene	2.0×10^{-2}	32	–	0.8
cis-1,2-Dichloroethene	1.9×10^{-2}	31	–	1.1
trans-1,2-Dichloroethene	2.8×10^{-2}	44	–	0.8
Free cyanide	0.3×10^{-3}	40×10^{-3}	–	3.5×10^{-3}
Thiocyanate	6.2	620	–	62
Chloride	39 [b]	390 [b]	–	123 [b]
Sulphate	-	-	-	-

- = not derived.
[a] Not derived since C_b for Boron is not available.
[b] This value is only valid for soils that have not been influenced by brackish or salt waters.
[c] ERLs are possibly not protecting for secondary poisoning, although the current datasets are too small to draw conclusions.
[d] This value is not protecting for secondary poisoning.

Table A2.8: Overview of the possible relevance of secondary poisoning for the derived ERLs for soil.

Compound	Is secondary poisoning deemed relevant?	Are predators at risk at MPC level?	Are predators at risk at SRC level?
Antimony	yes	no	no
Barium	yes	unlikely	unlikely
Boron	no	-	-
Selenium	yes	unlikely	unlikely
Thallium	yes	unlikely	not assessed [a]
Tin (inorganic)	yes	unlikely	possibly
Vanadium	yes	possibly	possibly
Dibutyltin (DBT)	yes	no	yes
Di-n-octyltin (DOT)	not assessed	not assessed	not assessed
Tributyltin (TBT)	yes	yes	yes
Triphenyltin (TPT)	yes	yes	yes
1,2-Dichloroethene	no	-	-
Free cyanide	no	-	-
Thiocyanate	no	-	-
Chloride	no	-	-
Sulphate	no	-	-

[a] Toxicity studies for birds and mammals with population relevant endpoints were not available.

Annex 3 Physicochemical data

This Annex presents the physicochemical data as used in the exposure model CSOIL 2000 to derive $SRCs_{human}$.

Table A3.1: Molecular weight (M).

Compound	CAS number	Molecular weight [g/mol]
Antimony	7440-36-0	121.76
Barium	7440-39-3	137.33
Boron	7440-42-8	10.81
Selenium	7782-49-2	78.96
Thallium	7440-28-0	204.38
Tin (inorganic)	7440-31-5	118.71
Vanadium	7440-62-2	50.94
Dibutyltin dichloride	683-18-1	303.85
Di-n-octyltin oxide	870-08-6	361.1
Tributyltin chloride	215-958-7	325.51
Tributyltin hydride	688-73-3	291.09
Tributyltin oxide	56-35-9	596.12
Triphenyltin acetate	900-95-8	409.0
Triphenyltin chloride	639-58-7	385.5
Triphenyltin hydroxide	76-87-9	367.0
Cis-1,2-dichloroethene	156-59-2	96.94
Trans-1,2-dichloroethene	156-60-5	96.94
Free cyanide	57-12-5[1]	26.02
Thiocyanate	540-72-7[2]	58.8
Chloride	16887-00-6	35.45
Sulphate	7757-82-6[3]	96.06

[1]CAS number of cyanide anion. [2]CAS number of NA-SCN. [3]CAS number of NA_2SO_4

Table A3.2: Soil-water partition coefficient (Kp) for metals and metalloids. Bold means new value.

Compound	Kp [dm^3/kg]	Reference
Antimony	**85**	Verschoor et al. (2006)
Barium	**2500**	Otte et al. (2001)
Boron	**2.6**	Van Herwijnen and Smit (2010)
Selenium	**15**	Sauve et al. (2000)

Thallium	160	Kreule and Swartjes (1998)
Tin (inorganic)	**1140**	Verschoor et al. (2006)
Vanadium	309	Kreule and Swartjes (1998)

Table A3.3: Water solubility (S).

Compound	**Water solubility [mg/l]**	**Water solubility [mg/l] at 10 °C**	**Reference**
Dibutyltin dichloride	92 (at 20 °C)	60.93	Van Herwijnen (2012)
Di-n-octyltin	0.23 (estimate 20 °C)	0.15	RPA (2005)
Tributyltin chloride	17 (at 20 °C)	11.26	Van Herwijnen (2012)
Tributyltin hydride	5.3 (at 25 °C)	3.96	Van Herwijnen (2012)
Tributyltin oxide	4 (at 20 °C)	2.65	Van Herwijnen (2012)
Triphenyltin acetate	9 (at 20 °C)	5.96	Van Herwijnen (2012)
Triphenyltin chloride	40 (at 20 °C)	26.49	Van Herwijnen (2012)
Triphenyltin hydroxide	0.4 (at 25 °C)	0.30	Van Herwijnen (2012)
Cis-1,2-dichloroethene	4561 [a] (at 25 °C)	3405	Fleuren et al. (2009), De Jong et al. (2011)
Trans-1,2-dichloroethene	6286 [a] (at 25 °C)	4694	Fleuren et al. (2009), De Jong et al. (2011)
Free cyanide	1×10^6 (at 25 °C)[b]	7.5×10^5	EC, in prep./ EPIWIN, exp.
Thiocyanate	5.8×10^5 (estimate 25 °C)[c]	4.3×10^5	SRC
Chloride	$>3.0 \times 10^5$ (estimate 10 °C)	$>3.0 \times 10^5$	Verbruggen et al. (2008)

Sulphate	2.8×10^5 (at 25 °C)	2.1×10^5	CRC-handbook

[a] Geomean of experimental values for 25 °C, the geomean for *cis*- and *trans* together is 5354 mg/L. In De Jong et al. (2011) a geometric mean for all values of *cis* and *trans* is reported of 3810 mg/L; this value could not be reproduced.
[b] Temperature not reported in EPIWIN of SRC; this is here not really relevant considering the high solubility. EC (in prep.). Free cyanide – prepared under the Water Framework Directive. European Commission, Brussels.
[c] Temperature not reported in EPIWIN or SRC; not really relevant here considering the high solubility.

Table A3.4: Vapour pressure (V_p).

Compound	**Vapour pressure [Pa]**	**Vapour pressure [Pa] at 10 °C**	**Reference**
Dibutyltin dichloride	0.16 (at 25 °C)	0.06	Van Herwijnen (2012)
Di-n-octyltin	0.095 (at 25 °C)	0.04	RPA (2005)
Tributyltin chloride	30 (at 20 °C)	15.6	Van Herwijnen (2012)
Tributyltin hydride	766.6 (at 25 °C)	293	Van Herwijnen (2012)
Tributyltin oxide	0.001 (at 20 °C)	5.2×10^{-4}	Van Herwijnen (2012)
Triphenyltin acetate	3.8×10^{-5} (at 20 °C)	2.0×10^{-5}	Van Herwijnen (2012)
Triphenyltin chloride	8×10^{-4} (at 25 °C)	3.06×10^{-4}	Van Herwijnen (2012)
Triphenyltin hydroxide	4.7×10^{-5} (at 25 °C)	1.8×10^{-5}	Van Herwijnen (2012)
Cis-1,2-dichloroethene	2.6×10^4 [a] (at 25 °C)	9940	De Jong et al. (2011)
Trans-1,2-dichloroethene	4.2×10^4 [a] (at 25 °C)	1.6×10^4	De Jong et al. (2011)
Free cyanide	9.9×10^4 (at 25 °C)	3.8×10^4	EC, in prep.
Thiocyanate	0.021 (at 25 °C)[b]	8.03×10^{-3}	EPIWIN, exp.
Chloride	n.a.	n.a.	Verbruggen et al. (2008)
Sulphate	2.3×10^{-17} (at 25 °C)	$8.8. \times 10^{-18}$	EPIWIN, estimated

n.a. = not applicable.

[a] geomean of experimental values for 25 °C; the geomean for *cis*- and *trans* together is 34004 Pa.
[b] For the experimental value no temperature is given; it is presumed that this value is for 25°C because a lower temperature would deviate even more from the estimated value.

Table A3.5: Henry's law constant (H).

Compound	**Henry constant [Pa.m^3/mol]**	**Henry constant [-] at 10 °C**	**Reference**
Dibutyltin dichloride	1.38 (at 25 °C)	1.3 x 10^{-4}	Van Herwijnen (2012)
Di-n-octyltin oxide	149 (at 25 °C?)	0.04	Calculated
Tributyltin chloride	574 (at 20 °C)	0.19	Van Herwijnen (2012)
Tributyltin hydride	4.2 x 10^{4} (at 25 °C)	9.16	Van Herwijnen (2012)
Tributyltin oxide	0.15 (at 20 °C)	5.0 x 10^{-5}	Van Herwijnen (2012)
Triphenyltin acetate	0.0017 (at 20 °C)	5.8 x 10^{-7}	Van Herwijnen (2012)
Triphenyltin chloride	0.0036 (at 20 °C)	1.89 x 10^{-6}	Van Herwijnen (2012)
Triphenyltin hydroxide	43 (at 25 °C)	9.4 x 10^{-6}	Van Herwijnen (2012)
Cis-1,2-dichloroethene	373.9[a] (at 20 °C)	0.12	De Jong et al. (2007)
Trans-1,2-dichloroethene	894.5[a] (at 20 °C)	0.14	De Jong et al. (2007)
Free cyanide	2.58 (at 25 °C)	5.6 x 10^{-4}	Calculated
Thiocyanate	2.1 x 10^{-6} (at 25 °C)	4.6 x 10^{-10}	Calculated
Chloride	n.a.	n.a.	Verbruggen et al. (2008)
Sulphate	7.9 x 10^{-21} (at 25°C)	1.7 x 10^{-24}	Calculated

[a] geomean of experimental values for 20 °C.

Table A3.6: Octanol-water partition coefficient (Log K_{ow}).

Compound	Log K_{ow} [-]	Reference
Dibutyltin dichloride	1.56	Van Herwijnen (2012)
Di-n-octyltin oxide	8	RPA (2005)
Tributyltin chloride	4.25	Van Herwijnen (2012)
Tributyltin hydride	4.1	Van Herwijnen (2012)
Tributyltin oxide	3.84	Van Herwijnen (2012)
Triphenyltin acetate	3.43	Van Herwijnen (2012)
Triphenyltin chloride	4.19	Van Herwijnen (2012)
Triphenyltin hydroxide	3.53	Van Herwijnen (2012)
Cis-1,2-dichloroethene	1.86	De Jong et al. (2007)
Trans-1,2-dichloroethene	2.09	De Jong et al. (2007)
Free cyanide	-0.25	EPIWIN, exp.
Thiocyanate	-2.52	EPIWIN, estimated
Chloride	-	Verbruggen et al. (2008)
Sulphate	-[a]	EPIWIN

[a] EPIWIN gives an estimated log Kow of -4.38.

Table A3.7: Organic carbon normalized soil-water partition coefficient (Log K_{oc}).

Compound	Log K_{oc} [-]	Reference
Dibutyltin dichloride	4.62	Van Herwijnen (2012)
Di-n-octyltin oxide	6.6	RPA (2005)
Tributyltin chloride	4.6	Van Herwijnen (2012)
Tributyltin hydride	4.1	Van Herwijnen (2012)
Tributyltin oxide	4.5	Van Herwijnen (2012)
Triphenyltin acetate	3.3	Van Herwijnen (2012)
Triphenyltin chloride	4.8 [a]	Van Herwijnen (2012)
Triphenyltin hydroxide	4.0 [b]	Van Herwijnen (2012)
Cis-1,2-dichloroethene	1.61	De Jong et al. (2007)
Trans-1,2-dichloroethene	1.78	De Jong et al. (2007)
Free cyanide	1.18[c]	EC in preparation
Thiocyanate	[c]	Otte et al. (2001)
Chloride	n.a.[c]	Verbruggen et al. (2008)
Sulphate	[c]	-

[a] Average of 5 experimental Koc values.
[b] Average of 2 experimental Koc values.

[c] Due to the high solubility of the compound is assumed that it is completely dissolved in (pore) water.

Table A3.8: Bioconcentration factors (BCF) for metals and metalloids. Bold means new value.

Compound	**BCF roots [mg.kg^{-1} dw_{crop}]/ [mg.kg^{-1} dw_{soil}]**	**BCF leaves [mg.kg^{-1} dw_{crop}]/ [mg.kg^{-1} dw_{soil}]**	**Reference**
Metals and metalloids			
Antimony	0.6[a]	0.9[a]	Van den Berg et al. (1994)
Barium	0.005[b]	**0.037**	Otte et al. (2001)/**Romkens and Rietra (2011)**
Boron	0.6[a]	2.1[a]	Van den Berg et al. (1994)
Selenium	0.2[a]	0.37[a]	Kreule and Swartjes (1998)
Thallium	0.095	0.013[a]	Kreule and Swartjes (1998)
Tin (inorganic)	4 x 10^{-4}	0.0045[a]	Kreule and Swartjes (1998)
Vanadium	0.006[c]	0.006[c]	Kreule and Swartjes (1998)

[a] Measured. [b] Estimated. [c] Calculated.

Table A3.9: Bioconcentration factors (BCF) for organic compounds (calculated in CSOIL).

Compound	**BCF roots [mg/kg fw]/[mg/L]**	**BCF leaves [mg/kg fw]/[mg/L]**
Dibutyltin dichloride	0.92	0.018
Di-n-octyltin oxide	-	-
Tributyltin chloride	13.4	0.001
Tributyltin hydride	10.4	3.4 x 10^{-4}
Tributyltin oxide	6.7	1.4
Triphenyltin acetate	3.6	10.1
Triphenyltin chloride	12.1	7.5
Triphenyltin hydroxide	4.2	3.5
Cis-1,2-dichloroethene	0.99	3.4 x 10^{-5}
Trans-1,2-dichloroethene	1.07	3.9 x 10^{-5}

Annex 4: Output CSOIL calculations defaults scenario

Table A4.1 Output CSOIL, SRC_{human} and contribution of different exposurepathways.

Contribution (in %) of the different exposure pathways to total exposure

Land use residential with garden

Receptor lifelong average

contaminant	SRC human mg/kg d.s.	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
antimoon	151,67	3,1%	0,0%	0,0%	0,0%	0,0%	0,0%	96,9%	0,0%	0,0%	0,0%
barium	7169,99	56,3%	0,0%	0,0%	0,4%	0,0%	0,0%	43,3%	0,0%	0,0%	0,0%
boron	3056,16	1,9%	0,0%	0,0%	0,0%	0,0%	0,0%	98,1%	0,0%	0,0%	0,0%
selenium	322,94	7,9%	0,0%	0,0%	0,1%	0,0%	0,0%	92,0%	0,0%	0,0%	0,0%
thallium	8,74	26,8%	0,0%	0,0%	0,2%	0,0%	0,0%	73,0%	0,0%	0,0%	0,0%
tin	149068,48	91,3%	0,0%	0,0%	0,7%	0,0%	0,0%	8,0%	0,0%	0,0%	0,0%
vanadium	1244,55	78,8%	0,0%	0,0%	0,6%	0,0%	0,0%	20,6%	0,0%	0,0%	0,0%
dibutyltin (DBT) dichloride	149,59	73,3%	0,5%	6,4%	0,6%	0,4%	0,0%	17,1%	1,8%	0,0%	0,0%
tributyltin (TBT) chloride	0,52	8,6%	0,1%	0,7%	0,1%	80,9%	0,0%	9,3%	0,2%	0,0%	0,1%
tributyltin (TBT) hydride	3,47E-03	0,1%	0,0%	0,0%	0,0%	99,7%	0,0%	0,2%	0,0%	0,0%	0,0%
tributyltin (TBT) oxide	83,12	49,5%	0,3%	4,3%	0,4%	0,2%	0,0%	43,8%	1,6%	0,0%	0,0%
triphenyltin (TPT) acetate	9,26	4,5%	0,0%	0,4%	0,0%	0,0%	0,0%	92,6%	2,3%	0,0%	0,1%
triphenyltin (TPT) chloride	94,67	46,4%	0,3%	4,0%	0,4%	0,0%	0,0%	48,1%	0,8%	0,0%	0,2%
triphenyltin (TPT) hydroxide	57,04	27,9%	0,2%	2,4%	0,2%	0,1%	0,0%	66,1%	2,9%	0,0%	0,2%
cis-1,2-dichloroethene	2,74	0,0%	0,0%	0,0%	0,0%	98,8%	0,0%	1,1%	0,0%	0,0%	0,0%
trans-1,2-dichloroethene	3,36	0,0%	0,0%	0,0%	0,0%	98,9%	0,0%	1,0%	0,0%	0,0%	0,0%

Table A4.2 Contaminant concentrations in relevant compartments.

CONTAMINANT CONCENTRATIONS IN RELEVANT ENVIRONMENTAL COMPARTMENTS

Land use residential with garden
Receptor lifelong average

contaminant	total soil (mg/kg dm)	pore water (mg/dm3)	pore air (mg/dm3)	groundwater (ug.dm-3)	(an)organics C plant-root (mg/kg fw)	(an)organics C plant-leafe (mg/kg fw)	metals C potato (mg/kg fw)	metals C vegetables (mg/kg fw)	indoor air (mg.m-3)	C outdoor air child mg.dm-3	C outdoor air adult mg.dm-3
antimoon	1,517E+02	1,78E+00	0,00E+00	1,78E+03			1,52E+01	1,34E+01	0,00E+00	0,00E+00	0,00E+00
barium	7,170E+03	2,87E+00	0,00E+00	2,87E+03			5,99E+00	2,60E+01	0,00E+00	0,00E+00	0,00E+00
boron	3,056E+03	1,07E+03	0,00E+00	1,07E+06			3,06E+02	6,29E+02	0,00E+00	0,00E+00	0,00E+00
selenium	3,229E+02	2,12E+01	0,00E+00	2,12E+04			1,08E+01	1,17E+01	0,00E+00	0,00E+00	0,00E+00
thallium	8,743E+00	5,46E-02	0,00E+00	5,46E+01			1,39E-01	1,11E-02	0,00E+00	0,00E+00	0,00E+00
tin	1,491E+05	1,31E+02	0,00E+00	1,31E+05			9,96E+00	6,57E+01	0,00E+00	0,00E+00	0,00E+00
vanadium	1,245E+03	4,02E+00	0,00E+00	4,02E+03			1,25E+00	7,32E-01	0,00E+00	0,00E+00	0,00E+00
dibutyltin (DBT) dichloride	1,496E+02	6,19E-02	7,87E-06	6,19E+01	5,70E-02	1,48E-01	-	-	3,82E-06	5,31E-11	2,64E-11
tributyltin (TBT) chloride	5,205E-01	2,25E-04	4,32E-05	2,25E-01	3,02E-03	5,10E-04	-	-	2,10E-05	6,99E-11	3,47E-11
tributyltin (TBT) hydride	3,475E-03	4,75E-06	4,34E-05	4,75E-03	4,92E-05	3,41E-06	-	-	2,11E-05	7,42E-11	3,69E-11
tributyltin (TBT) oxide	8,312E+01	4,53E-02	2,25E-06	4,53E+01	3,05E-01	1,44E-01	-	-	1,09E-06	2,36E-11	1,17E-11
triphenyltin (TPT) acetate	9,262E+00	7,99E-02	4,66E-08	7,99E+01	2,88E-01	8,18E-01	-	-	4,45E-08	4,45E-11	2,21E-11
triphenyltin (TPT) chloride	9,467E+01	2,59E-02	4,90E-08	2,59E+01	3,12E-01	2,78E-01	-	-	2,38E-08	1,49E-11	7,40E-12
triphenyltin (TPT) hydroxide	5,704E+01	9,83E-02	9,20E-07	9,83E+01	4,10E-01	4,01E-01	-	-	4,47E-07	5,91E-11	2,94E-11
cis-1,2-dichloroethene	2,743E+00	1,04E+00	1,25E-01	1,04E+03	1,03E+00	2,72E-03	-	-	6,29E-02	3,72E-07	1,85E-07
trans-1,2-dichloroethene	3,363E+00	8,92E-01	1,25E-01	8,92E+02	9,53E-01	3,33E-03	-	-	6,30E-02	3,72E-07	1,85E-07

Table A4.3 Compound specific parameters.

SRC HUMAN AND COMPOUND SPECIFIC PARAMETERS

Land use residential with garden

Receptor lifelong average

contaminant	HUM-TOX EBVC	M	S	Vp	log Kow	log Koc	Kd	Dpe	BCF-org.	BCF-org.	BCF-metal	BCF-metal	groundwater	TCL	MPR
	mg/kg d.s.	g.mol-1	mg/dm3	Pa			metals		root crops	vegetables	potatoes	vegetables	max ug/dm3	mg/m3	mg/kg.d
antimoon	151,67	1,22E+02	3,00E+03	n.a.	n.a.	n.a.	8,50E+01	n.a.	-	-	6,00E-01	9,00E-01	1,88E+02	N.A.	6,00E-03
barium	7169,99	1,37E+02	3,00E+03	n.a.	n.a.	n.a.	2,50E+03	n.a.	-	-	5,00E-03	3,70E-02	6,28E+02	1,00E-03	2,00E-02
boron	3056,16	1,08E+01	3,00E+03	n.a.	n.a.	n.a.	2,60E+00	n.a.	-	-	6,00E-01	2,10E+00	6,28E+03	1,00E-02	2,00E-01
selenium	322,94	7,90E+01	3,00E+03	n.a.	n.a.	n.a.	1,50E+01	n.a.	-	-	2,00E-01	3,70E-01	1,57E+02	N.A.	5,00E-03
thallium	8,74	2,04E+02	3,00E+03	n.a.	n.a.	n.a.	1,60E+02	n.a.	-	-	9,50E-02	1,30E-02	1,26E+00	N.A.	4,00E-05
tin	149068,48	1,19E+02	3,00E+03	n.a.	n.a.	n.a.	1,14E+03	n.a.	-	-	4,00E-04	4,50E-03	6,28E+03	N.A.	2,00E-01
vanadium	1244,55	5,09E+01	3,00E+03	n.a.	n.a.	n.a.	3,09E+02	n.a.	-	-	6,00E-03	6,00E-03	6,28E+01	1,00E-03	2,00E-03
dibutyltin (DBT) dichloride	149,59	3,04E+02	6,09E+01	6,00E-02	1,56E+00	4,62E+00	n.a.	5,00E-07	9,22E-01	1,80E-02	n.a.	n.a.	7,85E+00	N.A.	2,50E-04
tributyltin (TBT) chloride	0,52	3,26E+02	1,13E+01	1,56E+01	4,25E+00	4,60E+00	n.a.	5,00E-07	1,34E+01	1,18E-03	n.a.	n.a.	7,85E+00	2,00E-05	2,50E-04
tributyltin (TBT) hydride	3,47E-03	2,91E+02	3,96E+00	2,93E+02	4,10E+00	4,10E+00	n.a.	5,00E-07	1,04E+01	3,37E-04	n.a.	n.a.	7,85E+00	2,00E-05	2,50E-04
tributyltin (TBT) oxide	83,12	5,96E+02	2,65E+00	5,20E-04	3,84E+00	4,50E+00	n.a.	5,00E-07	6,73E+00	1,38E+00	n.a.	n.a.	7,85E+00	2,00E-05	2,50E-04
triphenyltin (TPT) acetate	9,26	4,09E+02	5,96E+00	2,00E-05	3,43E+00	3,30E+00	n.a.	5,00E-07	3,61E+00	1,01E+01	n.a.	n.a.	7,85E+00	N.A.	2,50E-04
triphenyltin (TPT) chloride	94,67	3,86E+02	2,65E+01	3,06E-04	4,19E+00	4,80E+00	n.a.	5,00E-07	1,21E+01	7,16E+00	n.a.	n.a.	7,85E+00	N.A.	2,50E-04
triphenyltin (TPT) hydroxide	57,04	3,67E+02	3,00E-01	1,80E-05	3,53E+00	4,00E+00	n.a.	5,00E-07	4,17E+00	3,51E+00	n.a.	n.a.	7,85E+00	N.A.	2,50E-04
cis-1,2-dichloroethene	2,74	9,69E+01	3,41E+03	9,94E+03	1,86E+00	1,61E+00	n.a.	4,00E-08	9,87E-01	3,39E-05	n.a.	n.a.	9,42E+02	6,00E-02	3,00E-02
trans-1,2-dichloroethene	3,36	9,69E+01	4,69E+03	1,60E+04	2,09E+00	1,78E+00	n.a.	4,00E-08	1,07E+00	3,86E-05	n.a.	n.a.	9,42E+02	6,00E-02	3,00E-02

Annex 5: Output CSOIL specific scenario's Free cyanide and Thiocyanate

Table A5.1 Output CSOIL for free cyanide with default scenario but without vegetable consumption.

username	Gebruiker	**date**	28 november 2008
model	CSOIL	**release**	1.0
filename	C:\Users\Jet\Documents\Mijn Projecten\RIVM\CSoil 1.0 28-11-2008.xls		
contaminant	**cyanides (free)**	**CASnr.**	0
Land use	**residential with garden**	**considered receptor**	lifelong average
remarks			

HUMAN RISK LIMITS

SRC human	69,623	
risk index	1,00	(lifelong average)
soil content	6,962E+01	mg/kg ds
C gw-max	1,57E+03	ug/dm3

AVERAGE DAILY EXPOSURE FOR CHILDREN, ADULTS AND LIFELONG

in mg/kg l.g. *d

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	4,64E-04	1,43E-06	2,84E-05	1,09E-06	9,09E-03
adult	4,97E-05	4,47E-07	5,42E-06	6,22E-07	5,54E-03
lifelong average	8,53E-05	5,31E-07	7,39E-06	6,62E-07	5,85E-03
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	2,02E-05	0,00E+00	1,88E-02	5,83E-04	9,36E-05
adult	2,25E-06	0,00E+00	8,06E-03	3,29E-04	3,80E-05
lifelong average	3,79E-06	0,00E+00	8,98E-03	3,51E-04	4,28E-05

CONTRIBUTION (IN %) OF THE DIFFERENT EXPOSURE PATHWAYS TO TOTAL EXPOSURE CHILDREN, ADULT AND LIFELONG

in %

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	1,60%	0,00%	0,10%	0,00%	31,26%
adult	0,35%	0,00%	0,04%	0,00%	39,53%
lifelong average	0,56%	0,00%	0,05%	0,00%	38,19%
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	0,07%	0,00%	64,64%	2,01%	0,32%
adult	0,02%	0,00%	57,44%	2,35%	0,27%
lifelong average	0,02%	0,00%	58,61%	2,29%	0,28%

CONTAMINANT CONCENTRATIONS IN RELEVANT ENVIRONMENTAL COMPARTMENTS

total soil (mg/kg dm)	6,96E+01	(an)organics C plant-leaf	1,02E-01	indoor air (mg.m-3)	2,04E-02
pore water (mg/dm3)	6,18E+01	(an)organics C plant-root	5,17E+01	crawl space (mg.m-3)	2,04E-01
pore air (mg/dm3)	3,46E-02	(an)organics C plant-leaf	-		
		(an)organics C plant-root	-		

Table A5.2 Output CSOIL for free cyanide, exposure by single ingestion of 5 gr soil for a child and without vegetable consumption.

username	Gebruiker	**date**	28 november 2008
model	CSOIL	**release**	1.0
filename	C:\Users\Jet\Documents\Mijn Projecten\RIVM\CSoil 1.0 28-11-2008.xls		
contaminant	**cyanides (free)**	**CASnr.**	0
Land use	**land use to define**	**considered receptor**	child
remarks			

HUMAN RISK LIMITS

SRC human	43,296	
risk index	1,00	(child)
soil content	4,330E+01	mg/kg ds
C gw-max	1,57E+03	ug/dm3

AVERAGE DAILY EXPOSURE FOR CHILDREN, ADULTS AND LIFELONG

in mg/kg l.g. *d

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	1,44E-02	8,86E-07	1,77E-05	6,77E-07	5,65E-03
adult	3,09E-05	2,78E-07	3,37E-06	3,87E-07	3,45E-03
lifelong average	1,44E-02	8,86E-07	1,77E-05	6,77E-07	5,65E-03
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	1,25E-05	0,00E+00	1,17E-02	3,63E-04	5,82E-05
adult	1,40E-06	0,00E+00	5,01E-03	2,05E-04	2,36E-05
lifelong average	1,25E-05	0,00E+00	1,17E-02	3,63E-04	5,82E-05

CONTRIBUTION (IN %) OF THE DIFFERENT EXPOSURE PATHWAYS TO TOTAL EXPOSURE CHILDREN, ADULT AND LIFELONG

in %

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	44,78%	0,00%	0,05%	0,00%	17,54%
adult	0,35%	0,00%	0,04%	0,00%	39,53%
lifelong average	44,78%	0,00%	0,05%	0,00%	17,54%
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	0,04%	0,00%	36,27%	1,13%	0,18%
adult	0,02%	0,00%	57,44%	2,35%	0,27%
lifelong average	0,04%	0,00%	36,27%	1,13%	0,18%

CONTAMINANT CONCENTRATIONS IN RELEVANT ENVIRONMENTAL COMPARTMENTS

total soil (mg/kg dm)	4,33E+01	(an)organics C plant-leaf	6,34E-02	indoor air (mg.m-3)	1,27E-02
pore water (mg/dm3)	3,85E+01	(an)organics C plant-root	3,22E+01	crawl space (mg.m-3)	1,27E-01
pore air (mg/dm3)	2,15E-02	(an)organics C plant-leaf	-		
		(an)organics C plant-root	-		

Table A5.3 Output CSOIL for thiocyanate with default scenario but without vegetable consumption or inhaltion of vapours.

username	Gebruiker	**date**	28 november 2008
model	CSOIL	**release**	1.0
filename	C:\Users\Jet\Documents\Mijn Projecten\RIVM\CSoil 1.0 28-11-2008.xls		
contaminant	**thiocyanate**	**CASnr.**	0
Land use	**residential with garden**	**considered receptor**	lifelong average
remarks			

HUMAN RISK LIMITS

SRC human	8914,143	
risk index	1,00	(lifelong average)
soil content	8,914E+03	mg/kg ds
C gw-max	3,46E+02	ug/dm3

AVERAGE DAILY EXPOSURE FOR CHILDREN, ADULTS AND LIFELONG

in mg/kg l.g. *d

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	5,94E-02	0,00E+00	0,00E+00	1,39E-04	0,00E+00
adult	6,37E-03	0,00E+00	0,00E+00	7,96E-05	0,00E+00
lifelong average	1,09E-02	0,00E+00	0,00E+00	8,47E-05	0,00E+00
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	0,00E+00	0,00E+00	0,00E+00	0,00E+00	0,00E+00
adult	0,00E+00	0,00E+00	0,00E+00	0,00E+00	0,00E+00
lifelong average	0,00E+00	0,00E+00	0,00E+00	0,00E+00	0,00E+00

CONTRIBUTION (IN %) OF THE DIFFERENT EXPOSURE PATHWAYS TO TOTAL EXPOSURE CHILDREN, ADULT AND LIFELONG

in %

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	99,77%	0,00%	0,00%	0,23%	0,00%
adult	98,77%	0,00%	0,00%	1,23%	0,00%
lifelong average	99,23%	0,00%	0,00%	0,77%	0,00%
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	0,00%	0,00%	0,00%	0,00%	0,00%
adult	0,00%	0,00%	0,00%	0,00%	0,00%
lifelong average	0,00%	0,00%	0,00%	0,00%	0,00%

CONTAMINANT CONCENTRATIONS IN RELEVANT ENVIRONMENTAL COMPARTMENTS

total soil (mg/kg dm)	8,91E+03	(an)organics C plant-leaf	3,22E+04	indoor air (mg.m-3)	0,00E+00
pore water (mg/dm3)	3,57E+04	(an)organics C plant-root	2,97E+04	crawl space (mg.m-3)	0,00E+00
pore air (mg/dm3)	0,00E+00	(an)organics C plant-leaf	-		
		(an)organics C plant-root	-		

Table A5.4 Output CSOIL for thiocyanate, exposure by a single ingestion of 5 gr soil for a child and without vegetable consumption and inhalation of soil vapours.

username	Gebruiker	**date**	28 november 2008
model	CSOIL	**release**	1.0
filename	C:\Users\Jet\Documents\Mijn Projecten\RIVM\CSoil 1.0 28-11-2008.xls		
contaminant	**thiocyanate**	**CASnr.**	0
Land use	**land use to define**	**considered receptor**	child
remarks			

HUMAN RISK LIMITS

SRC human	32,998	
risk index	1,00	(child)
soil content	3,300E+01	mg/kg ds
C gw-max	3,46E+02	ug/dm3

AVERAGE DAILY EXPOSURE FOR CHILDREN, ADULTS AND LIFELONG

in mg/kg l.g. *d

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	1,10E-02	0,00E+00	0,00E+00	5,16E-07	0,00E+00
adult	2,36E-05	0,00E+00	0,00E+00	2,95E-07	0,00E+00
lifelong average	1,10E-02	0,00E+00	0,00E+00	5,16E-07	0,00E+00
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	0,00E+00	0,00E+00	0,00E+00	0,00E+00	0,00E+00
adult	0,00E+00	0,00E+00	0,00E+00	0,00E+00	0,00E+00
lifelong average	0,00E+00	0,00E+00	0,00E+00	0,00E+00	0,00E+00

CONTRIBUTION (IN %) OF THE DIFFERENT EXPOSURE PATHWAYS TO TOTAL EXPOSURE CHILDREN, ADULT AND LIFELONG

in %

	soil intake	dermal uptake indoor	dermal uptake outdoor	inhalation of soil particles	inhalation of indoor air
child	100,00%	0,00%	0,00%	0,00%	0,00%
adult	98,77%	0,00%	0,00%	1,23%	0,00%
lifelong average	100,00%	0,00%	0,00%	0,00%	0,00%
	inhalation of outdoor air	consumption from own garden	consumption of drinking water	inhalation during showering	dermal uptake during bathing
child	0,00%	0,00%	0,00%	0,00%	0,00%
adult	0,00%	0,00%	0,00%	0,00%	0,00%
lifelong average	0,00%	0,00%	0,00%	0,00%	0,00%

CONTAMINANT CONCENTRATIONS IN RELEVANT ENVIRONMENTAL COMPARTMENTS

total soil (mg/kg dm)	3,30E+01	(an)organics C plant-leaf	1,19E+02	indoor air (mg.m-3)	0,00E+00
pore water (mg/dm3)	1,32E+02	(an)organics C plant-root	1,10E+02	crawl space (mg.m-3)	0,00E+00
pore air (mg/dm3)	0,00E+00	(an)organics C plant-leaf	-		
		(an)organics C plant-root	-		

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