(Contract 041757)
EVALUATION OF THE SWEDISH
GUIDELINE VALUES FOR CONTAMINATED SITES
- CADMIUM AND POLYCYCLIC AROMATIC HYDROCARBONS -

TECHNICAL REPORT

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Study accomplished under the authority of JM

2006/IMS/R/390

VITO   **RIVM**

November 2006

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LIST OF ABBREVIATIONS AND SYMBOLS

This list only applies to abbreviations and symbols used in the main report. In the appendices more abbreviations and symbols can be found.

a_i	regression constant in the Freundlich-like plant-soil relation [-]
API	American Petroleum Institute (USA)
ATSDR	Agency for Toxic Substances and Disease Registry (USA)
$AWQC$	Ambient Water Quality Criteria for fish consumption from fresh water [mg/l]
$BCF_{(x)}$	bioconcentration factor for stem or root crops, can be expressed in different dimensions: [(mg/kg fresh plant)/(mg/l pore water)], [(mg/kg fresh plant)/(mg/kg soil dw)]or [(mg/kg dry plant)/(mg/kg soil dw)]
b_i	regression constant in the Freundlich-like plant-soil relation [-]
bw	body weight [kg]
C	custom distribution
C_a	vapour concentration in the pore air [mg/dm³]
C_{ad}	annual average concentration in inhailed air [mg/m³]
CB	concrete basement
CCME	Canadian Council of Ministers for the Environment (Canada)
C_{du}	reference soil concentration for the dermal pathway [mg/kg]
CF	concrete floor (slab-on-grade)
C_{gw}	concentration in groundwater [mg/l]
c_i	regression constant in the Freundlich-like plant-soil relation [-]
C_{ia}	concentration in indoor air [mg/dm³]
C_{id}	reference soil concentration for the dust inhalation pathway [mg/kg dw]
C_{if}	reference soil concentration for for the fish consumption pathway [mg/kg dw]
C_{ig}	reference soil concentration for the vegetable consumption pathway [mg/kg dw]
C_{is}	reference soil concentration for the soil ingestion pathway [mg/kg dw]
C_{iv}	reference soil concentration for the vapour inhalation pathway [mg/kg dw]
C_{iw}	reference soil concentration for the drinking water pathway [mg/kg dw]
C_{KM}	integrated human health based value for land with sensitive land-use [mg/kg dw]
CL	clay loam
$clay\%$	clay content of the soil [%]
CLEA	Contaminated Land Exposure Assessment (UK)
C_{MKM}	integrated human human health based value for land with less sensitive land-use and no groundwater extraction [mg/kg dw]
$C_{MKM\,GV}$	integrated human human health based value for land with less sensitive land-use and groundwater extraction [mg/kg dw]
CR	initial soil concentration (Johnson & Ettinger) [µg/kg dw]
C_s	total concentration in soil [mg/kg dw]
C_{sw}	total concentration in surface water [mg/l]
C_v	total plant concentration [mg/kg fw]
C_w	concentration in soil pore water [mg/l]
CWQC	Canadian Water Quality Criteria for freshwater aquatic life [mg/l]
d_a	thickness of the phreatic aquifer [m]

DF_{gw}	dilution factor soil pore water to groundwater [-]
DF_{ia}	dilution factor indoor air to soil air [-]
DF_{sw}	dilution factor groundwater to surface water [-]
d_i	regression constant in the Freundlich-like plant-soil relation [-]
d_{mix}	thickness of the mixing zone in the aquifer [m]
DP	soil-building pressure differential (Johnson & Ettinger) [g/cm.s]
dw	dry weight
dw	ratio dry weight to fresh weight [kg dw/kg fw]
DWG	drinking water guideline [mg/l]
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
e_i	regression constant in the Freundlich-like plant-soil relation [-]
E_{KM}	ecotoxicological values for on-site effects, for land with sensitive land-use [mg/kg dw]
E_{MKM}	ecotoxicological values for on-site effects, for land with less sensitive land-use [mg/kg dw]
ER	indoor air exchange rate (Johnson & Ettinger) [h^{-1}]
E_{sw}	ecotoxicological value for the aquatic system [mg/kg dw]
EUSES	European Union System for the Evaluation of Substances
f_{du}	substance specific relative absorption factor for dermal uptake [-]
f_{exp}	fraction of time spent on the site [-]
f_h	fraction of vegetables grown on the site [-]
f_i	regression constant in the Freundlich-like plant-soil relation [-]
f_{leaf}	fractional consumption of leaf and stem vegetables in the total vegetable consumption, calculated on dry weight basis [-]
f_{OC}	organic carbon content in soil [%]
f_{OM}	organic matter content in soil [%]
f_{root}	fractional consumption of root crops in the total vegetable consumption, calculated on dry weight basis [-]
fw	fresh weight
GI	gastrointestinal
H	Henry's law constant, c.q. the dimensionless Henry coefficient [-]
h_A	thickness of soil stratum [cm]
H_B	enclosed space width (Johnson & Ettinger) [cm]
HESP	Human Exposure to Soil Pollutants
i	hydraulic gradient [m/m]
I	infiltration rate [m/yr]
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System (US-EPA)
k	hydraulic conductivity of the soil [m/yr]
K_{AW}	air-water partition coefficient, i.e. the dimensionless Henry-coefficient [-]
K_d	distribution coefficient soil-water [l/kg]
K_H	Henry's coefficient [Pa m^3/mol]
KM	Känslig Markanvändning; refers to land with sensitive land-use; all types of land use can be permitted
K_{OA}	octanol-air partition coefficient [l/kg]
K_{OC}	partitioning coefficient organic carbon-water [l/kg]
K_{OW}	partitioning coefficient octanol-water [l/kg]
K_{pl}	total plant concentration factor [(mg/kg fresh plant)/(mg/kg dry soil)]
k_t	turnover rate of the lake [yr^{-1}]

K_v	soil vapour permeability (Johnson & Ettinger) [cm²]
L	length of the contaminated area in the direction of the groundwater flow [m]
L_B	enclosed space floor length (Johnson & Ettinger) [cm]
L_{crack}	enclosed space floor thickness (Johnson & Ettinger) [cm]
L_F	depth below grade to bottom of enclosed space floor (Johnson & Ettinger) [cm]
LN	log-normal distribution
LSA	leaf surface area [m²];
L_w	width of the contaminated area perpendicular to the direction of the groundwater flow [m]
L_T	depth below grade to top of contamination (Johnson & Ettinger) [cm]
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MDEP	Massachusetts Department of Environmental Protection (USA)
MKM	Mindre Känslig Markanvändning; refers to land with less sensitive land-use but with no groundwater extraction
MKM GV	Mindre Känslig Markanvändning med GrundVattenskydd; refers to land with less sensitive land-use and groundwater extraction
MOE	Ministry of the Environment (Canada)
MW	molecular weight [g/mol]
n	soil total porosity (Johnson & Ettinger) [-]
N	normal distribution
$OC\%$	organic carbon content of the soil [%]
P	vapour pressure [Pa]
PAH	Polycyclic Aromatic Hydrocarbons
$PTWI$	Provisional Tolerable Weekly Intake
Q_{di}	discharge of groundwater from the contaminated site to the surface water [m³/yr]
Q_{sw}	water flow rate in the surface water [m³/yr]
Q_{transp}	transpiration rate [m³/yr]
R	universal gas constant [Pa m³ /(mol K)]
RAF	relative absorption factor [-]
RfC	reference concentration [mg/m³]
RfD	reference dose [mg/kg.d]
RIVM	National Institute of Public Health and the Environment (the Netherlands)
RME	reasonably worst case
ρ_b	dry soil bulk density [kg/dm³]
R_{du}	average daily dermal exposure [mg/kg.d]
RfC	Reference Concentration in air [mg/m³]
RfD	Reference Dose [mg/kg.d]
R_{ig}	average daily consumption of vegetables [kg vegetables/kg bw.d]
R_{is}	average daily soil intake [mg/kg.d]
R_{id}	average daily inhalation of dust [mg/kg.d]
R_{iv}	average daily inhalation of vapour [(mg/kg.d)/(g/m³)]
R_{iw}	average daily water consumption [l/kg.d]
S	medium till fine sand
S-EPA	Swedish Environmental Protection Agency (Naturvårdsverket)
SETAC	Society of Environmental Toxicology and Chemistry

SRC_{eco}	ecotoxicological Serious Risk Concentration, as part of the Intervention Value from the Netherlands [mg/kg dw]
S-RISK	transfer and exposure model to calculate human health effects adapted to specific conditions in a delineated part of South-Sweden and based on the current Swedish methodology to derive generic guideline values for contaminated soil
S-RISK Excel	S-RISK model incorporated in an Excel environment; S-RISK Excel includes the S-EPA and S-RISK models and the S-EPA and S-RISK databases
SS	silty sand
SSL	Soil Screening Level (US-EPA)
T	triangular distribution
T	ambient temperature [K]
θ_a	soil air content [dm³ air/dm³ soil]
TCA	Tolerable Concentration in Air [mg/m³]
TDI	Tolerable Daily Intake [mg/kg.d]
TEF	Toxic Equivalence Factor [-]
TRV	Toxicological Reference Value [mg/kg.d]
T_s	average soil temperature (Johnson & Ettinger) [°C]
TSP	total suspended particles in air [mg/m³]
TSCF	transpiration stream concentration factor [-]
θ_t	total soil porosity [dm³/dm³]
θ_w	soil water content [dm³ water/dm³ soil]
U	uniform distribution
USDA	United States Department of Agriculture (USA)
US-EPA	United States Environmental Protection Agency (USA)
VITO	Flemish Institute for Technological Research (Flanders, Belgium)
Vlier-Humaan	Flemish instrument for the evaluation of human risks (Flanders, Belgium)
VOC	volatile organic contaminant
VOLASOIL	risk assessment model based on CSOIL for soils contaminated with volatile compounds (the Netherlands)
V_{sw}	volume of the lake [m³]
w	floor-wall seam crack width (Johnson & Ettinger) [cm]
W_B	enclosed space floor width (Johnson & Ettinger) [cm]
WHO	World Health Organization
W_p	the washout factor [-]
X	distance from the contaminated area to the well [m]

1   INTRODUCTION

1.1   Objectives

Guideline values for soil pollutants are used as a reference to decide if soils are fit for normal use or need special environmental attention or treatment. They try to reflect risk levels for predefined receptors, which can be humans, the ecosystem, or water bodies. With regard to the protection of human health, the guideline values are often based on predicted potential exposure of persons coming into contact with the soil either directly or indirectly. The exposure models are based on the state of scientific knowledge of the transfer processes of pollutants in soil, air, water, food, etc, and of the exposure pathways such as ingestion of soil and food, dermal contact with soil and water, inhalation of vapours and particles. Exposure models may show differences depending on interpretation of scientific information, local characteristics and political decisions.

Swedish Guideline Values for soil quality were developed in 1996, according to the procedure described in "report 4639" of the Swedish Environmental Protection Agency (Naturvårdsverket, 1996b). International comparisons (e.g EuroRisk study, SETAC 2004) show that the exposure model used in Sweden to propose these guideline values leads to relatively high exposure estimates for cadmium (Cd) and polycyclic aromatic hydrocarbons (PAHs). Hence, the PAH and Cd guideline values are in the lower range of European soil quality standards. As a consequence, it is often observed that diffuse enrichment of Swedish urban soils with cadmium and PAHs leads to measured concentrations exceeding the guideline values.

The Flemish Institute for Technological Research (VITO) and the National Institute of Public Health and the Environment (RIVM) were asked by JM AB to evaluate the Swedish Guideline Values for cadmium and polycyclic aromatic hydrocarbons with regard to the human health part. Therefore, the human exposure models, their parameter values and the toxicological reference values were evaluated to see whether they are still in line with the current state-of-the-art of contaminated sites risk assessment. This evaluation has resulted in the proposal of alternative soil quality guidelines for Cd and PAHs for the Swedish situation.

The project envisaged a revision of a predefined list of fate and transfer and of exposure equations within the basic concept of the present guideline value framework (Naturvårdsverket, 1996b). New parameter values for PAHs and Cd with regard to fate and transfer and toxicology are provided. The revision is limited to the human-health based values and excludes the ecotoxicology-based values. It should be reminded however, that there is a need to review the ecotoxicological values also with regard to their state-of-the-art.

The revised guideline values are not considered truly 'generic'. They are derived on the basis of (soil) data provided by JM, which are assumed to correspond with the properties of the filling material used on JM developed locations. Meteorological data were provided by JM for the geographical region where most of their projects are situated. This region is defined as the land south of the line going from Göteborg at the West Coast to Gävle at the East Coast.

1.2   Elements of evaluation

Four aspects of the Swedish guideline values are addressed:
−   evaluation of model equations and parameter values for transfer calculations:
  • transfer of vapour from soil to indoor air
  • transport from soil to groundwater (dilution factor);
  • transport of contaminants in plants;
  • emission of particles from soil to ambient air;
−   evaluation of model equations and parameter values for exposure calculations:
  • soil ingestion;
  • dermal contact;
  • vegetable consumption;
−   evaluation of contaminant-specific parameter values for use in the equations (PAHs and cadmium);
−   modification of the risk calculations (comparison of dose with toxicological reference value).

A new spreadsheet model, including the revisions, is developed. In this report, the model is referred to with the name S-RISK. The spreadsheet model is called S-RISK Excel.

1.3   Outline of this report

Chapter 2 discusses briefly the procedure which was used for the derivation of the current Swedish soil guideline values. In Chapter 3, the development of the S-RISK model is outlined. If the current model equations are altered, both non-adjusted and adjusted formulae are debated concisely. Chapter 4 briefly discusses the model for environmental risk assessment. Chapter 5 gives the revised parameter values for Cd and PAHs. Finally, the comparison between current generic guideline values and those calculated using S-RISK is provided in chapter 6. General conclusions are found in chapter 7. In the appendices, a more elaborated discussion is provided on the derivation of formulae and parameter values given in the main report. Also, an overview of the default parameter values used in the S-RISK model and compound specific properties (S-RISK database) are given in appendix H.

A summary report, presenting the main findings and results, is published separately.

2 CURRENT SWEDISH SOIL GUIDELINE VALUES METHODOLOGY

2.1 Introduction

The current Swedish soil quideline values have been based on a framework for the analysis of risks associated with contaminated soils (Naturvårdsverket, 1996b). These guideline values are intended to indicate critical contaminant concentrations, above which unacceptable effects for human health and/or the soil ecosystem may occur. They can also be used to indicate the degree of contamination on a site, to develop clean-up goals and to evaluate clean-up results.

2.2 Methodology for human health

The methodology used for the development of generic Swedish guideline values is based on the methodologies and data from the Netherlands: CSOIL (Van den Berg, 1991, 1995), HESP (ECETOC, 1990, 1992; SHELL, 1994), the USA: Massachusetts Department of Environmental Protection (MDEP, 1994), US-EPA Soil Screening Levels (US-EPA, 1996a), and Canada: CCME (1994), Ontario MOE (1994, 1996).

Models for estimation of soil guideline values typically address the following topics:
- distribution and transport of the contaminant in the environment;
- pathways for exposure of humans to the contaminant;
- estimation of human health risk from exposure;
- estimation of ecotoxicological risks.

The following transport pathways are considered in the current Swedish model:
- transport of vapour from the soil to indoor air;
- transport of contaminants to a groundwater well;
- transport of contaminants to surface waters;
- transport of contaminants to plants.

Based on a potential future use of the site a set of exposure pathways is defined. For each exposure pathway the exposure is estimated using simple mathematical expressions. Subsequently, the exposure from the different pathways is added up and compared to a critical reference exposure.

2.3 Principals and assumptions for the Swedish generic guideline values

Generic guideline values have been developed for a range of inorganic and organic substances of importance at contaminated sites, including polycyclic aromatic hydrocarbons and heavy metals (Naturvårdsverket, 1996b). They are developed for typical Swedish conditions concerning exposure, geology, hydrology and the sensitivity of the site and are suitable for a large number of sites in Sweden. In case the generic values are not applicable, a detailed site-specific analysis may be necessary.

The following basic assumptions are used:

- The concentration in the soil is assumed to be constant with time, i.e. no contaminant removal by transport away from the site or degradation occurs. This assumption is motivated by the limited influence of removal by transport and the very large uncertainties associated with predictions of degradation of organic substances. The assumption is conservative, especially in the case of substances where the lifetime risk is of importance (i.e. genotoxic carcinogenic substances).
- The distribution of the contaminant between soil solids, pore water solution and pore air is assumed to be in equilibrium. The equilibrium concentrations are based on the fugacity model (Mackay and Paterson, 1981).
- The distribution of the contaminant between soil solids and soil solution is assumed to be linear with respect to contaminant concentration, and is governed by a distribution coefficient soil-water (K_d). For metals and other inorganic substances, empirical K_d-values are used. The values have been chosen from the literature based on the general behaviour of the substances in typical Swedish low sorbing soils. K_d-values for metals are often very sensitive to pH. The values were chosen to be conservative within the pH-range 5 to 7 with respect to the exposure pathways for which transport via groundwater or surface water is important. Lower pH may increase mobility of heavy metals, higher pH may increase mobility of arsenic.
- For organic substances, the K_d-value is related to the content of organic carbon in the soil, f_{OC}, which is assumed to be 2% by weight. If available the distribution factor between water and organic carbon, K_{OC}, has been used. If this is not available, the K_{OC}-value is estimated from the partitioning coefficient between water and octanol, K_{OW}. The value of K_{OC} is then given by the Karickhoff-equation (Karickhoff, 1981). For ionizing organic substances the K_{OC}-value decreases with increasing pH. Values for a pH of 6.8 have been chosen as a reasonably conservative estimate.
- The distribution of the contaminant between the soil solution and the soil atmosphere is estimated using Henry's constant.

Three types of land-use are distinguished. Each of these land-use types are defined by a number of transfer and exposure pathways, through which human beings can be exposed, and by the characteristics of these pathways:
- Land with sensitive land-use (KM): all types of land-use should be possible, e.g. residential areas, kindergarten, agriculture, groundwater extraction, etc. The exposed persons may be children and adults permanently residing in the area. The exposed persons are assumed to have normal habits as regards consumption and activities (although not necessarily average). The on-site ecosystem, the ecosystem of recipient water bodies or downstream discharge zones, should be capable of supporting the *full range* of ecological functions.
- Land with less sensitive land-use and groundwater extraction (MKM GV), e.g. land used for offices, industry, roads, etc. Groundwater extraction occurs in the vicinity of the site. Adults are assumed to be in the area during working hours. Children are assumed to be in the area temporarily. The on-site ecosystem should be capable of supporting a *limited range* of ecological functions (e.g. growth of ornamental plant species, support transient animal species). The ecosystem in recipient water bodies or downstream discharge zones, should be capable of supporting the *full range* of ecological functions.
- Land with less sensitive land-use as above but with no groundwater extraction (MKM). Characteristics as mentioned above but without groundwater extraction in the area affected by the site.

For human health effects the following exposure pathways are considered:
- direct ingestion of contaminated soil;
- dermal contact with contaminated soil and dust;
- inhalation of dust from the contaminated site;
- inhalation of vapours;
- intake of contaminated drinking water for land-use with groundwater extraction;
- consumption of vegetables grown on the contaminated site (land with sensitive land-use);
- fish consumption from nearby surface water (land with sensitive land-use).

Exposure due to intake of domestic animal products (e.g. meat, milk, eggs) is excluded.

The pathways differ according to land-use type. They are presented in Table 1.

Table 1: Exposure pathways for three types of land-use.

Exposure pathway	KM	MKM GV	MKM
Direct intake of soil	X	X	X
Dermal contact	X	X	X
Inhalation of dust	X	X	X
Inhalation of vapors	X	X	X
Intake of groundwater	X	X	
Consumption of vegetables	X		
Consumption of fish	X		

Health risks are assessed by comparing calculated exposure to a given contaminant with critical exposure. For non-carcinogenic compounds, a reference dose for a particular adverse effect is employed as critical exposure. For most contaminants, this threshold level is expressed as a tolerable daily intake (*TDI*) for the oral exposure pathways. For the inhalation pathway, a reference air concentration (*RfC*) is used. For genotoxic carcinogenic compounds, it is not possible to determine a reference dose as even low doses increase the cancer risk. Increased doses do not affect the severity of the effect, but do increase the probability of the effect to occur. Therefore, mathematical extrapolation models which are linear in the low dose region are used to determine the exposure to a chemical which is equivalent to an acceptable risk level. S-EPA employs a lifetime excess cancer risk of 1 in 100,000.

Toxicological reference data for the oral and inhalation pathway are obtained from WHO, US-EPA/IRIS, CSOIL, IMM (1990, 1991), Nord (1988), SLV (1995) and UBA (1993). For dermal contact, relative absorption factors were taken from MDEP (1994). For the drinking water pathway, drinking water concentration limits from the Swedish Food Administration (SLV, 1993) or WHO were used. In Table 2 a summary of the preferable references of toxicological data for chronic effects, as applied by S-EPA are listed. For the fish consumption pathway, protection was assumed to be sufficient if the surface water concentration was below the residue value of US-EPA's Ambient Water Quality 1980-1993. In addition to chronic effects acute effects also have been considered for arsenic and cyanide (these compounds are not considered in this report).

Table 2: Preferable references of toxicological data (for chronic effects) by S-EPA.

Contaminant	Hypothesis of treshold for effect	Intake of contaminant			
		Oral exposure (daily intake [mg/kg/d])	Inhalation (concentration in air [mg/m³])	Dermal exposure (relative absorption factors)	Drinking water
Non-carcinogenic effects	Yes	WHO, 1993 US-EPA(IRIS), 1995 CSOIL (Van den Berg, 1995) IMM, 1990 Nord, 1988 SLV, 1995 (PCBs)	IMM,1991 WHO, 1987 UBA, 1993	MDEP, 1994	SLV, 1993 WHO, 1993
Non-genotoxic carcinogenic effects	Yes	WHO, 1993	IMM,1991 WHO, 1987 UBA, 1993	MDEP, 1994	SLV, 1993 WHO, 1993
Genotoxic carcinogenic effects	No	WHO,1993	WHO, 1987 US-EPA(IRIS), 1995	MDEP, 1994	SLV, 1993 WHO, 1993

The health risk based soil guideline values are estimated by performing a backward exposure calculation. For non-carcinogenic compounds, the average daily exposure to the contaminated contact media is estimated per kg of body weight (bw), e.g. the ingestion of contaminated soil per body weight and day. The average daily exposure is then used to derive the soil contaminant concentration resulting in an exposure which corresponds to the toxicological reference value (see Figure 1). This concentration is referred to as the reference soil concentration. Factors for the distribution, transport and dilution of the contaminant and unit conversion factors are used in the calculations. For most exposure pathways the chronic exposure is based on the estimated exposure of a child with a body weight of 15 kg.
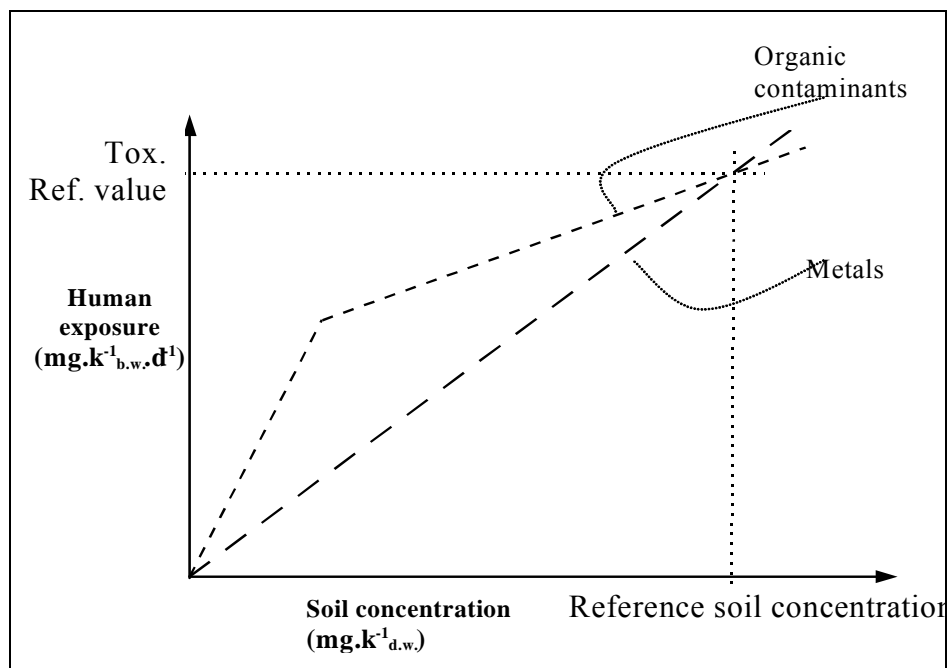
Figure 1: Derivation of the reference soil concentration as a function of exposure and toxicological reference value according to S-EPA.

A separate calculation is performed for integrated lifetime exposure, which is applicable for genotoxic carcinogenic substances. The integrated lifetime exposure is based on the time-weighted average of the exposure of a child (0-6 years) and the exposure of an adult (7-64 years). The body weight of the child was assumed to be 15 kg and the body weight of the adult 70 kg. This corresponds to the assumptions used for the derivation of the Dutch Intervention Values, with the important exception that for the Intervention Values the integrated lifetime exposure was used for all substances, genotoxic or not (Swartjes, 1999). The approach used for the Swedish model will result in a more conservative estimate for non-genotoxic substances (Naturvårdsverket, 1996b).

Ecotoxicological effects both on the contaminated site and due to transport of contaminants from the site have been taken into account. The basic principle for setting the generic guideline values is to select the lowest of the human health based value and the ecotoxicologically based value. For substances where smell and odour problems can occur at lower concentrations this has been taken into consideration. However, a less conservative perspective is put on smell and odour problems compared to toxicological problems. Background concentration is taken into account in that no guideline value should be below the 90th percentile of the measured background concentration in rural environment. Information on the background levels of metals in urban and rural environments have been obtained from Andersson (1977) and Naturvårdsverket (1996c,d).

3   DEVELOPMENT OF THE S-RISK MODEL

3.1   Introduction

The model developed within this project is called S-RISK. It is a model to calculate site-specific guideline values based on soil properties and climatological/geological characteristics for JM redevelopment locations. In its default version (parameter values) the model is limited to use in the region of South-Sweden as specified in Figure 2.

Figure 2: Delineation of the geographical area for which S-RISK default values are applicable.

S-RISK is based on the current Swedish methodology to derive generic guideline values for contaminated soil. It is basically an actualisation of the current methodology on four aspects: (i) model equations for transfer and exposure calculations, (ii) default parameter values for use in the equations, (iii) contaminant-specific parameter values for use in the equations, and (iv) risk calculations.

In the present chapter, S-RISK equations and default parameter values for use in these equations are discussed. If the current model equations are altered, both non-adjusted and adjusted formulae are debated concisely. Reference is made to the appropriate appendices for detailed information.

3.2 Fate and transfer

3.2.1 Mathematical description of contaminant distribution in the soil

- S-EPA

The starting point is the total concentration in the soil, C_s [mg/kg dw]. From C_s, the concentration in pore water, C_w [mg/l], is derived as:

$$C_w = C_s \left[K_d + \frac{(\theta_w + \theta_a H)}{\rho_b} \right]^{-1}$$

where

K_d: soil-water distribution coefficient [l/kg];
θ_w: soil water content [dm³ water/dm³ soil];
θ_a: soil air content [dm³ air/dm³ soil];
H: Henry's law constant [-];
ρ_b: dry soil bulk density [kg/dm³].

The vapour concentration in pore air, C_a [mg/dm³] is given by:

$$C_a = H \times C_w$$

S-EPA reports that f_{OC}, the fraction of organic carbon in the soil, is assumed to be 2% by weight. The relation between f_{OC} and f_{OM}, the fraction organic matter, is given by:

$$f_{OC} = f_{OM} \times 0.58$$

The values adopted for the basic soil parameters are given in Table 3.

Table 3: Basic soil parameters used by S-EPA (Naturvårdsverket, 1996b, 2005).

Parameter	Value	Unit
f_{OM} (organic matter content)	0.02	-
ρ_b (soil bulk density	1.5	kg/dm³
θ_w (soil water content)	0.3	dm³/dm³
θ_a (soil air content)	0.2	dm³/dm³

- S-RISK

The equations for calculating C_w and C_a are not altered in S-RISK. However, the parameter values for f_{OC}, ρ_b, θ_w, θ_a (and θ_t: soil total porosity) are revised to apply to typical filling and construction material used by JM on its sites (see next section). The parameter values used in S-RISK are summarized in Table 4.

Parameter	Value	Unit
f_{OM} [1]	0.02	-
ρ_b [2]	1.69 (medium till fine sand)	kg/dm³
	1.56 (silty sand)	
	1.42 (clay loam)	
θ_w [2]	0.058 (medium till fine sand)	dm³/dm³
	0.111 (silty sand)	
	0.181 (clay loam)	
θ_a [2]	0.3 (medium till fine sand)	dm³/dm³
	0.276 (silty sand)	
	0.263 (clay loam)	
n [2]	0.358 (medium till fine sand)	dm³/dm³
	0.387 (silty sand)	
	0.444 (clay loam)	

Table 4: Basic soil parameter values used in S-RISK.

[1]: arithmetic mean of 0.0029 and 0.0116: 0.0075 (Table 5) multiplied by 1.72.
[2]: arithmetic mean of extreme values (Table 5).

3.2.2 Transport of vapour from soil to indoor air

- S-EPA

Transfer of vapour from soil to indoor air can be a critical pathway for volatile and semi-volatile compounds. It has been assumed that there is an equilibrium between the soil air and the indoor air concentration which can be described by a dilution factor. The concentration in indoor air C_{ia} [mg/dm³] is given by:

$$C_{ia} = C_a \times DF_{ia}$$

where

C_{ia}: concentration in indoor air [mg/dm³];
C_a: concentration in pore air [mg/dm³];
DF_{ia}: dilution factor indoor air to soil air [-].

The two following methods for calculating the dilution factor exist:

- Method 1 – theoretical model

The first method, used in CSOIL and HESP, is based on theoretical models for the release of vapour from the soil and the dilution that occurs in indoor air. This method has the advantage of being able to take into account parameters such as contaminated soil depth, porosity and water content and substance dependent diffusivities. However, it is difficult to obtain values for a number of important parameters.

- Method 2 – empirical relationship

The second method is based on the use of empirical relationships between soil air and indoor air concentrations. It is also difficult to find reliable empirical data for use with this method. Most of the available data have been derived for radon, for which the relatively short half-life will limit the ability to penetrate into buildings. Furthermore the attenuation coefficient can be very low for radon that develops close to the building and does not necessary represent soil contaminants that migrate from a distance towards the building.

- Final choice for the generic guidelines values

The second method, based on the empirical data from MDEP (1994) was used for the derivation of the generic guidelines values. MDEP used a dilution factor of 1/20,000 between soil and indoor air, and this factor was thought to be most appropriate. For comparison, the dilution factor in CSOIL and HESP for an open floor basement (crawl space) is about 1/5,000 and in HESP for a concrete floor basement about 1/70,000.

- S-RISK

The relationship between soil contamination and indoor air is influenced by a large number of processes and parameters, which are subject to variability. VOC (volatile organic contaminants) can appear in the soil in pure form, dissolved in the pore water, adsorbed to particles or as vapour in the soil air. The distribution of VOC in the soil depends on the concentration of the VOC in the soil, soil type, porosity, water content and organic carbon fraction, and are controlled by the physical-chemical properties of the VOC. VOC in the soil gas phase can migrate by molecular diffusion or convection. From the top boundary of the contaminant source, molecular diffusion moves the volatilised contaminant towards the upper layers until it reaches the zones under influence of the building. Here convective air movement within the soil column transports the vapours through cracks which are situated between the foundation and the basement slab floor. This convective sweep effect is induced by a negative pressure within the structure caused by a combination of wind effects and stack effects due to building heating and mechanical ventilation. Once VOC have entered the building, their indoor concentration will be determined by building and ventilation characteristics.

Selection of the model
The DF_{ia} values, which were used in the S-RISK model, were calculated by using the Johnson & Ettinger model (J&E) (Johnson and Ettinger, 2004) or a combination of the Volasoil (Waitz et al., 1996) and the J&M model.

The J&E model is a one-dimensional analytical solution of diffusive and convective transport of vapours into indoor spaces. The model calculates an attenuation factor that relates the vapour concentration in the indoor space to the vapour concentration at the source. It was developed for use as a screening level model and consequently is based on a number of simplifying assumptions regarding contaminant distribution and occurrence, subsurface characteristics, transport mechanisms, and building construction. The procedures used to estimate the soil permeability of the soil stratum in contact with the building floor and walls assumes isotopic soils and constant soil moisture content. In addition, the calculations do not account for preferential pathways due to soil fractures or rocks,

vegetation root pathways, or the effects of a gravel layer below the floor slab or backfill. These items may act to increase the vapour permeability of the in situ soils. The house is seen as a homogeneous compartment with complete mixing.

The Volasoil model again considers both diffuse and advective transport from soil, but uses as building concept a house with a crawl space. The crawl space is in direct contact with the soil without a floor, a floor is present between crawl space and the indoor environment. The indoor compartment is also assumed to be completely mixed.

Selection of the building concept and conceptual site model
For the calculation of the dilution factor two building types were selected, a concrete floor directly on the soil (slab-on-grade) and a concrete basement. Both types of building are most frequently built by JM and therefore separate dilution factors were calculated.
The J&E model was used on its own for the slab-on-grade situation. However, according to the information provided by JM, houses with basements have protective measures with regard to radon intrusion and as such also prevent or reduce vapour intrusion for other contaminants. The model should therefore consider two barriers: the first barrier between soil and basement and the second barrier between basement and indoor environment. As none of the published models allows for this combination, a two-step procedure was followed. In a first step, the J&E model was used with the assumption of that the building consisted solely of a basement, resulting in a first attenuation factor. Secondly the Volasoil model was used to calculate an attenuation factor between crawl space and indoor air for varying floor quality (Volasoil results: for a bad ($DF_{BI} = 1$), normal ($DF_{BI} = 24$) and good quality ($DF_{BI} = 2263$) floor). Combination of both attenuation factors resulted in the overall attenuation or dilution factor.. Furthermore, a study from Fast et al. (1987) showed that the average measured DF between a crawlspace and the indoor air equalled 20 for a wooden floor and 10 for a concrete floor.

Figure 3 describes the conceptual model for the two building types.
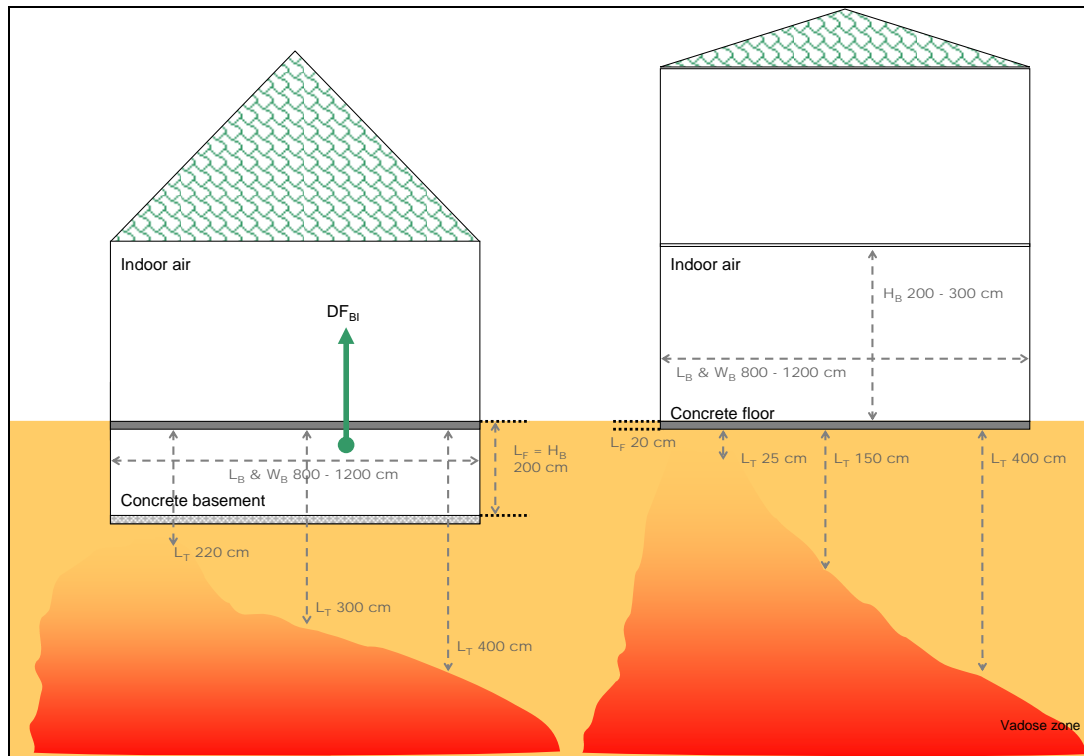
Figure 3: Conceptual site model for a house or apartment with a concrete floor (slab-on-grade) and a house with a concrete basement

Figure 3 shows some of the model parameters given in Table 5. Enclosed space floor length (L_B) and width (W_B) are shown on the same line, because the picture is two-dimensional. The enclosed space height (H_B) is chosen in such a way that the total height (volume) of the building is taken into account. These parameter values will differ from house to house and a reasonable variation is chosen from building plans received from JM. The house with a basement is related to a house with a garage under the house. Depth below grade to bottom of enclosed space floor (L_F) is therefore a fixed value. The L_F for a house with a concrete floor is related to the bottom apartment in an apartment building. The calculated DF_{ia} for a 'house – apartment' with a concrete floor can also be applied to single houses with a concrete floor. It is assumed that indoor air does not migrate extensively to above situated apartments or indoor spaces. The depth below grade to the bottom of the enclosed space floor(L_F) for a house with a concrete floor equals the enclosed space floor thickness.

The pollution can be situated very close to the bottom of the enclosed space floor or also further away, this variation is taken into account when deriving the new DF_{ia}.

Selection of soil types
During the building process JM is using backfilling material on the construction site. The filling material below and under the houses is rather heterogeneous in texture. Hence, calculations were done for three soil types: medium till fine sand (S), silty sand (SS) and clayey loam (CL). The selection of the soil types is based on analysis of 6 samples, taken from typical filling and construction material that JM uses on its sites. The soil texture from 6 samples was determined according to the USDA classification system. Most soil samples were classified as loamy sand (road filling Ludvig, fine crushed rock Stockholm, road construction material Ludvig). The sample representing road filling Stockholm was classified as sandy clay loam and the sample from fine crushed rock Stockholm as loamy

sand. The sample from dry clay was classified as clay. It should be noted that 3 out of 6 samples existed for 55% to 71% of particles > 2 mm. They contained course sand and small pieces of crushed rock. The presence of pieces of crushed rock can enhance the gas phase transport towards buildings by preferential pathways. Therefore, a lower DF_{ia} should be chosen then one would select based on the soil type (0 – 2 mm). Road filling Ludvig is most commonly used on sites. Even though the most common soil type seems to be loamy sand there is a possibility that the soil contains more sand or clay. To include this variability in the calculated DF_{ia} the 3 above mentioned soil types were selected.

Probabilistic approach

Soil characteristics, building characteristics and parameters for the transfer process are subject to uncertainty and variability. To account for this uncertainty and variability, probability density functions replace the single value parameter estimation where possible or relevant. The dilution factors were calculated by adding a one-dimensional uncertainty analysis with the Crystal Ball® (2000) software to the J&E model. This uncertainty analysis provides a frequency distribution on the output, i.e. the dilution factor. For further calculation of the soil guideline values, the lower 5th percentile of the dilution factor values is taken. Table 5 gives the parameter values and distributions for the vadose zone.

Selection of contaminant of concern

The new dilution factors are based on calculations for the most volatile PAH of the list, being naphthalene. The contamination is considered to be present in the vadose zone (unsaturated zone). The calculated DF_{ia} is applicable for all volatile PAHs.

Table 5: Parameter values and distributions for the calculation of the dilution factor DF_{ia} for the vadose zone.

Soil and structural properties of the building	Symbol	Unit	Distributions	Remark	Reference
Initial soil concentration	CR	µg/kg	U ($10^{-2};10^5$)	Practical range	Not applicable
Average soil temperature	T_s	°C	(10)	Between 5 and 100 cm below groundlevel	Geocentrum Uppsala
Depth below grade to bottom of enclosed space floor	L_F	cm	CF (20) CB (200)	Buildings Källarvåning, Torpargrund & Platta på mark	JM information: building plans
Depth below grade to top of contamination	L_T	cm	CF T (25;150;400) CB T (220;300;400)	Based on depth of a contaminant	JM information: site information
Thickness of soil stratum	h_A	cm	$h_A = L_T$	Resulting from L_T	
Soil vapour permeability	K_v	cm²	S U ($10^{-8};10^{-6}$) SS U ($10^{-9};10^{-8}$) CL U ($10^{-10};10^{-9}$)	Practical range	Johnson and Ettinger, 2004; JM information
Soil dry bulk density	ρ_b	g/cm^3	S U (1.63:1.75) SS U (1.49:1.63) CL U (1.35:1.49)	Practical range	Johnson and Ettinger, 2004; JM information
Soil total porosity	n	-	S U (0.340:0.375) SS U (0.375:0.399) CL U (0.399:0.489)	Practical range	Johnson and Ettinger, 2004
Soil water-filled porosity	θ_w	cm^3/cm^3	S U (0.04:0.076) SS U (0.076:0.146) CL U (0.146:0.216)	Practical range	Johnson and Ettinger, 2004; JM information
Soil organic carbon fraction	f_{oc}	-	U (0.0029;0.0116)	Practical range	JM information
Enclosed space floor thickness	L_{crack}	cm	T (10;12;20)	Practical range	JM information: building plans
Soil-building pressure differential	DP	g/cm-s	T (0;2;20)	Literature	Johnson and Ettinger, 2004
Enclosed space floor length	L_B	cm	U (800;1200)	Practical range	JM information: building plans
Enclosed space floor width	W_B	cm	U (800;1200)	Practical range	JM information: building plans

Soil and structural properties of the building	Symbol	Unit	Distributions	Remark	Reference
Enclosed space height	H_B	cm	CF U (200;300) CB (200)	Practical range	JM information: building plans
Dilution factor basement-indoor air	DF_{BI}	-	CF (N.A.) CB T (1;24;2263)	Practical range	van Wijnen H.J., Lijzen J.P.A., 2006 Fast T., Kliest J., van de Wiel H., 1987
Floor-wall seam crack width	w	cm	T (0.05;0.1;1.0)	Practical range	Johnson and Ettinger, 2004; JM information
Indoor air exchange rate	ER	1/h	T (0,18;1.0;2.0)	Practical range	Johnson and Ettinger, 2004; JM information

N: normal; LN: log-normal; U: uniform; T: triangular; C: custom.
CF: concrete floor (slab-on-grade); CB: concrete basement.
S: medium till fine sand; SS: silty sand; CL: clay loam.

Table 6 and Table 7 give the percentile values for the two selected types of houses and for different soil types. These tables also contain the statistical description of the $1/DF_{ia}$ frequency distribution for both houses (concrete floor and basement). These houses were assumed to be built on soil consisting out of medium/fine sand (S), silty sand (SS) or clayey loam (CL) respectively. The values are expressed as $1/DF_{ia}$ for easer interpretation.

Table 6: Percentile values and statistical description of the inverse value of the dilution factor ($1/DF_{ia}$) for a house with a concrete floor (CF) built on medium/fine sand (S), silty sand (SS) or clayey loam (CL).

Percentiles + Statistics	Concrete floor		
	medium/fine sand (S)	silty sand (SS)	clayey loam (CL)
5,0%	724	16,917	51,579
50,0% (median)	2,432	55,254	171,860
95,0%	11,741	204,352	605,651
97,5%	19,079	267,419	749,108
Trials	10,000	10,000	10,000
Range Minimum	165	3,964	11,844
Mean	3,995	76,004	230,021
Range Maximum	136,562	721,642	2,179,284
Standard Deviation	6,382	68,932	190,616

Table 7: Percentile values and statistical description of the inverse value of the dilution factor ($1/DF_{ia}$) for a house with a concrete basement (CB) built on medium/fine sand (S), silty sand (SS) or clayey loam (CL).

Percentiles + Statistics	Concrete basement		
	medium/fine sand (S)	silty sand (SS)	clayey loam (CL)
5,0%	83,090	3,114,220	8,221,083
50,0% (median)	1,018,192	34,909,635	92,963,909
95,0%	10,226,060	190,186,401	517,606,314
97,5%	16,928,023	254,103,635	701,783,526
Trials	10,000	10,000	10,000
Range Minimum	3,969	82,440	199,672
Mean	2,714,714	57,504,805	156,175,080
Range Maximum	295,547,195	1,244,071,534	2,231,289,672
Standard Deviation	7,488,079	71,174,061	191,972,009

Table 8 and Table 9 give the sensitivity ranking of the various parameters in the calculation of the dilution factor. Each table starts with the parameter that has the highest sensitivity.

The parameter with the highest sensitivity ranking can be considered to be most important in the model calculations of DF_{ia}. The parameter with the lowest sensitivity ranking is the least important one in the model.

Table 8: Sensitivity Data – contribution to the variation in DF_{ia} for a house with a concrete floor (CF) built on medium/fine sand (S), silty sand (SS) or clayey loam (CL).

Statistics	Contribution to the variation in the dilution factor (DF_{ia})		
	Medium / fine Sand (S)	Silty Sand (SS)	Clayey Loam (CL)
Soil vapour permeability	27.3%	19.4%	1.8%
Pressure difference	21.7%	31.5%	2.3%
Indoor air exchange rate	30.4%	27.9%	28.2%
Depth grade - top contaminant	15.5%	-	-
Soil water-filled porosity	0.9%	1.3%	3.3%
Crack width between wall and floor	-	15.6%	56.5%
Soil total porosity	-	-	1.0%
Floor thickness	-	-	2.5%
Height enclosed space	3%	2.1%	2.9%
Width enclosed space floor	-	0.9%	
Total	98.8%	98.7%	98.6%

Table 9: Sensitivity Data – contribution to the variation in the dilution factor (DF_{ia}) for a house with a concrete basement (CB) built on medium/fine sand (S), silty sand (SS) or clayey loam (CL).

Statistics	Contribution to the variation in the dilution factor (DF_{ia})		
	Medium / fine Sand (S)	Silty Sand (SS)	Clayey Loam (CL)
Soil vapour permeability	20.1%	4.6%	-
Pressure difference	16.9%	8,7%	-
DF_{BI} (basement air - indoor air)	52.9%	65.3%	62.3%
Basement air exchange rate	8.4%	10,7%	11.1%
Depth grade - top contaminant	0,9%	-	-
Soil water-filled porosity	-	0.7%	1.4%
Crack width between wall and floor	-	8,7%	22.2%
Floor thickness	-	-	1.0%
Total	99.2%	98.7%	98.0%

Table 6 and Table 7 show the statistical description for the different types of houses build on different soils (filling material). Both tables are showing that the $1/DF_{ia}$ for sand is the lowest, followed by silty sand and clayey loam soils. An increase in $1/DF_{ia}$ indicates that less soil air is transported from the source into the indoor air.

The inverse values of the dilution factor ($1/DF_{ia}$) for a house with a concrete floor are lower then these for a house with a concrete basement. So it is calculated that there is a higher dilution from soil air to basement air and from basement air to indoor air when a concrete basement is present. The most likely reason is related to the two compartment approach that was used for the concrete basement. The soil air has to enter the building through the

foundation (concrete basement floor), followed by transport through the structure (indoor space floor) of the house. Hence, this is reflected in a higher dilution.

The three model parameters that most strongly influence the variation in DF_{ia} (Table 8) for a house with a concrete floor built on sand or silty sand, are soil permeability (19-27%), pressure difference (21-31%) and indoor air exchange rate (27-30%). For a house built on clayey loam the two most dominant parameters are the indoor air exchange rate (28%) and the crack width (56%).

The three most dominant model parameters for a house with a concrete basement built on sand were BF_{BI} (53%), soil vapour permeability (20%) and pressure difference between soil and basement (17%) (Table 9). For a house built on silty sand or clayey loam the two most dominant parameters were BF_{BI} (62-65%) and basement (indoor) air exchange rate (10-11%).

The difference in most dominant parameters between soil types could be related to the decreasing influence of convective air flow, and the increasing influence of diffusive air flow, in the range from sand to clayey loam. For a soil that consists of clayey loam certain parameters will be of less influence than for high permeable soils like sand and loamy sand. This is reflected in the higher $1/DF_{ia}$ for clayey loam and lower $1/DF_{ia}$ for sandy loamy soils.

The soil vapour permeability and crack width were, according to the new manual of the J&E software program, scientifically supported by literature. The pressure difference and indoor air exchange rate were based on JM data and literature. The literature values however are supported with sufficient measured values from various soils and regions.

The proposed dilution factors, according to soil type and building type, are given in Table 10. For comparison, not only the 95% values, but also the median values are given.

Table 10: Proposed dilution factors (DF$_{ia}$) for S-RISK.

	medium to fine sand	silty sand	clayey loam
Concrete floor (slab-on-grade)			
5 %	1/700	1/16,000	1/52,000
50 % (median)	1/2,432	1/55,000	1/172,000
Concrete basement			
5 %	1/83,000	1/3,000,000	1/8,000,000
50 % (median)	1/1,000,000	1/35,000,000	1/93,000,000

The proposed dilution factors only apply for buildings with concrete floors or a good barrier between basement and indoor air. In case of other construction types, dilution factors should be calculated accordingly.

If pieces of crushed rock are present in the soil a lower $1/DF_{ia}$ should be chosen, the values for medium to fine sand can be selected as an approximation.

3.2.3   Transport of contaminants to a groundwater well

- S-EPA

The leaching of contaminants from soils and transport to groundwater depends on site-specific conditions, determined by a number of factors that may vary over a wide range. The model for the generic Swedish guideline values is based on a simplified model similar to that used by HESP (SHELL, 1994, 1995) and for the derivation of US-EPA Soil Screening Level (SSL) values (US-EPA, 1996a). The Swedish model estimates the dilution between pore water concentration and the concentration in a well situated downstream from the contaminated site, either at the site boundary or some distance away from the site. The model assumes that the contaminants are leached by water infiltrating through the soil of the contaminated site and are transported down to the groundwater. The initial concentration in the leachate is assumed to be equal to the equilibrium pore water concentration. As the leachate reaches the groundwater, it will be diluted by groundwater from upstream of the site, and if the well is placed far away from the site also by water infiltrating between the site and the well. The model contains several conservative assumptions:

- no sorption or degradation is considered during transport from the upper soil layers to the groundwater surface and to the well;
- dilution by lateral dispersion in the aquifer is neglected, since the source may have a wide extent perpendicular to the flow direction.

The concentration in groundwater C_{gw} [mg/l], is given by:

$$C_{gw} = C_w \times DF_{gw}$$

where

DF_{gw}:   soil pore water to groundwater dilution factor [-].

The dilution factor DF_{gw} is given by:

$$DF_{gw} = \frac{L \times I}{(k \times i \times d_{mix}) + (L + X) \times I}$$

where:

L: the length of the contaminated area in the direction of the groundwater flow [m];
X: the distance from the contaminated area to the well [m];
I: the infiltration rate [m/yr];
k: the hydraulic conductivity of the soil [m/yr];
i: the hydraulic gradient [m/m];
d_{mix}:   the thickness of the mixing zone in the aquifer [m].

The thickness of the mixing zone d_{mix} is given by:

$$d_{mix} = \sqrt{0.0112(L + X)^2} + d_a \left[1 - \exp\left(\frac{-L \times I}{k \times i \times d_a} \right) \right]$$

where d_a is the aquifer thickness [m]. The thickness of the mixing zone cannot be greater than the aquifer thickness.

In the case of land with sensitive land-use (KM), the well is assumed to be located at the site boundary. A dilution factor 1/15 was chosen in the S-EPA approach as a reasonably conservative value for the calculation of the generic guideline values.

In the case of land with less sensitive land-use and groundwater extraction (MKM GV), the well is assumed to be located 500 m from the site. A dilution factor of 1/30 was chosen as a reasonably conservative value for the calculation of the generic guideline values.

The S-EPA approach is slightly different from the HESP or US-EPA SSL-model with regard to the position of the receptor: while the S-EPA assumes the receptor is a well either at the site boundary or some distance away from the site depending on type of land-use, the HESP and US-EPA always assume the receptor is located in the groundwater underlying the contaminated site (i.e. X=0; the above equations are then reduced to the ones used in HESP, US-EPA and Tier 2 of the UK Environment Agency Remedial Targets approach (Marsland and Carey, 1999)). The distinction in the S-EPA approach is based on a difference in human exposure. The consequence of the approach is that in the case of land with less sensitive land-use and groundwater extraction (MKM GM) the receptor is located at a distance from the source zone and a groundwater body with a length of 500 m could be polluted at levels

above groundwater quality objectives. Disregarding groundwater beneath the site as a receptor is not in line with the view of the Groundwater Directive (COM(2003)550), which considers groundwater to be an important natural resource and focuses on preventing groundwater pollution. Although it can be acceptable for the assessment of remedial objectives for historically contaminated soils and groundwater, it could be subject to discussion in the development of generic guidelines.

However, the basics of the methodology stated above are not under discussion. The dilution factors calculated in Table 2.2 of the S-EPA report could not be reproduced using the above equations. Deviations were especially large in the cases where the distance from the contaminated area to the well (X) is different from zero. A closer inspection of the numbers given in Table 2.2 revealed that the calculated dilution factor could be obtained using the mixing depth given but the mixing depth could not be calculated from the given numbers using the above equation. In the S-EPA site specific approach, values for the dilution factor are adjusted to 1/13 (KM) and 1/55 (MKM GV) (Naturvårdsverket, 2005). The new value for land with sensitive land-use is probably a correction based on recalculations. The new value for land with less sensitive land-use is obtained by taking into account lateral dilution of the groundwater plume during migration.

- S-RISK

Because of the large natural variation in physical properties of Swedish aquifers and the scarcity of available data, a similar probabilistic approach as for the dilution factor in air is adopted. First, a conceptual model of a 'typical' building site near the waterfront is developed and assumptions on the distribution of the most important variables (I, k, i, d_a) are made. Next, the range of values for the dilution factor DF_{gw} is calculated and the 5th percentile is selected as a worst-case value for the dilution factor under the assumptions made in the site description.

The conceptual model for leaching to groundwater is given in Figure 4.
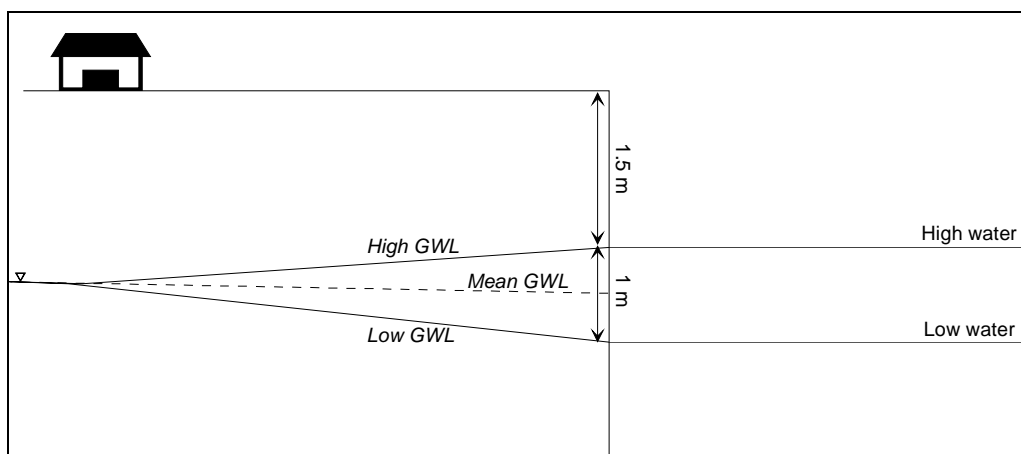
Figure 4: Conceptual site model for a building site near the waterfront.

For a 'typical' building site near the waterfront, daily fluctuations in water level amount to roughly 1 metre, causing high and dynamic gradients in groundwater level. For the calculations, the largest gradient towards surface water is considered and is assumed to be

equal to the regional hydraulic gradient (directed towards surface water) with an extra 0.005 m m^{-1} added (0.5 m (difference between mean groundwater level and low groundwater level) over 100 metres (assumed range of influence of the surface water dynamics)). Mean groundwater level at the site is at 2 metres beneath the soil surface.

The soil at the building site consists of 2 layers: a layer of filling material on top of the original aquifer material. Two cases can be distinguished:
  - The groundwater fluctuation takes place only in the (less permeable) aquifer material. Thickness of the water conducting layer is then assumed to be between 2 and 10 metres.
  - The groundwater level is inside the layer of the filling material. In this case, groundwater movement will take place mainly in the more permeable filling material. Thickness of the water conducting layer is assumed to be between 0.5 m (total thickness of filling material is 3 m) and 4.5 m (total thickness of filling material is 7 m).

The dilution factor is calculated for land with sensitive land-use (KM) and land with less sensitive land-use (MKM GV) and the distances between the site and the receiving groundwater well of X=0 and X=500 m are taken from the original S-EPA approach.

The dilution factor is calculated from the equations given in the previous section. The length of the contaminated site in the direction of groundwater flow L is set equal to 50 m. The assumptions for I, k, i and d_a are listed in Table 11.

Table 11: Assumptions on the distribution of the main variables for the calculation of DF_{gw}.

Variable	Distribution	Minimum	Maximum	Likeliest value
I (m yr^{-1})	uniform	0.2	0.5	-
i (m m^{-1})	triangular	0.006	0.055	0.015
Aquifer material				
k (m s^{-1})	triangular	1E-10	1E-06	1E-07
d_a (m)	uniform	0.5	4.5	-
Filling material				
k (m s^{-1})	triangular	1E-04	1E-02	1E-03
d_a (m)	uniform	2.0	10.0	-

The range for the infiltration rate I is based on meteorological data for Stockholm, Malmö and Göteborg. The range for the hydraulic conductivity k is based on Carlsson and Gustavsson (1984) as quoted in S-EPA report 5053 (Naturvårdsverket, 2002). The range for the hydraulic gradient i is based on the values used in the original S-EPA approach with an additional gradient of 0.005 m m^{-1} due to the fluctuations in surface water level.

The inverse value of the dilution factor (1/DF_{gw}) was calculated using the values from Table 11 for 4 scenarios:
  - Land with sensitive land-use with groundwater level in the filling material (KM fi)
  - Land with sensitive land-use with groundwater level in the aquifer material (KM aq)
  - Land with less sensitive land-use with groundwater level in the filling material (MKM GV fi)
  - Land with less sensitive land-use with groundwater level in the aquifer material (MKM GV aq).

The resulting frequency distribution for $1/DF_{gw}$ for the first scenario is given in Figure 5.
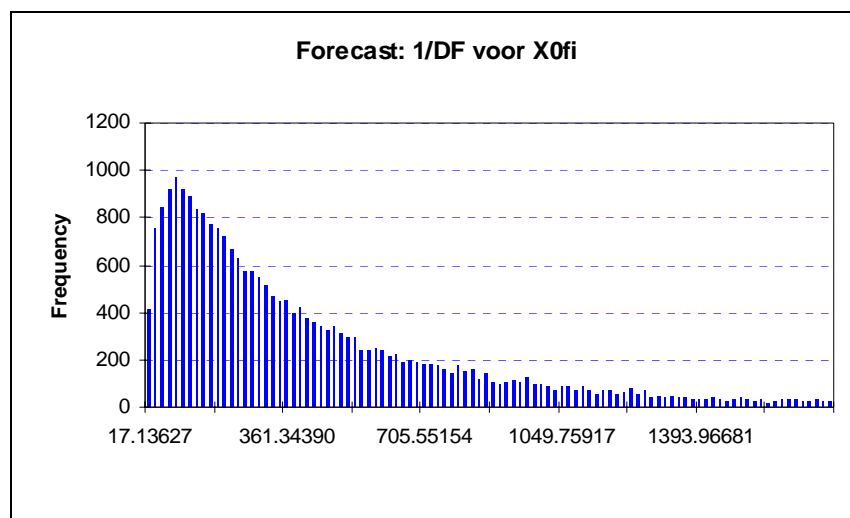
Figure 5: Frequency distribution of 1/DF_gw calculated for land with sensitive land-use with groundwater level in the filling material (X=0).

The highest contribution to the variance in calculated $1/DF_{gw}$s can be attributed to the hydraulic conductivity (see Table 12). This variable has a very high natural variability and can vary over several orders of magnitude depending on location. Other significant contributions (18 to 28%) come from the thickness of the water-conducting layer and the hydraulic gradient. Variations in infiltration over the considered range only have a minor effect on the calculated $1/DF_{gw}$.

Table 12: Sensitivity analysis for calculation of DF_gw measured by contribution to variance (%).

Variable	KM		MKM GV	
	fi	aq	fi	aq
k	48.1	55.3	48.3	55.1
d_a	27.3	18.8	27.5	18.8
i	18.7	19.1	18.5	19.8
I	5.9	6.8	5.6	6.4
Sum	100.0	100.0	99.9	100.1

Different percentiles from the calculated frequency distributions for $1/DF_{gw}$ under the 4 scenarios are given in Table 13. The 5th percentile was selected as a realistic worst-case value. These values are given in Table 14. For the scenarios with groundwater level in filling material, $1/DF_{gw}$ is higher than in the original S-EPA approach. For the scenarios with groundwater level in the aquifer material, calculated $1/DF_{gw}$s are lower than in the original approach. For the case where X=0 and groundwater level is in the aquifer material, there is virtually no dilution in groundwater.

Table 13: Percentile values of the inverse value of the dilution factor (1/DF_gw) for land with sensitive and less sensitive land-use and for groundwater level (GWL) in filling material or in aquifer material

Percentile	KM		MKM GV	
	fi	aq	fi	aq
0.0 %	3.42	1.00	13.68	11.00
2.5 %	30.49	1.01	39.95	11.01

5.0 %	**44.00**	**1.01**	**53.80**	**11.01**
50.0 %	296.15	1.07	308.06	11.07
95.0 %	1394.77	1.33	1384.48	11.32
97.5 %	1767.26	1.40	1760.05	11.40
100.0 %	4722.54	2.13	5002.55	12.11

Table 14: Calculated 1/DF$_{gw}$ (5th percentile) for sensitive and less sensitive land-use and for groundwater level (GWL) in filling material or in aquifer material.

	KM	MKM GV
GWL in filling material	44.0	53.8
GWL in aquifer material	1.0	11.0
original S-EPA approach	15.0	30.0

Calculated inverse values of the dilution factor (1/DF_{gw}s) are higher than the ones in the original S-EPA approach when it is assumed that groundwater level is in the filling material and lower than the ones in the original approach when it is assumed that groundwater fluctuations are confined to the less permeable aquifer material. However, the values are relatively close to the ones from the original approach considering the uncertainty introduced by the assumptions made in the conceptual model. Therefore it is recommended to maintain the values for the dilution factor from the original S-EPA approach in S-RISK.

3.2.4 Transport of contaminants to surface water

The transfer equations of contaminants to surface water are not revised.

The model for transport of contaminants to surface waters and the dilution in surface water is based on a simplification of a model used in HESP. In the S-EPA model, the effect of surface erosion had been neglected. Leaching of contaminants from the soil is represented as described above in the model for transport of contaminants to groundwater. The groundwater is assumed to discharge into a lake or a river with a certain turnover time or annual flow rate. The dilution factor of 1/15 is used to represent dilution of groundwater at the site boundary by the surface water.

The concentration in the surface water, C_{sw} [mg/l] is given by:

$$C_{sw} = C_{gw} \times DF_{sw}$$

where

$$DF_{sw} = \frac{Q_{di}}{Q_{sw}} = \frac{k \times i \times d_{mix} \times L_w}{Q_{sw}}$$

and

Q_{di}: the discharge of groundwater from the contaminated site to the surface water [m³/yr];
Q_{sw}: the water flow rate in the surface water [m³/yr];

L_w: the width of the contaminated area perpendicular to the direction of the groundwater flow [m].

For lakes the water flow rate is determined as:

$$Q_{sw} = V_{sw} \times k_t$$

where

V_{sw}: the volume of the lake [m³];
k_t: the turnover rate of the lake [yr^{-1}].

Assuming a groundwater discharge of 250 m³/a and a water flow rate of 1,000,000 m³/a in the surface water (0.03 m³/s), a dilution factor of 1/4,000 has been adopted (a total dilution factor of 1/60,000 from pore water to surface water). This factor is thought to be representative of dilution in a small size lake or stream.

3.2.5 Transport of contaminants to plants

A crucial parameter for estimating the metal concentration in vegetables and subsequently human exposure through consumption of vegetables is the bioconcentration factor (*BCF*), i.e. the ratio of the concentration in the edible part of the vegetable to the total soil concentration. The *BCF* can be expressed in different dimensions: [(mg/kg fresh plant)/(mg/l pore water)], [(mg/kg fresh plant)/(mg/kg soil)] or [(mg/kg dry plant)/(mg/kg soil)]. *BCF*s are dependent on soil properties and are highly crop-specific. To determine the risk of vegetable consumption for the south of Sweden the *BCF* must apply to the respective soil conditions. To be able to determine the 'representative concentration' in vegetables the type of vegetable(s) that is/are relevant for the risks due to vegetable consumption from contaminated sites have to be selected. In regard to generic quality standards the policy on soil contamination is related to *the possibility* to grow vegetables without experiencing adverse effect on human health. This political basic requirement implies that the attention should be focused on a hypothetical 'average representative vegetable package' applicable to the south of Sweden.

- S-EPA

A simplification of the method used in the Netherlands (CSOIL and HESP) is used in the Swedish model. The Dutch model estimates transfer of contaminants in two ways: direct uptake from the soil by roots and deposition of dust from the contaminated area on aerial plant parts. The S-EPA considers the bioconcentration factor for direct uptake from soil sufficiently conservative to represent uptake via both pathways.

- *Uptake of metals*

The Swedish *BCF* values for cadmium uptake by aboveground vegetables and root vegetables (Naturvårdsverket, 1996b) are taken from Bockting and Van den Berg (1992). These values were used for the derivation of the Dutch soil quality standards from a wide range of crops, in the early nineteen nineties. The *BCF* values were derived from field data,

laboratory experiments and estimations, from which the geometric mean was used. The dataset included crops that are irrelevant for consumption. Besides, no attention was given to the vegetable selection related to consumption rates, metal soil content or to the respective soil properties.

- *Uptake of organic compounds*

The uptake of organic substances from soils is based on the relationship between K_{OW} and the bioconcentration factor *BCF*, described by Briggs et al. (1982, 1983). This approach, which is adopted in the Dutch CSOIL model and the HESP model, is based on the concentration of the contaminant in the soil pore water, C_w (determined from the soil concentration and the K_d-value). The BCF for the stem [(mg/kg fresh plant)/(mg/l pore water)] is given by:

$$BCF_{stem} = \left(10^{((0.95 \times \log K_{OW})-2.05)} + 0.82\right) \times 0.784 \times 10^{\left(-0.434 \times \frac{(\log K_{OW}-1.78)^2}{2.44}\right)}$$

and the BCF for the root [(mg/kg fresh plant)/(mg/l pore water)] by:

$$BCF_{root} = \left(10^{((0.77 \times \log K_{OW})-1.52)} + 0.82\right)$$

the total plant concentration factor [(mg/kg fresh plant)/(mg/kg dry soil)] is calculated as:

$$K_{pl} = \left(\left(BCF_{stem} \times f_{leaf}\right) + \left(BCF_{root} \times f_{root}\right)\right) \times \frac{\rho_b}{\left(\theta_w + \left(K_d \times \rho_b\right) + \left(H \times \theta_a\right)\right)}$$

where

f_{leaf}: the fractional consumption of leaf and stem vegetables;
f_{root}: the fractional consumption of root vegetables in the consumption.

Vegetable consumption is assumed to comprise 50% leaf and stem vegetables and 50% root vegetables of the total consumption rates. The last part of the equation relates the concentration in the soil pore water to the total soil concentration.

- S-RISK

- *Uptake of metals*

For heavy metals, the *BCF* concept with values based on empirical data, is maintained. Weighted average plant uptake factors are derived from plant specific uptake factors and the average food consumption pattern. In the framework of the revision of the Dutch Intervention Values an improved model for the assessment of metal uptake by homegrown vegetables was developed (Versluijs and Otte, 2001). In this study Freundlich-like plant–

soil relations have been derived for the calculation of the concentration in vegetables as a function of total soil concentration and the major soil properties, for consumption vegetables only. For each vegetable with sufficient and proper data available, the following equation was derived:

$$\log[C_{v,dw}] = a_i + b_i \log[C_s] + c_i\, pH_{soil} + d_i \log[clay\%] + e_i \log[OC\%] + f_i [other\ factors]$$

where

$C_{v,dw}$:	cadmium concentration in the edible part of the vegetable [mg/kg dw];
C_s:	total cadmium concentration in the soil [mg/kg dw];
pH:	- log [H_3O^+ soil] [-];
$clay\%$:	clay content of the soil [-];
$OC\%$:	organic carbon content of the soil [-].

In section 3.2.5.1 limitations of the Bockting and Van den Berg (1992) set of *BCF*s were listed. In Table 15 a more detailed comparison of the Bockting and Van den Berg (1992) approach, on which the *BCF*s for S-EPA has been based, and the Versluijs and Otte (2001) approach is presented. The table gives a general outline of the underlying data and concepts, the assumptions made and practicability.

Table 15: Comparison of two approaches to derive plant-soil relations (BCF values).

APPROACH	Bockting and Van den Berg, 1992	Revised proposal (Versluijs and Otte, 2001)
General	Average or median BCF based on field data	Crop-specific plant-soil relations, obtained by linear regression using field data
Dependent on degree of contamination	No	Yes
Soil properties dependent	No	Yes: pH, clay and organic matter content
Consumption pattern considered	No, only the consumption of potatoes and aboveground vegetables	yes, based on the average consumption pattern
UNDERLYING DATA		
Data sets	Data set includes estimations and data from pot experiments	Field data from different sources
Field data	Dominant	Exclusive
Home grown crops	Dominant	Exclusive
Consumable parts	Sometimes	Exclusive
VALIDITY/USE		
Potential risk assessment	Yes	Yes
Metal concentration range	Probably around Target Value level	depends on metal concentration
Site-specific risk	No	Yes, within certain ranges for pH, clay and organic matter content

From this comparison it can be concluded that generic *BCF*s are not applicable to a specific soil type, for example for representative soil conditions in the south of Sweden. For that reason an alternative approach, analogous to the approach used as technical basis for the Dutch Soil Protection Act, is proposed for the S-RISK model.

Linear regression was performed to determine the coefficients (a_i, b_i, c_i, d_i and e_i) of the plant-soil relations for each vegetable separately. The influence of other factors (e.g. climate, land management, environment) was considered as noise on the data. The resulting plant-soil relations were used to derive crop-specific *BCF*s. Table 16 gives the coefficients of the derived plant-soil relations for cadmium for several vegetables, according to Versluijs and Otte (2001). The available data did not permit the specific consideration of the variable 'f_i' for 'other factors'.

Table 16: Regression coefficients of the plant-soil relations for cadmium according to Versluijs and Otte (2001).

Coefficients Crop	a_i Const.	b_i C_s	c_i pH	d_i OC%	e_i Clay%
Potato	-0.86	0.36	0.06	-0.13	-0.27
Red beet	1.90	0.37	-0.18	-0.30	-0.80
Carrot	0.74	0.45	-0.16	0.20	0.09
Radish	0.00	0.12	-0.32	0.00	0.00
Leek	0.70	0.31	-0.20	-0.29	0.00
Curly kale	1.00	0.39	-0.14	-0.50	-0.40
Lettuce	1.00	0.28	-0.18	-0.19	0.16
Endive	0.00	0.42	-0.10	0.10	0.30
Spinach	1.30	0.28	-0.22	-0.64	0.37
Beans	11.00	0.13	-0.13	-0.10	-9.00

The equations were evaluated for statistical correctness and significance (Versluijs and Otte, 2001). Unfortunately, the statistical significance for the majority of the relationships was insufficient. However, for cadmium the relationships were significant for a considerable amount of crops. Predictions with the derived plant-soil relations have a relatively large uncertainty when using extreme values (either high or low) for the soil concentration, pH, organic matter content and clay content. Versluijs and Otte (2001) proposed that the application range for the derived models is within the 5 and 95 percentiles of the underlying data for soil concentration, pH, organic matter content and clay content. Outside these boundaries the *BCF*s were fixed at a constant value.

For the Swedish total consumption rate and consumption pattern (i.e. the contribution of the crops to the total consumption rate) no detailed information is available. However, the Dutch average consumption pattern is considered a valid basis to calculate a representative *BCF* for the south of Sweden. For the derivation of the consumption rate weighted plant-soil relations, 31 vegetables (including potatoes) were considered. Table 17 gives the average consumption pattern in the Netherlands (Dooren-Flipsen et al., 1996). The consumption pattern, expressed in gram fresh product per day, is converted to the consumption pattern in gram dry weight per day. The water content of the different crops is also given in Table 17. Although the consumption pattern in the Netherlands (and in Sweden) might have changed since these data were reported it is believed that Dooren-Flipsen et al. (1996) is the best representation of the actual Dutch consumption pattern (Swartjes et al., in progress).

Table 17: Average consumption pattern in the Netherlands (Dooren-Flipsen et al., 1996).

no	Group	Crop	Average consumption [g fresh weight per day]	Water content [g/100 g product]	Average consumption [g dry weight per day]
0	Potatoes	Potatoes	179.7	83.3	30.0
1	Root vegetables	Beetroot	5.2	87.3	0.65
		Carrots	13.4	87.8	1.64
		Celeriac	0.8	88.0	0.09
		Turnip	0.8	91.9	0.07
		Radish	0.4	94.8	0.02
		Winter carrot	0.2	87.8	0.02
2	Bulbous vegetables	Onions	17.0	90.8	1.56
		Leek	12.9	83.0	2.19
3	Fruit vegetables	Tomatoes	26.1	94.0	1.56
		Cucumber	8.0	96.1	0.31
		Melon	2.2	89.7	0.23
		Maize	1.4	76.0	0.34
4	Cabbages	Cauliflower	16.0	92.3	1.23
		brussels sprouts	4.7	86.0	0.65
		White cabbage	7.0	95.3	0.33
		Red cabbage	5.1	91.6	0.43
		ox heart cabbage	2.0	95.3	0.10
		Curly kale	4.9	84.5	0.76
		Broccoli	2.0	90.7	0.18
5	Leafy vegetables	lettuce (head)	8.5	95.4	0.39
		Endive	7.4	93.8	0.46
		Spinach	10.4	91.6	0.88
		Chicory	9.2	95.3	0.43
6	Legumes (peas and beans)	Green bean	11.7	90.3	1.13
		String/bush bean	3.1	90.3	0.30
		broad/horse/fava bean	2.5	88.9	0.28
		garden pea	14.8	88.9	1.64
7	Beans	haricot bean	0.9	77.1	0.20
		kidney bean	1.8	77.1	0.40
8	Stem and stalk vegetables	Rhubarb	0.7	93.6	0.05
		Asparagus	1.7	92.3	0.13

Details on the consumption pattern are given in Appendix A.

When for a specific crop no valid plant-soil relation could be derived, the geometric mean of the dataset was used, in analogy with Versluijs and Otte, 2001. This approach is summarized in Figure 6.
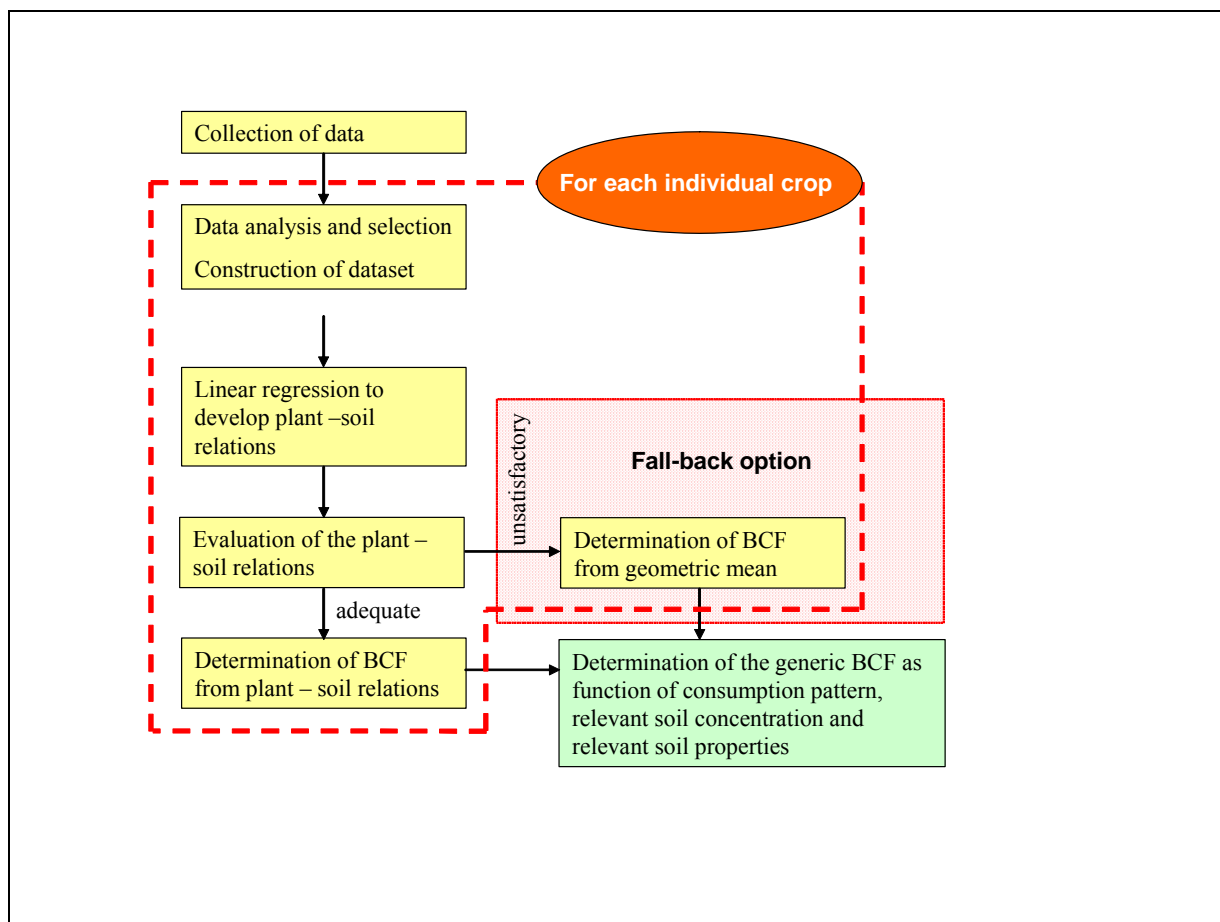
Figure 6: Schematisation of the applied procedure to derive vegetable-specific plant-soil relations.

For the plant-soil relations for cabbage and tomatoes the F-test proved that the derived coefficients were insignificant (Versluijs and Otte, 2001). Furthermore, Monte Carlo analyses showed a high uncertainty of the plant-soil relations for beans (Versluijs and Otte, 2001). Therefore, the geometric mean was used for cabbage and beans (see fall-back option Figure 6). For tomatoes, the quality of the data was doubted, since other data sources showed significantly lower uptake. Therefore, the geometric mean was not used in the calculations, either.

It is expected that with increasing clay content and increasing organic matter content the plant uptake will decrease, resulting in lower *BCF* values. However, for some crops the coefficients for clay and organic matter in the Freundlich equation show a positive relationship, i.e. higher *BCF*s with higher organic matter and clay contents. This positive relationships cannot be explained with general knowledge on the influence of organic matter and clay on bioavailability and, hence, on plant uptake. However, because there is statiscally is insufficient evidence to reject them, these plant-soil relations are used

Bioavailability, and hence the *BCF*, is a function of total soil concentration and soil properties. The average cadmium concentration in the south of Sweden is 0.3 mg/kg dw (Naturvårdsverket, 1997; J&W, 2001; Scandiaconsult, 2001; Sweco vbb viak, 2002). Because this concentration is in the lower concentration range of the data from which the plant-soil relations have been derived the *BCF* used in S-RISK is based on a soil concentration of 1.0 mg/kg dw. This means that the guidelines are primarily related to a soil

concentration around a value of 1.0 mg/kg dw. It has been assumed that the clay content in a typical soil in the south of Sweden is low. For this reason a clay percentage of 3%, which is the minimum of the range for which the plant-soil relations are considered reliable, is adopted. The soil properties that represent soil conditions for southern Sweden are presented in Table 18. The ranges in which the plant-soil relations are considered reliable are also given in the table.

Table 18: Soil properties representing southern Sweden soil conditions and ranges in which the plant-soil relations are considered reliable.

Parameter	Value	*BCF* reliability range
soil concentration [mg/kg dw]	1.8	> 0.12 and < 3.2
pH [-]	5.5	> 5.4 and < 7.5
f_{OC} [%]	1.16	> 0.9 and < 13
clay% [%]	3.0	> 3 and < 33

Table 19 gives the resulting *BCF* values based on the plant-soil relations or geometric means for all vegetables for which data were available.

When all individual *BCF*s would be used, independent of the respective crop groups, some crop groups for which *BCF*s for a relatively large number of different crops are available would have more weight in the overall consumption rate weighted *BCF*. With the purpose to prevent that too much stress is given to a specific crop group, the overall consumption rate weighted *BCF* is based on the average *BCF*s for the *crop groups* instead of on the average *BCF*s for the individual crops. For this reason, the consumption rate weighted average *BCF*s for each crop group has also been given in Table 19.

The overall consumption rate weighted *BCF*s are also given in Table 19. On a dry weight basis [(mg/kg dw plant)/(mg/kg dw soil)], the following *BCF*s are calculated: 0.158 for root crops and 0.483 for aboveground vegetables On a fresh weight basis [(mg/kg fw plant)/(mg/kg dw soil)], the *BCF* for root vegetables is 0.024, the *BCF* for above-ground vegetables is 0.052, and the overall *BCF* is 0.031. Note that this value is very sensitive to the high contribution of potatoes from 62%.

Table 19: BCF values for cadmium based on the regression equations or geometric mean, for several crops and crop groups and consumption rate weighted BCFs.

Crop group	Crop	Weighting factor crop	Moisture content %weight basis	BCF dry weight	BCF wet weight	Model (1) or geom. mean (0)	Participation yes(1)/ no (0)	BCFcrop group dry weight	BCFcrop group wet weight	Weighting factor group	Participation group yes(1)/ no (0)	soil content	pH	OC%	Clay%
0 potatoes	potatoes	61,6	83,30	0,124	0,021 0,000	1	1	0,124	0,021	61,6	1	selected: 1,8	5,0	1,16	0,10
1 root vegetbales	beetroot	1,3	87,30	0,460	0,058	1	1	0,574	0,071	5,09	1	adapted: 1,8	5,7	3,40	13,00
	carrots	3,4	87,80	0,626	0,076	1	1					adapted: 1,8	5,1	1,16	1,00
	celeriac	0,2	88,00												
	turnip	0,1	91,90												
	radish	0,05	94,80	0,005	0,000	1	1					adapted: 2,6	6,0	4,10	24,00
	winter carrot	0,04	87,80												
2 bulbous crops	onions	3,2	90,80					0,170	0,029	7,7	1				
	leek	4,5	83,00	0,170	0,029	1	1					adapted: 1,8	5,7	3,40	12,00
3 fruit vegetables	tomato	3,2	94,00	3,020	0,181	0	1	3,020	0,181	5,0	0	adapted: 1,8	6,8	2,90	12,00
	cucumber	0,6	96,10												
	melon	0,5	89,70												
	maize	0,7	76,00												
4 cabbages	cauliflower	2,5	92,30					0,500	0,054	7,6	1				
	brussels sprout	1,3	86,00												
	white cabbage	0,7	95,30	0,288	0,014	0	1					adapted: 1,8	5,7	2,90	12,00
	red cabbage	0,9	95,30	0,559	0,026	0	1					adapted: 0,4	5,7	3,40	12,00
	oxheart cabbage	0,2	95,30												
	curlt kale	1,6	84,50	0,559	0,087	1	1					adapted: 0,4	5,7	3,40	12,00
	brocolli	0,4	90,70												
5 leafy vegetables	lettuce	0,8	95,40	0,750	0,034	1	1	1,018	0,076	4,4	1	adapted: 1,8	5,7	2,90	12,00
	endive	0,9	93,80	0,456	0,028	1	1					adapted: 1,8	5,7	3,40	12,00
	spinach	1,8	91,60	1,417	0,119	1	1					adapted: 1,8	5,0	1,16	3,00
	chicory	0,9	95,30												
6 legumes	green beans	2,3	90,30					0,000	0,000	6,9	0				
	string/bush beens	0,6	90,30												
	broad/horse/fava beans	0,6	88,90												
	gaden peas	3,4	88,90												
7 beans	haricot bean	0,4	77,10	0,427	0,098	0	1	0,427	0,098	1,2	1	adapted: 1,8	5,1	1,50	12,00
	kidney beans	0,8	77,10	0,427	0,098	0	1					adapted: 1,8	5,1	1,50	12,00
8 stem and stalk vegetables	rhubarb	0,1	93,60					0,000	0,000	0,4	0				
	asparagus	0,3	92,30									average: 1,6	5,6	2,7	11,5
All vegetables		99,89								99,89					

	BCF above ground vegetables:	BCFroot crops:	BCF overall:
dry weight	0,483	0,158	
wet weight	0,052	0,024	0,031

A comparison with the *BCF*s of Bockting and Van den Berg (1992) and from other researchers has been described in Appendix B. From this comparison it can be concluded that the *BCF* value from the proposed plant-soil relations from Versluijs and Otte (2001), suited for the south of Sweden, is similar to the Bockting and Van den Berg (1992) *BCF* and of the same order of magnitude as the *BCF*s from other vegetable accumulation models.

- *Uptake of organic compounds: modelling equations*

A concept for estimating PAH concentrations in vegetables due to soil contamination is detailed in Appendix C and is shortly described hereafter. This concept include uptake from soil and both gaseous and particle deposition on the plant. The model does not account for soil splash.

For uptake from the soil and air (gas phase), the simplified version of the PlantX model from Trapp & Matthies (1995) is suitable for incorporation in a guideline development concept. It is also used in the European EUSES model for risk assessment of new and existing substances and the Dutch CSOIL exposure model. Besides the conceptual PlantX model, also the findings of McLachlan and co-workers are incorporated in the final model equations of S-RISK. McLachlan (and co-workers) discussed a theoretical framework on both dry gaseous deposition and particle-bound deposition of semi-volatile organic compounds (McLachlan, 1999; McLachlan et al., 1995, 1999). To account for plant concentrations attributable to wet plus dry deposition, an additional model equation (Lorber et al., 1994) is incorporated.

Modelled *BCF*s for the different soil-to-plant and air-to-plant pathways suggest that the concentration of any given PAH in the above-ground vegetables is mainly due to particle bound deposition of that compound. For the heavier PAHs which - according to the framework of McLachlan (1999) - the dominant route in the air-to-plant transfer is particle bound deposition, this will most likely be the case for benzo(g,h,i)perylene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene.

However, for the lighter PAH compounds who are totally or almost exclusively found in the gas phase and for which the dominant route in the air-to-plant is equilibrium partitioning, the modelled concentration in the above-ground vegetables will be overestimated if particle bound deposition is included. This will be the case for acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, naphthalene, phenanthrene and pyrene.

For the intermediate weight PAHs, another factor complicates the unrestricted use of the modelled particle deposition *BCF* in the overall estimation of the concentration in above-ground vegetables. Following McLachlan's framework, the dominant route in air-to-plant transfer of PAH compounds with $8.8 < \log K_{OA} < 11$ is kinetically limited gaseous deposition. This means that dry gaseous deposition is still the dominant uptake process but the storage capacity of the vegetables for the chemical is so high that an equilibrium is not approached over the time of exposure. This means that the modelled particle deposition *BCF* would theoretically approach zero (or at least: gaseous deposition *BCF* >> particle deposition *BCF*). However, measured data of gas and particle phase concentrations of these PAHs (benzo(a)antracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene and chrysene) suggest that these compounds are almost exclusively found in the particle phase.

Another remark is that in literature volatilization of (semi) volatile organic compounds from soil is generally not accounted for: concentrations of these substances are mainly related to ambient gas/particle phase concentrations (immission data). It is assumed that ambient gas phase concentrations of PAHs, especially in urban regions will probably be higher than the gas phase concentrations originating from the contamination in soil.

Given the above mentioned remarks, it is concluded that in S-RISK the use of measured bioconcentration factors in the estimation of the concentration in vegetables should be favoured.

- *Uptake of organic compounds: measured BCF*

To obtain insight in measured *BCF* values, in contrast to modelled *BCF* values for PAHs, a literature survey was accomplished. In this survey two major sources were used: (1) literature already available to the author and (2) reference databases like CurrentContents and ToxLine. For the latter a combination of the keywords: 'PAH', 'uptake', 'accumulation', 'vegetables', and 'plant' was used sometimes in combination with more specific plant species (like 'ryegrass') or substances (like 'pyrene' and 'phenanthrene'). From the provided references a selection was made between the reported studies using to the following conditions:

- Reported concentrations were obtained by measurements, not models.
- Measurements were done in actual (natural or artificial) soils, not in solution cultures.
- The *BCF* values are based on the ratio of PAH concentration in dry weight plant tissue with PAH concentration in dry weight soil.
- For each *BCF* value, the accompanying soil concentration must be available.

The literature study revealed that information about *BCF* values based on measurements is limited. Only a few peer-reviewed studies were found. The small number of studies was expected based on earlier experiences with plant studies.

Table 20 shows the *BCF* values obtained from literature data. In the upper part of the table the (ranges of) *BCF* values are given, the lower part of the table shows the accompanying soil concentrations. Due to the limited amount of data, *BCF* values for non-vegetables were included too.

The study of Tao et al (2004) considered PAH concentrations in miscellaneous vegetables grown on two types of loamy soil, one site was moderately contaminated and another heavily contaminated site close to pollution sources and irrigated with waste water. The selected vegetables included cabbage, celery, cauliflower, spinach, and turnip. Kipopoulou et al (1999) studied PAH concentrations in vegetables grown in the greater industrial area of Thessaloniki. They considered both soil-to-vegetables and air-to-vegetables *BCF* values.

The two studies from Xu et al (2005) and Ling and Gao (2004) were pot experiments were soils were spiked with phenanthrene and pyrene. Since only abstracts were available no exact details about the experimental setup and result can be given.

The data from Wild and Jones (1992) are also used within the Dutch framework of risk assessment in relation to soil pollution. It describes an experiment where carrots were grown on sludge amended soils. The liquid sewage sludge was spiked with PAHs and mixed with soil. The experimental setup aimed at conditions that maximise plant uptake. After 82 days the carrots were harvested, cleaned, and peeled with a normal kitchen peeler. Both core as peeling were analysed for PAHs.

Table 20: BCF values and soil concentrations from selected journal articles. Soil refer to the soil type for which concentrations are given in the second part of the table.

BCF [mg/kg dry weight plant / mg/kg dry weight soil]

Plant	Part	Soil	Organic Matter soil [%]	Naphthalene	Acenaphtene	Acenaphtylene	Fluorene	Phenanthrene	Fluoranthene	Anthracene	Pyrene	Benzo[a]anthracene	Chrysene	Benzo[b]fluoranthene	Benzo[k]fluoranthene	Benzo[a]pyrene	Indeno[123cd]pyrene	Dibenz[ah]anthracene	benzo[ghi]prylene	Reference
Miscellaneous	root	1	0.93[1]	0.101	0.076	0.266	0.269	0.069	0.024	0.295	0.019	0.002	0.004	0.021	0.006	0.068				1
		2	3.29[1]	0.078	0.039	0.264	0.705	0.062	0.009	0.080	0.008	0.001	0.002	0.002	0.008	0.028		0.003	0.001	1
Miscellaneous	aireal	1	0.93[1]	1.188	0.532	2.064	0.633	0.183	0.244	0.507	0.062	0.134	0.081	0.056	0.047	0.193				1
		2	3.29[1]	0.608	0.060	0.678	0.868	0.202	0.093	0.191	0.016	0.022	0.020	0.006	0.013	0.128	0.000	0.010	0.003	1
Carrot	core	3						3.200	1.11	1.400	1.400	0.170	0.230	0.120	0.140	0.190	0.160	0.200	0.130	2
Letuce/endive	leaves	3						8.290		2.620	7.830		1.930	0.530	0.360	0.160	0.170		0.110	2
Ryegrass	roots	4						0.24-4.25			0.58-2.28									3
	shoots	4						0.17-2.12			0.20-1.50									3
Amaranth	roots	5						0.136-0.776			0.603-1.43									4
	shoots	5						0.116-0.951			0.082-0.517									4
Carrot	peel	6	1.21	9.00			5.86	1.21	0.15	0.41	0.19		0.09	0.04	0.04	0.10			0.18	5
	core	6	1.21	3.56			3.32	0.20	0.01	0.12	0.00		0.00	0.01	0.01	0.09			0.02	5
	peel	7	1.69				4.55	0.39	1.20	0.94	0.93		0.27	0.13	0.09	0.16			0.22	5
	core	7	1.69	5.24			3.14	0.19	0.01	0.06	0.03		0.01	0.00	0.00	0.06			0.01	5
	peel	8	2.32				3.02	0.58	1.06	0.50	0.77		0.23	0.12	0.08	0.12			0.16	5
	core	8	2.32	2.21			2.33	0.16	0.03	0.09	0.05		0.04	0.00	0.00	0.05			0.00	5
	peel	9	4.46				0.83	0.20	0.49	0.04	0.46		0.09	0.04	0.03	0.04			0.14	5
	core	9	4.46	0.65			0.49	0.05	0.01	0.01	0.02		0.01	0.00	0.00	0.01			0.01	5

Soil Concentration [mg/kg kg dry weight]

		Soil	Organic Matter soil [%]	Naphthalene	Acenaphtene	Acenaphtylene	Fluorene	Phenanthrene	Fluoranthene	Anthracene	Pyrene	Benzo[a]anthracene	Chrysene	Benzo[b]fluoranthene	Benzo[k]fluoranthene	Benzo[a]pyrene	Indeno[123cd]pyrene	Dibenz[ah]anthracene	benzo[ghi]prylene	Reference
		1	0.93[1]	0.265	0.008	0.011	0.029	0.173	0.042	0.122	0.094	0.045	0.097	0.043	0.112	0.041				1
		2	3.29[1]	0.969	0.039	0.024	0.048	0.498	0.448	0.712	0.544	0.357	0.624	0.520	0.375	0.332	0.313	0.111 0.0004	336	1
		3		0.017	0.170		0.101	0.009	0.007	0.001	0.005	0.005	0.004	0.003	0.001	0.002	0.002	3	3.6		2
		4						3.31-379			4.22-365										3
		5						7.45-457			8.01-489										4
		6	1.21	0.86			2.40	3.00	1.50	0.33	2.00		1.70	0.99	0.48	0.82			2.5	5
		7	1.69	5.25			14.64	18.30	9.15	2.01	12.20		10.37	6.04	2.93	5.00			15.25	5
		8	2.32	16.00			44.64 149.0	55.80	27.90	6.14	37.20		31.62 105.5	18.41	8.93	15.25			46.5 155.2	5
		9	4.46	53.41			4	186.30	93.15	20.49	124.20	7	61.48	29.81	50.92				5	5

[1] Measured as TOC in µg C/g. References: 1: Tao et al. (2004), 2: Kipopoulou et al. (1993), 3: Xu et al. (2005), 4: Ling and Gao (2004), 5: Wild and Jones (1992).

From Table 20 it is clear that large differences between reported *BCF* values exist. It is assumed that these differences are caused by the differences in each study like the determination of PAH in different plant species, the different soil types, spiked or not spiked, different plant parts and differences in analytical method.

In Table 21 the *BCF* values as used in the Flemish derivation of soil guideline values for PAHs are given (Nouwen et al., 2001). The *BCFs* are mainly based on data of Cröβmann (1992) and Wild and Jones (1992). Data of Cröβmann apply to a large extent to PAH uptake from the soil (pot experiments). Uptake from the atmosphere is considered negligible.

Table 21: BCF values used in Vlier-Humaan (Flanders) and calculated K_{pl} values for use in S-RISK.

PAH	$BCF_{above-ground}$ (mg/kg dw)/(mg/kg dw)	BCF_{root} (mg/kg dw)/(mg/kg dw)	K_{pl} * (mg/kg fw)/(mg/kg dw)
Acenaphthene	2.32	2.32	0.421
Acenaphthylene	2.32	2.32	0.421
Anthracene	0.022	0.002	0.0009
Benzo(a)anthracene	0.007	0.015	0.0025
Benzo(a)pyrene	0.002	0.012	0.002
Benzo(b)fluoranthene	0.014	0.005	0.0012
Benzo(g,h,i)perylene	0.004	0.011	0.002
Benzo(k)fluoranthene	0.003	0.015	0.002
Chrysene	0.008	0.013	0.002
Dibenzo(a,h)anthracene	0.0003	0.0005	0.0001
Fluoranthene	0.029	0.023	0.004
Fluorene	0.005	0.009	0.002
Indeno(1,2,3-cd)pyrene	0.0001	0.0002	0.00003
Naphthalene	2.92	2.92	0.53
Phenantrene	0.041	0.031	0.006
Pyrene	0.011	0.021	0.004

* $K_{pl} = (BCF_{above-ground} \times dw_{above-ground} \times f_{leaf}) + (BCF_{root} \times dw_{root} \times f_{root})$, where $dw_{above-ground} = 0.117$, $dw_{root} = 0.202$, $f_{leaf} = 0.24$, and $f_{root} = 0.76$.

It is proposed to use the *BCF* values of Nouwen et al. (2001) in S-RISK as best estimates since (i) they are based on a large database, (ii) data of Wild and Jones (1992) are also used within the Dutch framework of risk assessment in relation to soil pollution, and (iii) the overall database is used in the Flemish derivation of soil guideline values for PAHs.

The *BCF* values reported in Nouwen et al. (2001) are converted to K_{pl} values for input in S-RISK, following:

$$K_{pl} = \left(BCF_{above-ground,dw/dw} \times dw_{above-ground} \times f_{leaf}\right) + \left(BCF_{root,dw/dw} \times dw_{root} \times f_{root}\right)$$

where $BCF_{dw/dw}$ has the dimension (mg/kg dw plant)/(mg/kg dw soil), *dw* refers to the ratio dry weight to fresh weight (0.202 for roots and 0.117 for above-ground vegetables), and f_{leaf} (fractional consumption of leaf and stem vegetables in the total vegetable consumption, calculated on dry weight basis) and f_{root} (fractional consumption of root vegetables in the total vegetable consumption, calculated on dry weight basis) are 0.24 and 0.76 respectively (calculated on the basis of the contribution to the average consumption pattern, see Appendix A). Calculated K_{pl} values are given in Table 21.

3.3 Human exposure

In S-RISK, the exposure pathways for the three types of land-use are the same as in the current S-EPA methodology.

3.3.1 Intake of contaminated soil

- S-EPA

Oral exposure to contaminants in soil is assumed to occur as direct intake or via fingers and hands that are put in the mouth. Important parameters are average daily soil intake and bioavailability of the contaminant in the human body. The bioavailability of the contaminant in the soil is assumed to correspond to the bioavailability considered when deriving the toxicological data.

The reference soil concentration for the soil ingestion pathway, C_{is} [mg/kg], is:

$$C_{is} = \frac{TRV}{R_{is}} \times 10^6$$

where:

TRV: the toxicological reference value [mg/kg.d];
R_{is}:   the average daily soil intake [mg/kg.d], i.e. long-term soil intake for non-genotoxic substances and integrated lifetime soil intake for genotoxic substances (cfr. Table 22).

Table 22: Soil ingestion parameters used by S-EPA for land with sensitive land-use (Naturvårdsverket, 1996b).

Parameter	Land with sensitive land-use (KM)
average daily soil intake [mg/d]	150 child 50 adult
Long-term soil intake per unit body weight [mg/kd.d]	10 child 0.7 adult
Integrated lifetime soil intake [mg/kg.d]	1.5

In the case of land with less sensitive land-use, Naturvårdsverket (1996b) makes use of an integrated soil ingestion rate of 8 mg.year/kg.d (on the basis of MDEP's category S-2 soil: 50 mg/d x 5 days/week x 6 months/year).

Table 23: Soil ingestion parameters used by S-EPA for land with less sensitive land-use (Naturvårdsverket, 1996b).

Parameter	Land with less sensitive land-use (KM)
average daily soil intake [mg/d]	0.3 long term 0.1 integrated lifetime
exposure time [a]	27 long term 75 integrated lifetime

From this table, it is seen that only adults are considered in less sensitive use. The value of 0.3 is derived from the averaging on a yearly basis, where the value of 0.1 results from multiplying with 25/75 years. The definition of less sensitive land-use says that adults are considered to be present during working hours, while children could be present temporarily.

- S-RISK

In the framework of the revision of the Dutch Intervention Values (Lijzen et al., 2001) the amount of soil ingestion was evaluated for land with sensitive land-use (Otte et al., 2001). In Table 24 an overview is given of the available tracer studies for *children*. Compared to the evaluation of 2001 the last 3 references in Table 24 are added.

From Otte et al. (2001) follows an average amount of soil ingestion of about 100 mg.d^{-1} and a 95% confidence limit of the average amount of soil ingestion of (75-125 mg.d^{-1}). The median value is about half the mean value and is similar to the geometric mean. This indicates that soil ingestion by children has a positively skewed distribution. The 90th percentile of the soil ingestion by children is about 150 mg.d^{-1} and the 95th-percentile is about 200 mg.d^{-1}. Including additional studies the average amount of soil ingestion is 96 mg.d^{-1}. The median value is 39 mg.d^{-1}. The 90th percentile of the soil ingestion by children is about 135 mg.d^{-1} and the 95th-percentile is about 200 mg.d^{-1}.

In an ongoing study for the revision of the Flemish exposure model Vlier-Humaan the same literature references where interpreted slightly different. In this study and provisional report (Bierkens & Cornelis, in prep.) some references were excluded, because new statistical interpretations were carried out on earlier datasets. The same research group did a revised statistical interpretation of the data, the old interpretation was excluded from calculating the mean. Following this approach five references were left out (Binder et al., 1986; Calabrese et al., 1989; Stanek en Calabrese, 1995a; Stanek en Calabrese, 1995b; Calabrese and Stanek, 1995). This leads to an average soil ingestion rate of 72 mg.d^{-1} (with a 95% upper limit of the confidence interval of 93 mg.d^{-1}). The median value is 56 mg.d^{-1}. The 95th percentile of the soil ingestion by children based on the selected studies is almost 200 mg.d^{-1}.

Table 24: Daily soil ingestion by children [mg/day] (source: Otte et al., 2001).

Reference	Number of children	Tracer	Mean	SD	95% conf. limit	Median (50-perc.)	90-perc. (logn. distr.)	95-perc. (logn. distr.)
Binder et al., 1986	59	Al	181	203		121		584
		Si	184	175		136		578
		LTM	**108**	121		**88**		**386**
Clausing et.al., 1987 (NL)	18	Al	232	263		93	579	707
		LTM	**105**	67		**82**	**162**	**201**
		hospital (backgr.)	**49**	22		**48**	75	79
Calabrese et.al., 1989: soil	64	Al	153	852		29		223
soil data		Si	154	693		40		276
		Y	85	890		9		106
		mean Al, Si, Y	**131**			**26**		**202**
Davis et al.,1990	101	Al	39	14		25		
		Si	82	12		59		
		mean Al, Si	**61**			**42**		
Van Wijnen et al.., 1990 (NL)								
daycare 1+2	166	LTM	**162**	286		**114**		
campground	78	LTM	213			160		
background (hospital)	15	LTM	**93**	46	67-119	110		
daycare 1, selection	22	LTM					**265**	
daycare 2, selection	15	LTM					**172**	
campground, selection	32	LTM					258	
Stanek & Calabrese, 1995a	64	Al	122			29	131	254
		Si	139			32	206	224
		total	**179**			**45**	**186**	**208**
Stanek & Calabrese, 1995b	229	median Al, Si, Ti	**113**			37	**194**	**249**
		median best 4 tracers	**104**			**37**	**156**	**217**
Calabrese et a.l, 1997. (not used)	64	median best 4 tracers	7	75		1	73	160
Average of all references (Otte et al., 2001)			*102*			*42*	*150*	*230*
Calabrese et al, 1996.	64	Al	136					
		Si	133					
		Y	97					
		mean Al, Si, Y	**122**					
Thompson en Burmaster, 1991	59	Al	97					
		Si	85					
		mean Al, Si	**91**					
Stanek et al., 2001a	64	best 4 tracers	**31**			17		106
Average of all references (Bierkens & Cornelis, in prep.)			*96*			*39*	*135*	*202*

[1] Bold data are used in calculations; 1 (averaged) value per reference.

From the evaluation of the Dutch Intervention Values (Otte et al., 2001) a realistic or conservative value for the amount of soil ingestion for children could be selected. In case average exposure parameters are chosen the average value of 100 mg.d[-1] is recommended. In case it is found appropriate to use a more safe value, 125 (upper limit of confidence interval) or 150 - 200 mg.d[-1] (90-95th -percentile) could be used.

In the current VITO study an average soil ingestion of 75 mg.d[-1] was derived. As a more certain estimate of the average the value of 100 mg.d[-1] was proposed. The 95-percentile of the soil ingestion also could be selected, being 200 mg.d[-1].

Overall, the value of 100 mg.d^{-1} is proposed as the amount of soil ingestion for children as a relative certain estimate of the mean value. Depending on the chosen protection level also a higher ingestion could be selected: in that case a more conservative value between 100 mg.d^{-1} and 200 mg.d^{-1} could be selected.

Besides these tracer studies, there are references in which the exposure of children (and adults) due to soil ingestion is estimated. In US-EPA (1997) an overview of these data is given. One of these references is Hawley (1985). Some of these articles are based on the evaluation of seven studies (see Table 24) and others on the measured soil and dust loading on hands, and the hand-mouth behaviour of children. These studies are considered to be supplementary to the quantitative data in the other references. Simon (1998) gives a fairly complete overview of the data on soil ingestion, but no conclusions are given on the best data.

For *adults* only a limited number of data on soil ingestion is available (see Table 25).

Table 25: Soil ingestion by adults [mg/day] (source: Otte et al., 2001).

Reference	Number of adults	Tracer	Mean	SD of mean	Median (50-perc.)	75-perc.	90-perc.	95-perc.	Min.	Max
Calabrese et al., 1990	6	median of Al, Si, Y, Zr	**38**		29					
		average of Al, Si, Y, Zr	**39**		30					
		Al	77	65	57					
		Si	5	55	1					
		Y	53	51	65					
		Zr	22	141	-4					
Stanek et al., 1997	10	*median of 4 best tracers over 4 weeks*	**6**	**165**	**-11**	**34**	**201**	**331**	**-400**	**620**
		week 1	67	202	-14	37	384	620	-39	620
		week 2	**44**	120	18	37	210	376	-52	376
		week 3	**49**	127	-5	120	269	285	-84	285
		week 4	-137	126	-143	-93	21	100	-400	100

Calabrese et al. (1990) and Stanek et al. (1997) quantified soil ingestion in two tracer studies with adults. Calabrese et al. (1990), being part of a childhood soil ingestion study, yielded mean soil ingestion values of 77 (Al), 5 (Si), 53 (Y) and 22 (Zr) mg.d^{-1}, from which an average of 39 mg.d^{-1} was derived. Stanek et al. (1997), also part of a larger study, involved 10 adults. Based on the 'best tracer method' (BTM) with Al, Si, Ti, Y and Zr the average daily soil ingestion over 4 weeks was 6 mg.d^{-1}. Based on this result, the average ingestion is concluded to be 10 mg.d^{-1}. Ignoring the results of the last week (in which very low values were measured), the mean of the first three weeks is 53 mg.d^{-1}. This illustrates that a lot of uncertainties are involved. Nevertheless, these studies give the best quantitative estimates currently available.

In Hawley (1985) estimates are based on soil and dust on hands and hand-to-mouth transfer. A soil ingestion of 480 mg per day was estimated for adults in outdoor activities and for indoor activities soil ingestion between 0.56 and 110 mg.d^{-1} was estimated. In US-EPA (1997) an estimate of 10 mg mg.d^{-1} is also mentioned, based on arsenic levels in urine,

hand-to-mouth transfer, and activity patterns. Because of the lack of direct measurements these studies have a lower reliability and are not incorporated in this study.

In the study for the revision of the Flemish exposure model Vlier-Humaan (Bierkens & Cornelis, in prep.), the same two literature references where used. The only difference is that the values of the first week and last week of the Stanek et al. (1997) study were left out, because of the uncertainties described previously. This leads to an average soil ingestion for adults of 42 mg.d^{-1} (with a 95% upper limit of the confidence interval of 61 mg.d^{-1}). The median value is 40 mg.d^{-1}. The 95th percentile of the soil ingestion by adults was estimated to be 71 mg.d^{-1}.

The proposed amount of soil ingestion for adults is 50 mg.d^{-1}. Depending on the chosen protection level also a higher ingestion could be selected: a more certain value for the average of 60 mg.d^{-1} or the estimate of the 95-percentile of the soil ingestion of 70 mg.d^{-1}.

In addition to the data on soil ingestion in the literature, the data used in risk assessment tools in different European Countries and the USA are summarized (Table 26). More details can be found in Rikken et al. (2001) and Swartjes (2002). In all models an age-adjusted soil ingestion factor is used, because unconscious soil ingestion is different for children and adults. The age categories used and corresponding soil ingestion rates are different for all models.

For *children* the yearly averaged soil ingestion rates of the other exposure models range from 50 mg.d^{-1} (the Flemish Vlier-Humaan model) up to 200 mg.d^{-1} (the Bask LUR and the Italian ROME01 models). An exception is the high value of 1,000 mg.d^{-1} used in the Danish CETOXhuman model mentioned in Swartjes (2002). However, the 'formal' value for the amount of soil ingestion for children in Denmark, on which the Danish Soil Quality Criteria are based, is 200 mg per day (pers. comm. L. Anderson), being a 90-95 percentile of a group of children (pers. comm. Larsen). For adults an amount of soil ingestion of 25 mg.d^{-1} is assumed.

Most models probably use (partly) the same literature data. Differences are the result of the use of the average or higher (90-95) percentile exposure rates or differences in interpretation.

For *adults* the yearly averaged soil ingestion rates range from 10 mg.d^{-1} (Caltox model) up to 100 mg.d^{-1} (the Italian ROME01 model). The value of 50 mg.d^{-1} is often used for this parameter.

Table 26: Soil ingestion rates used in different models (source: Rikken et al., 2001; Swartjes, 2002).

Model; country in which developed	Yearly average soil ingestion rate for adults [mg.d⁻¹]	Yearly average soil ingestion rate for children [mg.d⁻¹]
CLEA; UK	60	80 [1]
CalTOX; California, USA	10	60
UMS; Germany	16 [2]	123 (1-3 year) [3] 74 (4-8 year) [3]
CSOIL 1995; NL	50	150
CSOIL 2000; NL	50	100
CETOX-human; Denmark	25	200 /1,000 [4]
LUR; Spain	n.a.	200
NoNameFrance 2000; France	50	150
NoNameSweden; Sweden	50	150
ROME01; Italy	100	200
Vlier-Humaan; Belgium	20	50
NICOLE; n.a.	1	40

1) With unconscious soil ingestion: 114 mg/kg (5,000 mg per day for a maximum of 30 days in the second year of life).
2) The intake in the human body, based on a soil ingestion of 30 mg per day and an exposure duration of 190 d.year⁻¹.
3) Based on a soil ingestion of 250 mg per day (1-3) and 150 mg per day (4-8) and an exposure duration of 80 d.year⁻¹.
4) Although a value of 1,000 mg.d⁻¹ is mentioned, 200 mg.d⁻¹ is the formal figure.

It should be noted that acute exposure and toxicity due to deliberate soil ingestion by children ('pica behaviour') are not considered in this study. Further research on the comparison of the short-term exposure of deliberate soil eating to acute toxicity data is particularly recommended (Calabrese et al., 1997; Kempchen, 2000).

From the above evaluations, a soil ingestion rate of 100 mg/d for children and 50 mg/d for adults is proposed for land with sensitive land-use. If a higher protection level is desired, higher values can be used. The average soil intake values are calculated according to S-EPA methodology and are given in Table 27.

Table 27: Proposed soil ingestion parameters for land with sensitive land-use.

Parameter	Land with sensitive land-use (KM)
average daily soil intake [mg/d]	100 child 50 adult
Long-term soil intake per unit body weight [mg/kd.d]	7 child 0.7 adult
Integrated lifetime soil intake [mg/kg.d]	1.3

In the case of land with less sensitive land-use, there are no changes in the values as the adult soil ingestion value is maintained (

Table 28).

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Table 28: Proposed soil ingestion parameters for land with less sensitive land-use.

Parameter	Land with less sensitive land-use (MKM)
average daily soil intake [mg/kg.d]	0.3 long term 0.1 integrated lifetime
exposure time [a]	27 long term 75 integrated lifetime

3.3.2 Dermal contact with soil and dust

- S-EPA

The model for exposure due to dermal contact with soil and dust is based on the model used in CSOIL.

The reference soil concentration for the dermal pathway, C_{du} [mg/kg] is calculated as:

$$C_{du} = \frac{TRV}{f_{du} \times R_{du}} \times 10^6$$

where

TRV: toxicological reference value (oral) [mg/kg.d];
f_{du}: substance specific relative absorption factor for dermal uptake [-];
R_{du}: average daily dermal exposure [mg/kg.d], i.e. long-term dermal exposure for chronic exposure for non-genotoxic substances and integrated lifetime dermal exposure for genotoxic substances.

Substance specific relative absorption factors for dermal uptake, f_{du}, are at present originating from MDEP's *RAF*s (relative absorption factors).

A soil exposure of 0.51 mg/cm² is used for children as well as adults. No rationale for this choice is given. The value 0.51 mg/cm² is the amount of soil or dust on the skin of children during outdoor activities (ECETOC, 1992), implicating that indoor exposure to soil or dust is omitted. However, in MDEP (1994), this value is presented - on the basis of Hawley (1985) - as the amount of soil in contact with the skin for both children and adults, for the days the receptor is exposed both indoors and outdoors. Exposure of adults is quantified here as it is assumed that all ages have the opportunity for contact with the soil through playing or gardening (MDEP, 1994).

The exposure time on land with less sensitive land-use is assumed to be a third of the exposure time on land with sensitive land-use. Input values for R_{du} are given in Table 29. No rationale is given for the deviation from the ECETOC/CSOIL methodology.

Table 29: Dermal contact parameters used by S-EPA (Naturvårdsverket, 1996b).

Parameter	Land with sensitive land-use (KM)	Land with less sensitive land-use (MKM)
Long-term dermal soil exposure per unit body weight [mg/kd.d]	20 child 1.5 adult	7 child 0.5 adult
Integrated lifetime dermal soil exposure [mg/kg.d]	3	1

- S-RISK

The methodology for calculation of dermal exposure to soil and dust is not revised: the use of absorption factors is not discussed. However, the parameter values for the (relative) absorption factors are revised. An elaborated discussion can be found in Appendix D. The recommended values for f_{du} are presented in Table 30.

Table 30: Recommended values for f_{du} [-] for S-RISK.

Substance	Recommended value for f_{du}
Cadmium	0.04
Acenaphtene	0.13
Acenaphtylene	0.13
Anthracene	0.13
Benzo(a)anthracene	0.13
Benzo(a)pyrene	0.13
Benzo(b)fluoranthene	0.13
Benzo(g,h,i)perylene	0.13
Benzo(k)fluoranthene	0.13
Chrysene	0.13
Dibenzo(a,h)anthracene	0.13
Fluoranthene	0.13
Fluorene	0.13
Indeno(1,2,3-cd)pyrene	0.13
Naphthalene	0.13
Phenanthrene	0.13
Pyrene	0.13

3.3.3 Inhalation of dust

- S-EPA

In the S-EPA methodology for derivation of the generic guideline values for soil, two approaches for exposure due to inhalation of dust from the contaminated site. The first is used for substances where a toxicologically based reference air concentration (*RfC*) is available. The second is used for the other substances where an estimate is made of the daily average amount of dust that is inhaled. The exposure time on land with less sensitive land-use (MKM) is assumed to be a third of the exposure time on land with sensitive land-use (KM).

The average concentration of contaminated dust in inhaled air is estimated to be 41 µg/m³ based on data from CSOIL.

- *Reference air concentration available*

For substances where a reference air concentration is available, the reference soil concentration, C_{id} [mg/kg], for the dust inhalation pathway is calculated as:

$$C_{id} = \frac{RfC}{f_{exp} \times C_{ad}} \times 10^6$$

where

RfC: the toxicological reference concentration for non-genotoxic substances and the risk based concentration for genotoxic substances [mg/m³];

f_{exp}: the fraction of time spent on the site [-]; in the case of land with sensitive use, $f_{exp} = 1$, and for land with less sensitive land-use, $f_{exp} = 0.33$;

C_{ad}: the annual average dust concentration in inhaled air [mg/m³].

- *No reference air concentration available*

For substances where no toxicologically based reference air concentration is available, an estimate of the exposure is made according to the methodology used in CSOIL. The values used in the equation for the reference soil concentration are presented in Table 31.

Table 31: Parameters used by S-EPA for the dust inhalation exposure calculations (Naturvårdsverket, 1996b, 2005).

Parameter	Land with sensitive land-use (KM)	Land with less sensitive land-use (MKM)
Long-term inhalation exposure per unit body weight [mg/kd.d]	0.016 child 0.009 adult	0.005 child 0.003 adult
Integrated lifetime inhalation exposure [mg/kg.d]	0.01	0.003

The reference soil concentration for the dust inhalation pathway, C_{id} [mg/kg], is calculated as:

$$C_{id} = \frac{TRV}{R_{id}} \times 10^6$$

where

TRV: the toxicological reference value [mg/kg.d], i.e. *TDI* for non-genotoxic substances and risk based daily intake for genotoxic substances;

R_{id}: the average daily inhalation of dust [mg/kg.d].

- S-RISK

  - *Calculation of wind-blown soil emissions*

In general, inhalation of dust is not an important pathway, except for compounds which are more toxic by the inhalation pathway compared to the ingestion pathway (e.g. PAHs) or when high dust concentrations are present. At present, the concentration in air by dust emission is calculated from a default dust concentration and the assumption that 80 % (indoor) or 50 % (outdoor) of dust originates from the contaminated area. This estimate is highly uncertain and is revised.

Wind-blown emissions of fine dust (particle size < 10 μm) from soils is essentially caused by the bombardment of the soil surface by coarse sand particles having a size in the range 50-500 µm. At sufficiently high wind speeds, sand particles are extracted from the surface, and follow a more or less ballistic trajectory, extracting finer particles from the soil on impaction. Owing to their higher friction (relative to their mass), the fine particles remain airborne for a relatively long time.

The simulation of soil dust emission is generally done by a two-step procedure (Shao, 2000; Raupach and Lu, 2004), as follows:
- firstly, the saltation flux is calculated, i.e., the flux of sand particles following the flow (hence mainly horizontally), integrated over the vertical direction;
- subsequently, the vertical flux of fine soil dust is calculated as a function of the saltation flux, and of certain soil properties.

Since the saltation flux depends on wind speed and soil moisture in a strongly non-linear fashion, it is not feasible to employ annual average values for these quantities in the calculation of the emission fluxes. Daily and even sub-daily wind fluctuations have to be accounted for.

In Appendix E, a method is presented for the calculation of wind-blown soil dust emissions. Particular attention is given to the effect of obstacles in built-up areas (residential areas, industrial sites) on these emissions. The method relies on daily meteorological data and on site characteristics such as soil texture and obstacle density and height. Meteorological data are taken from the GLOBALSOD database (*Global Summary of the Day*, available from ftp://ftp.ncdc.noaa.gov/pub/data/globalsod/), which contains daily average observed values of wind speed, temperature, precipitation, among other meteorological parameters, for thousands of stations world-wide, for the period 1994-now.

The fluxes [μg/m².s] calculated with the methodology are given in

Table 32. The scenarios are considered worst case, with a value for z_{0g} of 10^{-5} m being rather unrealistic (desert conditions).

*Table 32: Calulated PM$_{10}$ fluxes using meteorological data from different Swedish stations, for a very flat soil between buildings located at a city edge next to a smooth plain, for different soil types (values in **bold** are used in calculations).*

z_{0g} (m)	soil	PM$_{10}$ flux [μg m^{-2} s^{-1}]							
		Skavasta/ Stockholm		Stockholm/ Bromma		Malmö/ Sturup		Göteborg/ Landvetter	
		mean	max	mean	max	mean	max	mean	max
10^{-5}	sand	6.4	1,672	-	-	3.1	1,094	0.92	333.7
	loam	15.8	4,248	-	-	7.1	2,555	3.4	1,231
	clay	389	102,024	-	-	184.4	66,010	58.3	21,120
10^{-4}	sand	0.026	8.6	-	-	0.002	0.69	0.05	17.5
	loam	**0.05**	18	-	-	**0.004**	1.4	**0.12**	43.7
	clay	1.5	501	-	-	0.11	38.9	2.9	1,060

An important remark is that these calculations only account for wind-induced emissions. Mechanical dust emission by playing, driving cars, digging, etcetera is not included but can be significant.

- *Calculation annual average concentration in air*

On the basis of the calculated PM$_{10}$ fluxes for loam soils and a z_{0g} value of 10^{-4} m, annual average concentrations of contaminated dust in inhaled air are calculated by using the same Box model as applied in the Vlier-Humaan model (Flanders) for the calculation of on-site concentrations in the gas phase. Details of the calculation are given in Appendix E.

The following concentrations of inhaled dust in outdoor air are calculated:
- Stockholm: 1.63×10^{-4} mg/m³;
- Malmö: 1.13×10^{-5} mg/m³;
- Göteborg: 4.19×10^{-4} mg/m³.

Since these calculated concentrations only consider wind-induced dust emissions originating from the site, and additional dust emissions due to activities on the site (e.g. children playing, agricultural activities,…) are not taken into account, the calculated concentrations of inhaled dust were multiplied by a safety factor of 10. Also, it is assumed that the concentration of inhaled dust in indoor air is the same as in outdoor air.

To derive an overall applicable annual average concentration of inhaled dust in the entire specified region, the calculated outdoor dust concentration in Göteborg (4.19×10^{-4} mg/m³) was multiplied with a safety factor of 10 and rounded to 5×10^{-3} mg/m³ (value used in S-RISK). At present, the average concentration of contaminated dust in inhaled air in S-EPA is estimated to be 4.1×10^{-2} mg/m³ (based on data from CSOIL).

- *Calculation of reference soil concentration for the dust inhalation pathway*

In order to reduce the two parallel equations for dust inhalation to a single equation, only *RfC* values are used. If no reference concentration or *RfC* value is available, the inhalation *RfD* is converted to an *RfC* by

$$RfC = \frac{RfD \times 70}{20} = 3.5 \times RfD$$

The remaining equation for calculation of the dust inhalation guideline value is then

$$C_{id} = \frac{RfC}{f_{\exp} \times C_{ad}} \times 10^6$$

3.3.4   Inhalation of vapours

- S-EPA

Two approaches are used for exposure due to inhalation of vapours. For substances where a toxicologically based reference air concentration is available, this concentration is compared with the estimated exposure time-corrected indoor air concentration. For the other substances an estimate is made of the daily average amount of vapour that is inhaled. The exposure time on land with less sensitive land-use (MKM) is assumed to be a third of the exposure time on land with sensitive land-use (KM).

- *Reference air concentration available*

For substances where a reference air concentration is available, the reference soil concentration for the vapour inhalation pathway, C_{iv} [mg/kg], is calculated as:

$$C_{iv} = \frac{RfC}{f_{\exp} \times H} \times \left[K_d + \frac{(\theta_w + (\theta_a \times H))}{\rho_b} \right] \times \frac{1}{DF_{ia}} \times \frac{1}{1000}$$

where

RfC: the toxicological reference concentration for non-genotoxic substances and the risk based concentration for genotoxic substances [mg/m³];

f_{exp}: the fraction of time spent on the site; in the case of land with sensitive land-use, f_{exp} = 1, and for land for land with less sensitive land-use, f_{exp} = 0.33;

K_d: the distribution coefficient soil-water [l/kg];

θ_w: the soil water content [dm³ water/dm³ soil];

θ_a: the soil air content [dm³ air/dm³ soil];

H: Henry's constant [-];

ρ_b: the soil bulk density [kg/dm³];

DF_{ia}: the dilution factor for indoor air.

- *No reference air concentration available*

For substances where no toxicologically based reference air concentration is available an estimate of the exposure is made according to the methodology used in CSOIL. This exposure is in this case expressed as exposure per unit concentration, i.e. mg of contaminant

inhaled per kg of body weight and day with a concentration of 1 g/m³. The values used in the equation for the reference soil concentration are presented in Table 33.

Table 33: Parameters used by S-EPA for the vapour inhalation exposure calculations (Naturvårdsverket, 1996b).

Parameter	Land with sensitive land-use (KM)	Land with less sensitive land-use (MKM)
Long-term inhalation exposure per unit body weight [(mg/kd.d)/(g/m³)]	500 child 285 adult	170 child 95 adult
Integrated lifetime inhalation exposure [(mg/kd.d)/(g/m³)]	300	100

The reference soil concentration for the vapour inhalation pathway, C_{iv} [mg/kg], is calculated as:

$$C_{iv} = \frac{TRV}{R_{iv} \times H} \times \left[K_d + \frac{\left(\theta_w + \left(\theta_a \times H\right)\right)}{\rho_b} \right] \times \frac{1}{DF_{ia}}$$

where

TRV: the toxicological reference value [mg/kg.d], i.e. *TDI* for non-genotoxic substances and risk based daily intake for genotoxic substances;

R_{iv}: the average daily inhalation of vapour [(mg/kg.d)/(g/m³)], i.e. long-term inhalation for non-genotoxic substances and integrated lifetime inhalation for genotoxic substances.

- S-RISK

This exposure pathway is not revised. However, to harmonize the calculation method of the reference soil concentration for the vapour inhalation pathway with the method to calculate the reference soil concentration for the dust inhalation pathway, a single equation is favoured. By analogy with the calculation method of the reference soil concentration for the dust inhalation pathway, the first approach to calculate C_{iv} is favoured, i.e. by comparing with a *RfC*.

If no reference concentration or *RfC* value is available, the inhalation *RfD* is converted to an *RfC* by:

$$RfC = \frac{RfD \times 70}{20} = 3.5 \times RfD$$

The final equation for calculation of the dust inhalation guideline value is then given by:

$$C_{iv} = \frac{RfC}{f_{exp} \times H} \times \left[K_d + \frac{\left(\theta_w + \left(\theta_a \times H\right)\right)}{\rho_b} \right] \times \frac{1}{DF_{ia}} \times \frac{1}{1000}$$

3.3.5   Intake of drinking water

- S-EPA

The reference soil concentration for the drinking water exposure pathway can be estimated either from toxicologically based drinking water guidelines, if these are available, or by estimating the exposure and comparing that with the toxicological reference value. Results from both procedures can differ. More information can be found in section 3.4.2.

- *Drinking water guideline available*

For substances where a toxicologically based drinking water guideline value is available, the reference soil concentration for the drinking water pathway, C_{iw} [mg/kg], is calculated as:

$$C_{iw} = DWG \times \left[K_d + \frac{\left(\theta_w + \left(\theta_a \times H \right) \right)}{\rho_b} \right] \times \frac{1}{DF_{gw}}$$

where

DWG: the toxicological drinking water guideline [mg/l];
K_d: the distribution coefficient soil-water [l/kg];
θ_w: the soil water content [dm³ water/dm³ soil];
θ_a: the soil air content [dm³ air/dm³ soil];
H: Henry's law constant [-];
ρ_b: the soil bulk density [kg/dm³];
DF_{gw}:   the dilution factor for well water.

- *No drinking water guideline available*

For substances where no toxicologically based drinking water guideline is available an estimate of the exposure is made according to the methodology used in CSOIL. The values used in the equation for the reference soil concentration are given inTable 34.

Table 34: Parameters used by S-EPA for the drinking water exposure calculations (Naturvårdsverket, 1996b, 2005).

Parameter	Land with sensitive land-use (KM)	Land with less sensitive land-use (MKM)
Long-term water consumption per unit body weight [l/kg.d]	0.067 child 0.029 adult	0.067 child 0.029 adult
Integrated lifetime drinking water consumption [l/kg.d]	0.03	0.03

The reference soil concentration for the drinking water pathway, $C_{iw}{}^1$ [mg/kg], is calculated as:

$$C_{iw} = \frac{TRV}{R_{iw}} \times \left[K_d + \frac{(\theta_w + (\theta_a \times H))}{\rho_b} \right] \times \frac{1}{DF_{gw}}$$

where

TRV: the toxicological reference value [mg/kg.d], i.e. *TDI* for non-genotoxic substances and risk based daily intake for genotoxic substances;

R_{iw}: the average daily water consumption [l/kg.d], i.e. long-term consumption for non-genotoxic substances and integrated lifetime consumption for genotoxic substances.

- S-RISK

For the derivation of the Swedish guideline values only the direct contamination of well water is considered. This exposure pathway is not revised. However, by analogy with the dust and vapour exposure pathways, the use of one single equation to calculate the reference soil concentration for the drinking water pathway is favoured. It is proposed to use the first approach since drinking water guidelines for Cd and PAHs are available (see Chapter 6).

3.3.6 Consumption of vegetables grown on the contaminated site

- S-EPA

Consumption of vegetables grown on the contaminated site is an exposure pathway which is only considered in the case of land with sensitive land-use (KM). The model for exposure due to consumption of vegetables is based on the model used in CSOIL and HESP.

The exposure is estimated from data provided from CSOIL and SCB (1995). The values used in the equation for the reference soil concentration is given in Table 34.

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[1] In Naturvårdsverket (1996b), the reference soil concentration for the drinking water pathway is called C_{iw} if a toxicologically based drinking water guideline is available. If no drinking water guideline is available, the reference soil concentration for this pathway is called C_{gw}. It is believed that by analogy with the names of the other reference soil concentrations for the other pathways, the same name should be used, irrespective of a drinking water guideline is available or not. Therefore, the reference soil concentration for the drinking water pathway is called C_{iw} henceforth.

Table 35: Parameters used by S-EPA for the vegetable consumption exposure calculations (Naturvårdsverket, 1996b, 2005).

Parameter	Land with sensitive land-use (KM)
Long-term consumption per unit body weight [kg/kg.d]	0.01 child 0.004 adult
Integrated lifetime consumption [kg/kg.d]	0.005

The reference soil concentration for the vegetable consumption pathway, C_{ig} [mg/kg], is calculated as:

$$C_{ig} = \frac{TRV}{R_{ig} \times f_h \times K_{pl}}$$

where

TRV: the toxicological reference value [mg/kg.d], i.e. *TDI* for non-genotoxic substances and risk based daily intake for genotoxic substances;

R_{ig}: the average daily consumption of vegetables [kg vegetables/kg bw.d], i.e. long-term consumption for non-genotoxic substances and integrated lifetime consumption for genotoxic substances;

f_h: the fraction of vegetables grown on the site [-];

K_{pl}: the plant-soil concentration ratio [(mg/kg plant)/(mg/kg soil)].

- S-RISK

Data on vegetable consumption rates in Sweden for the year 2000 have been summarised in Carlsson-Kanyama and Engström (2003). In this survey of consumption trends the following relevant data on homegrown vegetable consumption rates have been included:
- Root crops: 8.3 kg/yr fw;
- Fresh vegetables: 38.6 kg/yr fw;
- Other 'kitchen vegetables' (cooled or frozen, not from the own garden): 17.7 kg/yr fw;
- Potatoes: 44.8 kg/yr fw.

In the Netherlands a more detailed evaluation of vegetable consumption rates was performed (Swartjes et al., in progress). From this evaluation the average lifetime consumption rates as given in Table 36 result.

Table 36: The average lifetime consumption [g fresh weight per day) for above-ground vegetables and potatoes in the Netherlands (DFNCS, 1998; Swartjes et al., in progress).

Group	Time span	Above-ground vegetables	Potatoes
Babies and pre-scholars	1-6 year	58.3	59.5
adults and schoolgoing children	7-70 year	139	122

More details on the consumption rates in the Netherlands are given in Appendix A.

In Table 37 the consumption rates of the Netherlands are compared with consumption rates of Sweden (data from S-EPA as well as data from Carlsson-Kanyama and Engström, 2003). To this purpose the lifelong averaged consumption rate is calculated, with a consumption pattern for babies and preschoolers that contribute with a factor of 6/70 and for adults and schoolgoing children with a factor of 64/70 to total consumption rates.

Table 37: Comparison of lifelong averaged consumption rates.

Crop	Proposed revisions for the Netherlands (Swartjes et al., in progress) [g fw/d]	Recent data for Sweden (Carlsson-Kanyama and Engström, 2003) [kg fw/y]	[g fw/d]	Derivation Swedish guidelines (Naturvårdsverket, 1996b) [g fw/d]
Potatoes	117	44.8	123	-
Aboveground vegetables	132	38.6	106	-
Root crops	-	8.3	23	-
Other 'kitchen vegetables'	-	17.7	48	-
TOTAL	249		300	278

From Table 37 it is concluded that the lifelong averaged consumption rates are of the same order of magnitude: both Carlsson-Kanyama and Engström (2003) and S-EPA give slightly higher rates than the lifelong averaged consumption rate from the Netherlands.

Because the consumption rates in the Netherlands give more detailed information on specific crops and were evaluated in more detail, while the total rates are similar to the Swedish data, it is proposed to use the the consumption data of the Netherlands as basis for S-RISK.

Special attention is given to the relatively high total consumption rates of potatoes and aboveground vegetables by kitchen gardeners. No specific data on consumption rates for gardeners in Sweden are available. However, information is available on the different consumption rates for average individuals and gardeners from the Netherlands (Swartjes et al., in progress). The consumption amounts, based on the amounts of the general population corrected by the factors of difference for kitchen gardeners, are presented in Table 38. This difference for kitchen gardeners in relation with average individuals are 1.2 (babies and pre-scholars) and 1.8 (adults and schoolgoing children) for aboveground vegetables and 1.1 (babies, pre-scholars, adults and schoolgoing children) for potatoes (see Appendix A).

Table 38: The average lifetime consumption [g fresh weight per day] for above-ground vegetables and potatoes as corrected for kitchen gardeners.

Group	Time span	Above-ground vegetables	Potatoes
Babies and pre-scholars	0-6 year	70	65
adults and schoolgoing children	7-70 year	250	134

In Table 39 parameter values for use in S-RISK for the vegetable ingestion exposure calculations are presented, calculated on the basis of the data in Table 36 and Table 38. It can be seen from Table 39 that the calculated consumption rates on the basis of the uncorrected and corrected data are of the same order of magnitude. However, for the calculation of R_{ig} in S-RISK, the overall data are used since the data corrected for kitchen gardeners are considered to be too conservative.

*Table 39: Calculation of R_{ig} for land with sensitive land-use (parameter values used in S-RISK are given in **bold**).*

Parameter	Overall data	Data corrected for kitchen gardeners
Average consumption [kg/d]	0.1178 child 0.261 adult	0.135 child 0.384 adult
Long-term consumption per unit body weight [kg/kg.d]	**0.0079** child 0.0037 adult	0.009 child 0.0055 adult
Integrated lifetime consumption [kg/kg.d]	**0.0041**	0.0058

Since no information is available to justify the change of the value for the fraction of vegetables grown on the site (f_h), the same value as used in S-EPA is applied in S-RISK (c.q. 0.3)[1].

For the less sensitive land-use, either with or without groundwater extraction (MKM GV or MKM), no vegetable consumption is taken into account.

3.3.7   Fish consumption from nearby surface water

This exposure pathway is not revised.

Fish consumption from surface water is only considered in the case of land with sensitive land-use (KM). It is not explicitly modelled, but is covered by comparison of calculated concentrations in surface water with ambient water quality criteria. The reference soil concentration for the fish exposure pathway, C_{if} [mg/kg], is calculated as the soil concentration that is estimated to give a water concentration in a nearby surface water equivalent to the US-EPA Ambient Water Quality Criteria for fish consumption from fresh water. The following expression is used:

$$C_{if} = \frac{AWQC}{DF_{sw} \times DF_{gw}} \times \left[K_d + \frac{(\theta_w + (\theta_a \times H))}{\rho_b} \right]$$

where

AWQC: Ambient Water Quality Criteria for fish consumption from fresh water [mg/l];
DF_{sw}:   the dilution factor groundwater to surface water;

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[1] Since land with sensitive land-use includes agriculture, the fraction of vegetables grown on the site might be higher than 0.3. However, for kitchen gardeners it is assumed that the growing seison is too short to consume own-grown vegetables during the whole year.

DF$_{gw}$:   the dilution factor soil pore water to groundwater.

For contaminants for which no AWQC is available, exposure due to fish consumption has been neglected. However, exposure due to ingestion of fish was not found to be significant for any of the contaminants for which generic guideline values were derived by the S-EPA.

3.4   Risk characterization, calculation of guideline values

3.4.1   Integration of exposure from different pathways

In the S-EPA methodology, a reference soil concentration is calculated for each of the exposure pathways considered. This reference soil concentration for an exposure pathway corresponds to the level of contamination in the soil that is estimated to give an exposure equivalent to the reference dose considering only that single exposure pathway. Since the guideline value is presumed to consider simultaneous exposure through all possible exposure pathways, an integrated guideline value is determined for each type of land use.

The integrated human health value is defined as the inverse of the sum of the inverted reference soil concentrations.

For land with sensitive land-use (KM):

$$C_{KM} = \frac{1}{\frac{1}{C_{is}} + \frac{1}{C_{du}} + \frac{1}{C_{id}} + \frac{1}{C_{iv}} + \frac{1}{C_{iw}} + \frac{1}{C_{ig}} + \frac{1}{C_{if}}}$$

for land with less sensitive land-use with groundwater extraction (MKM GV):

$$C_{MKMGV} = \frac{1}{\frac{1}{C_{is}} + \frac{1}{C_{du}} + \frac{1}{C_{id}} + \frac{1}{C_{iv}} + \frac{1}{C_{iw}}}$$

and for land with less sensitive land-use (MKM):

$$C_{MKM} = \frac{1}{\frac{1}{C_{is}} + \frac{1}{C_{du}} + \frac{1}{C_{id}} + \frac{1}{C_{iv}}}$$

In S-RISK, the same methodology for the integration of exposure from the different pathways is applied.

This procedure assumes a linear relation between soil concentration and exposure. Because this is not the case for exposure due to consumption of vegetables (the *BCF* is decreasing with higher soil concentrations) the linear relation between the integrated human health values and the reference soil concentration for the vegetable consumption pathway (*Cig*) is

not valid. For this reason the final integrated human health values have to be calculated through interation.

3.4.2   Adjustments of values to correspond to tolerable daily intakes

When drinking water guidelines are set to correspond to an intake of a specified percentage of the *TDI* and in the case that exposure through that pathway is important for the specific land-use, the integrated value is adjusted upwards to obtain an integrated human health value corresponding to 100% of the *TDI*. However, because drinking water guidelines are applicable, an upward limit is set at the soil concentration that is estimated to give a water concentration in a nearby well equal to the drinking water guideline (i.e. the lowest of the integrated human health value and the value derived from the drinking water guidelines is adopted).

In S-RISK an adjustment is made for cadmium (c.q. 10%, i.e. the same as in S-EPA), motivated by the use of a drinking water guideline value corresponding to 10% of the *TDI*.

For none of the PAHs, this adjustment is made. The rationale for this is that for the non-carcinogenic PAH compounds, drinking water limits are adjusted to the water solubility (anthracene, benzo(g,h,i)perylene), and for the carcinogenic PAHs the drinking water limits *de facto* do not correspond to 10% of the *TDI*. Moreover, for the carcinogenic PAHs, the drinking water limits are sometimes set to 10% of the non-carcinogenic toxicological reference value for oral exposure (acenaphthene, fluoranthene, phenanthrene and pyrene) or are adjusted to the water solubility (benzo(b)fluoranthene, benzo(k)fluoranthene and chrysene).

3.4.3   Adjustments of values for background exposure

Since humans are also exposed from sources other than the contaminated site (primarily from food), and this (background) exposure can account for a considerably part of the *TDI*, a downward adjustment of the integrated guideline value is made for substances with a high background exposure in such a way that the sum of the background exposure and the estimated exposure from the site does nog exceed the *TDI*.

At present, for the evaluated substances, only adjustment for background exposure is made for cadmium (background exposure in percentage of *TDI*: 25%). The S-EPA report does not mention if an adjustment is made for PAHs.

The adjustment for Cd in S-RISK is the same as in S-EPA. No adjustments are made for PAHs. For carcinogenic PAHs, the assessment is done for an excess cancer risk, so no adjustment for background exposure is needed.

3.4.4 Adjustment of values for acutely toxic substances

For contaminants having such a high acute toxicity that the ingestion of relatively small amounts of soil can be harmfull, the guideline values have been adjusted to protect small children (since they are considered to experience the highest risks).

At present, in S-RISK as in S-EPA no adjustments of soil guideline values have been made for PAHs and Cd.

3.4.5 Other aspects

- Organoleptic parameters

For some contaminants, concentrations in water and air which are detectable by taste or smell have been taken into account. This was not the case for PAHs and Cd.

- Background concentrations

Background levels of contaminants in soils have been taken into account in so far as no guideline value has been set under the 90th percentile of the measured background concentration in rural environments. Information on the background levels of metals in urban and rural environments have been obtained from Andersson (1977) and Naturvårdsverket (1996c,d).

3.4.6 Integration of results

- S-EPA

The basic principle for setting the generic guideline values is to select the lowest of the human health based value and the ecotoxicological based value as soil guideline. For substances where smell and odor problems can occur at lower concentrations this has been taken into consideration. However, a less conservative perspective is put on smell and odor problems compared to toxicological problems.

No values are set below the 90th percentile for the background concentration in natural environments. No value is set below the detection limit for the appropriate analytical method.

- S-RISK

The newly derived human health based value is not integrated with the ecotoxicological based value. However, both values are compared with each other.

4   MODEL FOR ENVIRONMENTAL RISK ASSESSMENT

The S-EPA methodology for environmental risk assessment is not evaluated. It is included in this report for reasons of comprehensiveness.

In the calculation of environmental risk based guideline values, effects on both the contaminated site itself (on-site) and due to transport of contaminants from the site (off-site) have been considered for the different land-uses. For on-site effects the level of protection differs between the two types of land-use.

4.1   On-site effects

4.1.1   Land with sensitive land-use

The ecotoxicological value for on-site effects represents the level at which biodiversity is maintained at a specific level and there will be no serious disturbance of the soil's capacity to perform a range of ecological and enzymatic functions.

The ecotoxicological values used are based on the ecotoxicological Intervention Values derived in the Netherlands (Swartjes, 1999). The Dutch Intervention Values correspond to the soil concentration above which 50% of the species (biodiversity) or 50% of the ecological and enzymatic functions are protected.

Since protection of only 50% of the species and processes in the soil ecosystem was considered insufficient protection of the soil functions required for land with sensitive land-use, the ecotoxicological values for on-site effects, E_{KM} [mg/kg], used to derive the generic guideline values for contaminated soils in Sweden for land with sensitive land-use are set at half the Dutch Intervention Values.

$$E_{KM} = 0.5 \times SRC_{eco}$$

where

SRC_{eco}:   ecotoxicological Serious Risk Concentration, as part of the Intervention Value from the Netherlands [mg/kg dw].

The ecological protection level is a compromise between ecological acceptance (if 50% is protected the chance for recovery is acceptable) and practical use (the resulting contaminant concentrations in soil are high enough to avoid a huge part of the Netherlands being tagged as seriously contaminated). The extent of the adverse effects will vary among species and range from negligible to severe. An implication of this is that sensitive species are not protected at the level of the (ecotoxicological) risk limit.

4.1.2   Land with less sensitive land-use

In the case of land with less sensitive land-use (MKM), protection of the soil ecosystem is also relevant. However, the level of protection may be somewhat lower than land with sensitive land-use. An ecotoxicological value equal to the Dutch value has been adopted, which is assumed to be sufficiently protective to the species and ecological and enzymatic functions important for this land-use. In addition, this value is thought to be protective of off-site effects arising from contamination of groundwater and transport of contaminants to a discharge zone. The ecotoxicological value, E_{MKM} [mg/kg] is thus given as:

$$E_{KM} = E_{NL}$$

4.2   Off-site effects

The effects in nearby surface waters are assessed by comparing the calculated concentrations in surface waters with Canadian Water Quality criteria for the protection of freshwater aquatic life. The Canadian guidelines are set at concentrations which are protective of all forms of freshwater aquatic life and all aspects of the aquatic life cycles, and are based on the available data on the ecotoxicological value, E_{sw} [mg/kg], is given by:

$$E_{sw} = \frac{CWQC}{DF_{sw} \times DF_{gw}} \left[K_d + \frac{(\theta_w + (\theta_a \times H))}{\rho_b} \right]$$

where

CWQC: Canadian Water Quality Criteria for freshwater aquatic life [mg/l];
DF$_{sw}$:   the dilution factor groundwater to surface water [-];
DF$_{gw}$:   the dilution factor soil pore water to groundwater [-].

5 PARAMETER VALUES FOR CADMIUM AND PAHs

5.1 Cadmium

5.1.1 Physico-chemical properties and environmental behaviour

The only physico-chemical property of cadmium, relevant within the present guideline methodology, is the sorption coefficient, K_d.

The sorption of cadmium on soil is dependent on soil properties. A number of studies have derived empirical relations between K_d and soil properties. A review is given in de Meeus et al. (2002). A number of different relations are applied to a 'typical' Swedish soil, with an organic matter content of 2% (i.e. an organic carbon content of 1.16%) and a pH of 5.5 (lower end of pH range). Results are given in Table 40.

Table 40: Different empirical relations to calculate the sorption coefficient K_d for Cd from soil properties.

Algorithm	log K_d Swedish soil	Country	Reference
log K_d=-0.19+0.46pH	2.11	Belgium	Smolders et al., 2000
log K_d=-1.34+0.64pH	1.86	Belgium	Smolders et al., 1999
log K_d=-2.02+0.96log(%C)+0.6pH	1.04	Belgium	Smolders et al., 1999
log K_d =-1.35+0.,587(pH+0.4)+0.157(%OM)	2.13	Denmark	Christensen, 1989
log K_d =-0.738+0.529(pH+0.4)	2.12	Denmark	Christensen, 1989
log K_d =-1.16+0.56pH	1.64	Netherlands	Römkens and Salomons, 1998
log K_d =-1.0+0.44pH+1.03log(%OM)	1.51	Netherlands	Römkens, 2000
log K_d =-1.8+log(%OM)+0.59pH	1.45	French, Dutch and UK soils	Gerritse and Van Driel, 1984

Since the properties of a typical Swedish soil fall within the range of soil the regressions from Table 40 are developed for, but none of the regressions is made for Swedish soils in particular, all regressions are given equal weight. The average value for log K_d (or the geometric mean of the K_d values) is proposed to be used in S-RISK and corresponds to a K_d value of 102 l/kg.

As a comparison, it is refered to empirical data on K_d values for typical Dutch soils (on the basis of 46 locations; Verschoor et al., in progress). The calculated K_d of 102 l/kg fits between the 10th percentile value and the geometric mean (and 50th percentile) of Verschoor et al.

Table 41: Empirical K_d values [l/kg] for Dutch soils (Verschoor et al., in progress).

Minimum	6
Maximum	7,667
10 percentile	15
50 percentile	217
90 percentile	1,700
Geometric mean	160

5.1.2 Toxicology

In this paragraph, the most important toxicological properties of Cd are discussed. Also, recommended toxicological reference values for the oral and inhalation exposure pathway are given. An elaborated overview of the toxicology of cadmium and a rationale for the choice of the recommended reference values are given in Appendix F.

- Summary

It is generally agreed by IARC, US-EPA and the European Commission (EC) that there is no evidence of carcinogenicity by oral exposure. For the inhalation exposure pathway however, IARC classified Cd and Cd compounds in Group 1. The US-EPA (1985) classified Cd as a B2 (probable human) carcinogen. The European Commission classified cadmium chloride, cadmium oxide, and cadmium sulphate as substances which should be regarded as if they are carcinogenic to man (Group 2). Cadmium sulphide is classified as a substance which causes concern for man owing to possible carcinogenic effects, but in respect of which the available information is not adequate for making a satisfactory assessment (Group 3).

For chronic oral exposure, the kidney is considered the critical target organ for the general population as well as for occupationally exposed populations. Chronic obstructive airway disease is associated with long-term high-level occupational exposure by inhalation. There is some evidence that such exposure to cadmium may contribute to the development of cancer of the lung but observations from exposed workers have been difficult to interpret because of confounding factors (WHO, 1992a,b, 1996).

- Recommendations for S-RISK

A summary of the toxicological reference values for cadmium reported by different agencies is given in Table 42.

Table 42: Summary toxicological reference values for cadmium.

Agency	Non-carc. effects			Carc. effects (excess lifetime risk of 1/10⁵)		
	Oral [mg/kg.d]	Inhalation [mg/kg.d] ([mg/m³])	Drinking water limit [mg/l]	Oral [mg/kg.d]	Inhalation [mg/kg.d] ([mg/m³])	Drinking water limit [mg/l]
JECFA, 2001, 2003, 2004 WHO, 1989, 1993, 1998b, 2000, 2001	1×10^{-3}	8.57×10^{-5} (3×10^{-4}) (5×10^{-6}) *	3×10^{-3}	-		-
US-EPA, 1985, 2005	5×10^{-4} (water) 1×10^{-3} (food)	-	5×10^{-3}	-	1.7×10^{-6} (6×10^{-6})	-
EC, 2001a	-	1.43×10^{-6} (5×10^{-6}) #	-	-	$0.69\text{-}1.57 \times 10^{-6}$ $(2.4\text{-}5.5 \times 10^{-6})$ **	-

*: The finding of renal effects in areas contaminated by past emissions of cadmium indicates that the body burden of the general population in some parts of Europe cannot be further increased without endangering renal functions. To prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations, a guideline of 5 ng/m³ is established.

**: Derived from the range $2.4\text{-}5.5 \times 10^{-7}$ μg/m³ corresponding to an excess lifetime cancer risk of 10^{-6}.

#: Set as target value according to Directive 2004/107/EC.

- *Oral exposure*

An oral *TDI* of 1×10^{-3} mg/kg.d is selected on the basis of the *PTWI* of 7×10^{-3} mg/kg.week. This value is the same as the *RfD* (for food) reported by US-EPA. Also, JECFA has recently re-evaluated Cd and maintained this value.

The WHO-drinking water guideline value of 3×10^{-3} mg/l is recommended. It is established based on an allocation of 10% of the *PTWI* to drinking water (WHO, 1996).

- *Inhalation exposure*

The Tolerable Concentration in Air (to be considered a *RfC*) of EC (i.e. 5×10^{-6} mg/m³) is recommended for use in S-RISK. According to the Working Group of the EC, this value derived from non-cancer effects, provides also an appropriate level of protection from cancer risk due to exposure to cadmium. Also, this value is recommended by WHO to prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations.

5.2 Polycyclic aromatic hydrocarbons

5.2.1 Physico-chemical properties and environmental behaviour

Physico-chemical properties of PAHs were compiled from:
- ATSDR (2003). ATSDR's Toxicological Profiles on CD-ROM, Version 5:1. CRC Press UK, London, UK.
- EC (2000). International Uniform Chemical Information Database (IUCLID) CD-ROM, Year 2000 Ed., Public data on high volume chemicals. European

Commission, Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau, EUR 19559 EN, Ispra, Italy.
- Mackay, D., Shiu, W.Y., Ma, K.C. (1992). Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals. Volume II, Polynuclear aromatic hydrocarbons, polychlorinated dioxins and dibenzofurans. Lewis Publishers, Chelsea, Michigan, USA.
- Montgomery, J.H. (1996). Groundwater chemicals – Desk reference, 2nd Ed. CRC Press, Inc., Boca Raton, Florida, USA.
- Verschueren, K. (1996). Handbook of environmental data on organic chemicals, 3rd Ed. Van Nostrand Reinhold, New York, USA.

Only data reported at 298K were collected. Then, for each physico-chemical property, the geometric mean was calculated when the range of data spanned more than one order of magnitude, as this was considered to be the most likely value. If the data ranged over less than one order of magnitude, the arithmetic mean was taken as the most likely value. If the number of data for the organic carbon partition coefficient was less than one third of the number of data for the octanol-water partition coefficient, then the organic carbon partition coefficient was calculated according to the Karickhoff-equation (1981), else the geometric or arithmetic mean was calculated as described above. The octanol-air partition coefficient (K_{OA}) was calculated as $K_{OA}=K_{OW}/H$.

Table 43 gives a summary of the reviewed data.

5.2.2 Toxicology

In this paragraph, the most important toxicological properties of PAHs are discussed. Also, recommended toxicological reference values for the oral and inhalation exposure pathway are given. An elaborated overview of the toxicology of PAHs and a rationale for the choice of the recommended reference values are given in Appendix G.

Table 43: Physico-chemical properties of PAHs (reviewed data at 298K).

Chemical	MW^* [g/mol]	S [mg/l]		P^* [Pa]	H [-]		K_{OW} [l/kg]		K_{OC} [l/kg]		K_{OA} [l/kg]
		S-EPA	Reviewed	Reviewed	S-EPA	Reviewed	S-EPA	Reviewed	S-EPA	Reviewed	$
Acenaphthene	154.21 a	*	3.59 g	4.21×10^{-1} g	*	7.49×10^{-3} a	*	$10^{4.05}$ g	*	$10^{3.55}$ g	1.50×10^{6}
Acenaphthylene	152.20 a	3.9	6.71 a	9.45×10^{-1} a	6.1×10^{-2}	8.84×10^{-3} g	$10^{3.74}$	$10^{3.94}$ a	$10^{3.35}$	$10^{3.23}$ g	9.85×10^{5}
Anthracene	178.23 a	*	6.81×10^{-2} g	2.32×10^{-3} g	*	5.67×10^{-3} g	*	$10^{4.44}$ g	*	$10^{4.34}$ g	4.86×10^{6}
Benzo(a)anthracene	228.22 a	9.4×10^{-3}	1.59×10^{-2} g	1.68×10^{-5} g	1.37×10^{-4}	1.83×10^{-4} g	$10^{5.70}$	$10^{5.83}$ g	$10^{5.60}$	$10^{5.24}$ g	3.69×10^{9}
Benzo(a)pyrene	252.56 a	1.6×10^{-1}	3.23×10^{-3} g	1.09×10^{-6} g	4.6×10^{-5}	2.60×10^{-4} g	$10^{6.11}$	$10^{6.27}$ g	$10^{6.01}$	$10^{5.88}$ c	7.16×10^{9}
Benzo(b)fluoranthene	252.24 a	1.5×10^{-3}	2.48×10^{-3} g	8.91×10^{-6} g	4.55×10^{-3}	9.66×10^{-4} a	$10^{5.20}$	$10^{6.32}$ a	$10^{5.09}$	$10^{5.93}$ c	2.16×10^{9}
Benzo(g,h,i)perylene	276.34 a	2.6×10^{-4}	4.00×10^{-4} g	2.99×10^{-8} g	5.80×10^{-6}	2.17×10^{-5} g	$10^{7.23}$	$10^{6.91}$ g	$10^{6.85}$	$10^{6.52}$ c	3.75×10^{11}
Benzo(k)fluoranthene	252.24 a	8.0×10^{-4}	1.22×10^{-3} g	3.24×10^{-7} g	3.40×10^{-5}	2.30×10^{-3} a	$10^{5.20}$	$10^{6.55}$ g	$10^{5.09}$	$10^{5.82}$ g	1.54×10^{9}
Chrysene	228.28 a	1.6×10^{-3}	2.78×10^{-3} g	1.96×10^{-6} g	3.88×10^{-3}	8.82×10^{-4} g	$10^{5.70}$	$10^{5.78}$ g	$10^{5.60}$	$10^{5.12}$ g	6.83×10^{8}
Dibenzo(a,h)anthracene	278.36 a	2.5×10^{-3}	7.73×10^{-4} g	1.27×10^{-9} g	6.03×10^{-7}	8.72×10^{-6} g	$10^{6.70}$	$10^{6.54}$ g	$10^{6.58}$	$10^{5.95}$ g	3.98×10^{11}
Fluoranthene	202.20 a	2.1×10^{-1}	1.95×10^{-1} g	4.48×10^{-3} g	6.60×10^{-4}	9.30×10^{-4} g	$10^{5.12}$	$10^{5.19}$ g	$10^{5.03}$	$10^{4.97}$ g	1.67×10^{8}
Fluorene	166.22 a	2.0	2.03 g	2.29×10^{-1} g	2.6×10^{-3}	4.65×10^{-3} a	$10^{4.21}$	$10^{4.19}$ a	$10^{4.14}$	$10^{4.15}$ g	3.33×10^{6}
Indeno(1,2,3-cd)pyrene	276.33 a	2.2×10^{-5}	6.20×10^{-2} a	1.35×10^{-8} a	6.56×10^{-5}	8.54×10^{-6} a	$10^{6.65}$	$10^{6.28}$ a	$10^{6.54}$	$10^{7.09}$ g	2.23×10^{11}
Naphthalene	128.18 a	3.1×10^{1}	3.10×10^{1} g	1.27×10^{1} g	2.0×10^{-2}	2.12×10^{-2} a	$10^{3.60}$	$10^{3.38}$ g	$10^{3.30}$	$10^{3.17}$ g	1.20×10^{5}
Phenantrene	178.23 a	1.3	9.03×10^{-1} g	3.99×10^{-2} g	6.2×10^{-3}	1.66×10^{-3} g	$10^{4.46}$	$10^{4.50}$ g	$10^{4.07}$	$10^{4.15}$ g	1.90×10^{7}
Pyrene	202.27 a	1.4×10^{-1}	1.52×10^{-1} g	1.11×10^{-3} g	4.51×10^{-4}	9.51×10^{-4} g	$10^{5.11}$	$10^{5.05}$ g	$10^{5.02}$	$10^{4.78}$ g	1.18×10^{8}

*: No data are given in Naturvårdsverket (1996b).

a: arithmetic mean; g: geometric mean; c: calculated as: $K_{OC} = 0.411 \times K_{OW}$.

$: calculated as: $K_{OA} = K_{OW}/H$.

- Summary

   - *Carcinogenicity and genotoxicity*

Extensive mechanistic studies have shown that many PAH compounds are complete carcinogens, i.e. they can both induce cancer by producing mutations in DNA and promote cancer by affecting the proliferative capacity of affected cells (e.g. Baird et al., 2005).

Cancer associated with exposure to PAH-containing mixtures in humans occurs predominantly in the lung and skin following inhalation and dermal exposure, respectively. However, skin tumours have become rare because of better personal hygiene (WHO, 1998a). There are no data available for humans for the oral route. The extrapolation of risk to humans from animal data is complicated: the relevance of forestomach tumours in rodents when considering extrapolation to humans is not clear. However, it is anticipated that PAHs are carcinogenic by the oral pathway as well (WHO, 1998b).

On the basis of data derived from WHO/IARC and US-EPA/IRIS, the following contaminants are considered carcinogenic to humans: benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene. Fluoranthene is suspected of human carcinogenicity (WHO, 1998). However, according to the IARC and US-EPA, this compound is not classifiable as to human carcinogenicity. Anthracene, fluorene and naphthalene can be considered non-genotoxic compounds. With the exception of naphthalene, for which carcinogenicity is questionable due to the small database, these compounds are also considerd as non-carcinogenic by WHO (1998a). This is in agreement with the conclusion of US-EPA who classifies naphthalene as a possible human carcinogen. WHO classifies benzo(g,h,i)perylene also as a non-carcinogenic PAH; according to US-EPA, this compound is not classifiable as to human carcinogenicity; IARC did not provide a carcinogenicity assessment. It is presently unclear if acenaphthene, acenaphtylene, phenanthrene and pyrene are human carcinogens or not.

   - *Comparative cancer potency*

Most of the toxicity data for environmental chemicals are available for individual components. Hence, risks are calculated for individual compounds. Because PAHs occur in the environment as complex mixtures of varying composition, there is a need to develop reliable estimates of toxicity for these chemicals as a group. Developing such estimates requires using toxicity data derived from experiments with the mixture of interest. However, PAHs are handicapped by the lack of mixture-specific toxicity data and thus necessitate the use of approximations to predict toxicity (Ramesh et al., 2004; Reeves et al., 2001). The WHO-monograph on PAHs (WHO, 1998a) and the Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures (US-EPA, 2000a) describe three approaches commonly used to asses dose-response relationships for PAHs as a mixture: (i) use of toxic equivalence factors (TEFs), (ii) the comparative potency approach, and (iii) the benzo(a)pyrene surrogate approach. In the WHO-monograph, main advantages and disadvantages for each approach are given but no definite recommendation is given.

Use of TEF schemes in risk assessments is emerging. However, additivity of toxic effects (in this case carcinogenesis) of different PAH compounds, one of the assumptions of the TEF approach, is still debated. Studies suggest that in general, the carcinogenicity of a known PAH mixture (as a whole) is in good agreement with the sum of the respective potencies of its components, i.e. that additivity can be assumed (e.g. McClure & Schoeny, 1995), and that use of a TEF scheme would not alter significantly the outcome of risk assessments. Nevertheless, one of the major objections against use of TEFs is that complex environmental mixtures differ from defined synthetic mixtures in that they contain not only PAHs of known carcinogenicity but also hundreds of PAHs and other potentially carcinogenic non-PAH compounds for which carcinogenicity has not been established. In this way, the overall carcinogenic risk of the mixture as a whole will be underestimated. Including other (especially highly potent) PAHs in the TEF scheme could considerably enhance the outcome of the risk assessment. The use of a TEF approach for PAHs in food is contested by the (European) Scientific Committee on Food (SCF, 2002). On the basis of the published literature SCF concludes that using TEF factors underestimates the carcinogenicity of a PAH mixture. They conclude that the PAH profile in food and in coal tars is comparable and that, based on published studies, the carcinogenic potency of a mixture can be up to 5 times the carcinogenic potency of benzo(a)pyrene in the mixture. For that reason they recommend to use the oral slope factor of benzo(a)pyrene multiplied by a safe factor of 10 to estimate the carcinogenic potency of PAHs in food.

- **Recommendations for S-RISK**

For the development of generic guideline values for the PAH compounds under consideration, two questions must be addressed: (i) are the respective PAHs threshold compounds, and (ii) is application of a TEF scheme appropriate. In the proposed methodology, these two issues are handled together on the basis of consensus of opinion.

- *Threshold versus no threshold*

Genotoxic carcinogenic PAHs are considered to have no threshold for (these) effects. IARC (1983, 1987, 2005), US-EPA (1993) and WHO (1998, 2005) provide toxicological evaluations on the carcinogenic and genotoxic properties of the PAHs considered. These evaluations are not always conclusive. Also, there were not always enough data available to assess the genotoxicity and carcinogenicity. In this case, the compound can be put into S-RISK as a compound with either threshold or no-treshold for effects.

- *TEF-scheme*

Although use of TEF schemes is debated, application of TEF values provides a relatively simple risk assessment approach. Considering the different methods and applied studies employed for the derivation of the different TEF schemes, TEFs should best be expressed as order of magnitude. The proposed TEFs are derived taking into account the consistency of the different TEF schemes for each PAH compound. It must be emphasized that the evaluations of WHO and US-EPA/IRIS are of great importance in the overall derivation.

Although the use of the benzo(a)pyrene surrogate method could give a better (i.e. with less chance of underestimating) estimate of carcinogenic potency of a PAH mixture, the Swedish guideline method is directed towards individual PAHs (probably those for which it is expected that they contribute most to the carcienogenic potency of a PAH mixture). Changing the individual compounds approach towards a benzo(a)pyrene surrogate approach would have significant consequences for the guideline system and its application. As it was not the aim of the project, nor is it feasible within this context, to explore the impact of such a decision, it is found reasonable and scientifically defensible to use the TEF approach (including additivity assumptions) as a best estimate within the actual framework.

- *Overall evaluation*

On the basis of classification of the considered PAH compounds as to their genotoxic and carcinogenic properties, and the proposed TEF scheme, the final suggested toxicological input scenarios and reference values for PAHs are presented in Table 44 and Table 45 respectively.

Table 44: Overview TEF values and input scenarios S-RISK and comparison with values used in Flanders and the Netherlands.

PAH	S-RISK		Vlier-Humaan – Flanders (Nouwen et al., 2001)		CSOIL – the Netherlands (Baars et al., 2001)	
	Proposed TEF	**Proposed input scenario**	**TEF**	**Input scenario**	**TEF**	**Input scenario**
Acenaphthene	0.001	NT	0.001	NT	0.001	NT
Acenaphthylene	0.01	NT	0.01	NT	0.01	NT
Anthracene	0	T	0	T	0	T
Benzo(a)anthracene	0.1	NT	0.1	NT	0.1	NT
Benzo(a)pyrene	1.0	NT	1.0	NT	1.0	NT
Benzo(b)fluoranthene	0.1	NT	0.1	NT	0.1	NT
Benzo(g,h,i)perylene	0	T	0	T	0	T
Benzo(k)fluoranthene	0.1	NT	0.1	NT	0.1	NT
Chrysene	0.01	NT	0.01	NT	0.01	NT
Dibenzo(a,h)anthracene	1.0	NT	1.0	NT	1.0	NT
Fluoranthene	0.01	NT	0.01	NT	0.01	NT
Fluorene	0	T	0	T	0	T
Indeno(1,2,3-cd)pyrene	0.1	NT	0.1	NT	0.1	NT
Naphthalene	0	T	0	T	0	T
Phenanthrene	0.001	NT	0.001	NT	0 *	T
Pyrene	0.001	NT	0.001	NT	0.001	NT

NT: no threshold for effects (genotoxic and carcinogenic compound);
T: threshold for effects;
*: phenanthrene is considered to be carcinogenic but its carcinogenic potency is extremely low (<0.001) and therefore a *TDI*-approach is applied (Baars et al., 2001).

Table 45: Toxicological values used in S-RISK and comparison with Vlier-Humaan (Flanders) and CSOIL (the Netherlands).

PAH	S-RISK						Vlier-Humaan – Flanders (Nouwen et al., 2001)						CSOIL – the Netherlands (Baars et al., 2001)	
	Non-carcinogenic effects			Carcinogenic effects (1/10^5) @			Non-carcinogenic effects			Carcinogenic effects (1/10^5) @			Non-carcinogenic effects	Carcinogenic effects (1/10^5) #
	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Oral [mg/kg.d]
Acenaphthene	-	-	-	2.2x10^{-2}	3.4x10^{-5} (1.2x10^{-4} mg/m³)	1.8x10^{-1} £	-	-	-	2.2x10^{-2}	3.3x10^{-5} (1.2x10^{-4} mg/m³)	1.8x10^{-1} £	-	5x10^{-2}
Acenaphthylene	-	-	-	2.2x10^{-3}	3.4x10^{-6} (1.2x10^{-5} mg/m³)	7x10^{-2}	-	-	-	2.2x10^{-3}	3.3x10^{-6} (1.2x10^{-5} mg/m³)	7x10^{-2}	-	5x10^{-3}
Anthracene	3x10^{-1}	3x10^{-1} $ (1.1 mg/m³)	**6.81x10^{-2}**	-	-	-	3x10^{-1}	3x10^{-1} $ (1.1 mg/m³)	**7.5x10^{-2}**	-	-	-	4x10^{-2}	-
Benzo(a)anthracene	-	-	-	2.2x10^{-4}	3.4x10^{-7} (1.2x10^{-6} mg/m³)	7x10^{-3}	-	-	-	2.2x10^{-4}	3.3.10^{-7} (1.2x10^{-6} mg/m³)	7x10^{-3}	-	5x10^{-4}
Benzo(a)pyrene	-	-	-	2.2x10^{-5}	3.4x10^{-8} (1.2x10^{-7} mg/m³)	7x10^{-4}	-	-	-	2.2x10^{-5}	3.3x10^{-8} (1.2x10^{-7} mg/m³)	7x10^{-4}	-	5x10^{-5}
Benzo(b)fluoranthene	-	-	-	2.2x10^{-4}	3.4x10^{-7} (1.2x10^{-6} mg/m³)	**2.48x10^{-3}**	-	-	-	2.2x10^{-4}	3.3x10^{-7} (1.2x10^{-6} mg/m³)	**1.2x10^{-3}**	-	5x10^{-4}
Benzo(g,h,i)perylene	3x10^{-2}	3x10^{-2} $ (1.1x10^{-1} mg/m³)	**4x10^{-4}**	-	-	-	3x10^{-2}	3x10^{-2} $ (1.1x10^{-1} mg/m³)	**2.6x10^{-4}**	-	-	-	3x10^{-2}	-
Benzo(k)fluoranthene	-	-	-	2.2x10^{-4}	3.4x10^{-7} (1.2x10^{-6} mg/m³)	**1.22x10^{-3}**	-	-	-	2.2x10^{-4}	3.3x10^{-7} (1.2x10^{-6} mg/m³)	**7.6x10^{-4}**	-	5x10^{-4}
Chrysene	-	-	-	2.2x10^{-3}	3.4x10^{-6} (1.2x10^{-5} mg/m³)	**2.78x10^{-3}**	-	-	-	2.2x10^{-3}	3.3x10^{-6} (1.2x10^{-5} mg/m³)	**1.5x10^{-3}**	-	5x10^{-3}
Dibenzo(a,h)anthracene	-	-	-	2.2x10^{-5}	3.4x10^{-8} (1.2x10^{-7} mg/m³)	7x10^{-4}	-	-	-	2.2x10^{-5}	3.3x10^{-8} (1.2x10^{-7} mg/m³)	**5x10^{-4}**	-	5x10^{-5}
Fluoranthene	-	-	-	2.2x10^{-3}	3.4x10^{-6} (1.2x10^{-5} mg/m³)	4x10^{-3} £	-	-	-	2.2x10^{-3}	3.3x10^{-6} (1.2x10^{-5} mg/m³)	4x10^{-3} £	-	5x10^{-3}
Fluorene	4x10^{-2}	4x10^{-2} $ (1.4x10^{-1} mg/m³)	1.2x10^{-1}	-	-	-	4x10^{-2}	4x10^{-2} $ (1.4x10^{-1} mg/m³)	1.2x10^{-1}	-	-	-	4x10^{-2}	-
Indeno(1,2,3-cd)pyrene	-	-	-	2.2x10^{-4}	3.4x10^{-7} (1.2x10^{-6} mg/m³)	7x10^{-3}	-	-	-	2.2x10^{-4}	3.3x10^{-7} (1.2x10^{-6} mg/m³)	**1.10^{-4}**	-	5x10^{-4}
Naphthalene	2x10^{-2}	8.6x10^{-4} (3x10^{-3} mg/m³)	6x10^{-2}	-	-	-	2x10^{-2}	8.6x10^{-4} (3x10^{-3} mg/m³)	6x10^{-2}	-	-	-	4x10^{-2}	-
Phenanthrene	-	-	-	2.2x10^{-2}	3.4x10^{-5} (1.2x10^{-4} mg/m³)	1.2x10^{-1} £	-	-	-	2.2x10^{-2}	3.3x10^{-5} (1.2x10^{-4} mg/m³)	1.2x10^{-1} £	4x10^{-2} &	-
Pyrene	-	-	-	2.2x10^{-2}	3.4x10^{-5} (1.2x10^{-4} mg/m³)	9x10^{-2} £	-	-	-	2.2x10^{-2}	3.3x10^{-5} (1.2x10^{-4} mg/m³)	9x10^{-2} £	-	5x10^{-2}

@: calculated for the slope factor and an accepted excess lifetime cancer risk of 1/10^5.

$: The same as the toxicological reference value for oral exposure.

*: Calculated on the basis of the toxicological reference value for oral exposure, assuming a 2 liter drinking water consumption per day for a person weighing 60 kg; for non-carcinogenic PAHs, the drinking water limits corresponds to 10% of the *TDI*; for the carcinogenic PAHs, the drinking water limit corresponds to 100% of the toxicological reference value.

£: Calculated on the basis of 10% of the non-carcinogenic toxicological reference value for oral exposure, assuming a 2 liter drinking water consumption per day for a person weighing 60 kg.

( ): The maximal concentration in air and the inhalation dose are linked by taking into account a body weight of 70 kg and a daily consumption of 20 m³ air/day.

6.81x10^{-2}: Drinking water limits in bold and italic format are adjusted to the water solubility (since the calculated drinking water limit exceeds the water solubility).

#: In the Netherlands, the cancer risk estimate is expressed as an excess lifetime cancer risk of 1/10^4. The toxicological values for the carcinogenic compounds were recalculated to a lifetime excess cancer risk of 1/10^5.

&: Phenanthrene is considered to be carcinogenic but its carcinogenic potency is extremely low (<0.001) and therefore a *TDI*-approach is applied (Baars et al., 2001).

With the introduction of TEFs, some PAH compounds considered non-carcinogenic (i.e. with threshold) in the S-EPA methodology, have to be dealt with as carcinogenic (no threshold) compounds in S-RISK. Differences between S-EPA and S-RISK are summarized in Table 46.

Table 46: Compounds considered carcinogenic/non-carcinogenic in S-EPA and S-RISK (input scenario).

Input scenario	S-EPA	S-RISK
Carcinogenic (no threshold) PAHs	benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene,	acenaphthene, acenaphthylene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, fluoranthene, indeno(1,2,3-cd)pyrene, phenanthrene, pyrene
Non-carcinogenic (threshold) PAHs	acenaphthene, acenaphthylene, anthracene, benzo(g,h,i)perylene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene	anthracene, benzo(g,h,i)perylene, fluorene, naphthalene

- *Oral exposure, carcinogenic effects*

The toxicological values corresponding with an excess cancer risk of 10^{-5} for (only) benzo(a)pyrene, derived by WHO and US-EPA are 2.2×10^{-5} mg/kg.d and 1.4×10^{-6} mg/kg.d respectively. The WHO value is preferred. When using the TEF scheme as proposed above, toxicological reference values can be calculated for each carcinogenic or supposed carcinogenic PAH compound (Table 45).

- *Oral exposure, non-carcinogenic effects*

For non-carcinogenic effects, *RfDs* of US-EPA can be used. For those compounds for which no toxicological reference value is given, the use of *RfDs*, derived by the TPH Criteria Working Group is recommended. This is the case for benzo(g,h,i)perylene.

- *Drinking water*

A reference drinking water concentration can be calculated, using a (calculated) toxicological reference concentration for oral exposure. According to the WHO methodology, a 2 liter drinking water consumption for a person weighing 60 kg is assumed. For non-carcinogenic effects, 10% of the toxicological reference dose is the basis for the drinking water limit. For carcinogenic effects, the excess lifetime risk of $1/10^5$ is completely assigned to drinking water.

In case a calculated drinking water limit exceeds the water solubility, an additional adjustment is made. In case the drinking water limit corresponding to carcinogenic effects exceeds the drinking water limit for non-carcinogenic effects, the latter is used in the calculations.

- *Inhalation exposure, carcinogenic effects*

The basis of the toxicological reference values is the unit risk of 8.7×10^{-5} per ng/m³ derived by WHO (1987, 2000) and accepted by the EC Working Group on Polycyclic Aromatic Hydrocarbons. The corresponding concentration of benzo(a)pyrene producing an excess lifetime cancer risk of 1/100,000 is 0.12 ng/m³, the inhalation dose is 3.4×10^{-8} mg/kg.d. When using the TEF scheme as proposed above, toxicological reference values can be calculated for each PAH compound.

- *Inhalation exposure, non-carcinogenic effects*

For non-carcinogenic effects, *RfCs* of US-EPA can be used. If no *RfC* is available, a toxicological reference dose for inhalation exposure and a *RfC* can be calculated on the basis of the (oral) *RfD*. For those compounds for which no toxicological reference value is given, the use of *RfCs*, derived by the TPH Criteria Working Group is recommended. This is the case for benzo(g,h,i)perylene.

6   CALCULATION OF GUIDELINE VALUES IN S-RISK

The S-RISK model is incorporated in an Excel environment as a spreadsheet. It is referred to as S-RISK Excel. Reference soil concentrations are calculated by automatic input of the physico-chemical properties of the compound of interest and by loading a model. Compound specific properties can be chosen from the S-EPA or S-RISK databases. Both S-EPA and S-RISK models can be loaded. For most compounds, it is also possible to calculate reference soil concentrations by combining a model applied in one methodology (S-EPA or S-RISK) and input of compound specific properties from a database from another methodology. If a combination is incompatible, the user of S-RISK Excel is notified that the calculated reference soil concentrations are invalid.

A comparison of the default input parameter values used in S-RISK and S-EPA, as well as a summary of the physico-chemical properties for Cd and PAHs in the S-RISK database are given in Appendix H.

6.1   Calculated guideline values

Reference soil concentrations for the different pathways (C_{is}: soil ingestion, C_{du}: dermal pathway, C_{id}: dust inhalation, C_{iv}: vapour inhalation, C_{iw}: intake drinking water, C_{ig}: vegetable consumption, C_{if}: fish consumption), integrated human toxicological values (C_{KM}: land with sensitive land-use, $C_{MKM\ GV}$: land with less sensitive land-use and groundwater extraction, C_{MKM}: land with sensitive land-use), and ecotoxicological values for on-site effects (E_{KM}: land with sensitive land-use, E_{MKM}: land with less sensitive land-use), calculated with the S-RISK model and the S-RISK database are given in Table 47.
For Cd, calculations are made for silty sand. For PAHs, guideline values have been calculated for six scenarios: three soil types (medium till fine sand, silty sand, and clayey loam) and two building types (concrete floor and concrete basement, i.e. to account differences in DF_{ia}).

6.1.1   Cadmium

- *Calculation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM}*

For cadmium, limiting conditions in the calculation of the human toxicological integrated values are (i) adjustment of values to correspond to tolerable daily intakes (drinking water guideline value corresponds to 10% of the *TDI*) and (ii) adjustment of values for background exposure (background exposure in percentage of *TDI*: 25%). These limiting conditions in S-RISK are the same in as in the S-EPA methodology. The first limiting condition is not applicable since the integrated value C_{KM} is lower than C_{iw} (3.3 versus 4.6). On the other hand, reference soil concentrations are adjusted to take into account background exposure.

Table 47: Calculated reference soil concentrations for different pathways and integrated values in S-RISK (concentrations in mg/kg dw)

Scenario	Substance	KM									MKM							
		C_{is}	C_{du}	C_{id}	C_{iv}	C_{tw}	C_{ig}	C_{if}	C_{KM}	E_{KM}	C_{is}	C_{du}	C_{id}	C_{iv}	C_{tw}	$C_{MKM\,GV}$	C_{MKM}	E_{MKM}
Silty sand	Cadmium	143 107 A	1,250 938 A	1,000 1,000 A	C_a=0	4.6 4.6 A	14 10 A	AWQC	3.3 3.1 A	6	3,333 2,500 A	3,571 2,679 A	3,000 3,000 A	C_a=0	9.2 9.2 A	9.1 9.1 A	1,095 904 A	12
Medium till fine sand Concrete floor	Acenaphthene[c]	16,923	56,410	24,000	0.40	96	42	AWQC	0.39	-	220,000	169,231	72,000	1.2	192	1.2	1.2	-
	Acenaphthylene[c]	1,692	5,641	2,400	0.02	18	4.3	32	0.02	-	22,000	16,923	7,200	0.05	36	0.05	0.05	-
	Benzo(a)anthr.[c]	169	564	240	8	182	72	3,232	6.4	20	2,200	1,692	720	24	365	21	23	40
	Benzo(a)pyr.[c]	17	56	24	2.5	80	8.9	14,110	1.5	20	220	169	72	7.4	159	6.0	6.2	40
	Benzo(b)fl.[c]	169	564	240	7.4	317	149	15,831	6.4	-	2,200	1,692	720	22	633	20	21	-
	Benzo(k)fl.[c]	169	564	240	2.4	121	89	12,289	2.2	20	2,200	1,692	720	7.2	242	6.9	7.1	40
	Chrysene[c]	1,692	5,641	2,400	13	55	894	2,452	10	20	22,000	16,923	7,200	38	110	28	37	40
	Dibenzo(a,h)anth.[c]	17	56	24	86	94	179	16,577	6.8	-	220	169	72	258	187	28	35	-
	Fluoranthene[c]	1,692	5,641	2,400	8.4	56	447	NL	7.2	20	22,000	16,923	7,200	25	112	21	25	40
	Ind.(1,2,3-cd)pyr.[c]	169	564	240	12,101	12,918	5,962	228,830	82	20	2,200	1,692	720	36,303	25,836	400	406	40
	Phenanthrene[c]	16,923	56,410	24,000	7.2	254	2,981	263	6.8	20	220,000	169,231	72,000	21	509	21	21	40
	Pyrene[c]	16,923	56,410	24,000	53	814	4,472	1,121	47	-	220,000	169,231	72,000	160	1,627	145	159	-
	Anthracene	42,857	115,385	NL	29,715	224	140,647	AWQC	220	20	1,000,000	329,670	NL	89,145	447	444	61,535	40
	Benzo(g,h,i)per.	4,286	11,538	NL	NL	199	6,329	61,590	181	20	100,000	32,967	NL	NL	397	391	23,622	40
	Fluorene	5,714	15,385	NL	2,978	254	8,439	263	119	-	133,333	43,956	NL	8,933	509	474	6,984	-
	Naphthalene	2,857	7,692	600,000	1.6	13	16	AWQC	1.3	20	66,667	21,978	NL	4.7	27	4.0	4.7	40
Silty sand Concrete floor	Acenaphthene[c]	16,923	56,410	24,000	9.1	96	42	AWQC	7.0	-	220,000	169,231	72,000	27	192	24	27	-
	Acenaphthylene[c]	1,692	5,641	2,400	0.37	18	4.3	32	0.33	-	22,000	16,923	7,200	1.1	36	1.1	1.1	-
	Benzo(a)anthr.[c]	169	564	240	182	182	72	3,232	27	20	2,200	1,692	720	547	365	143	235	40
	Benzo(a)pyr.[c]	17	56	24	56	80	8.9	14,110	3.8	20	220	169	72	168	159	27	33	40
	Benzo(b)fl.[c]	169	564	240	169	317	149	15,831	36	-	2,200	1,692	720	508	633	167	227	-
	Benzo(k)fl.[c]	169	564	240	55	121	89	12,289	20	20	2,200	1,692	720	165	242	79	118	40
	Chrysene[c]	1,692	5,641	2,400	287	55	894	2,452	41	20	22,000	16,923	7,200	861	110	95	712	40
	Dibenzo(a,h)anth.[c]	17	56	24	1,692	94	179	16,577	7.4	-	220	169	72	5,887	187	33	41	-
	Fluoranthene[c]	1,692	5,641	2,400	193	56	447	NL	38	20	22,000	16,923	7,200	578	112	92	507	40
	Ind.(1,2,3-cd)pyr.[c]	169	564	240	276,595	12,918	5,962	228,830	83	20	2,200	1,692	720	829,784	25,836	404	411	40
	Phenanthrene[c]	16,923	56,410	24,000	163	254	2,981	263	70	20	220,000	169,231	72,000	490	509	248	485	40
	Pyrene[c]	16,923	56,410	24,000	1,217	814	4,472	1,121	304	-	220,000	169,231	72,000	3,650	1,627	1,095	3,352	-
	Anthracene	42,857	115,385	NL	679,318	224	140,647	AWQC	221	20	1,000,000	329,670	NL	NL	447	446	165,746	40
	Benzo(g,h,i)per.	4,286	11,538	NL	NL	199	6,329	61,590	181	20	100,000	32,967	NL	NL	397	391	23,622	40

Fluorene	5,714	15,385	NL	68,079	254	8,439	263	123	-	133,333	43,956	NL	204,238	509	500	27,665	-
Naphthalene	2,857	7,692	600,000	36	13	16	AWQC	6.0	20	66,667	21,978	NL	107	27	21	106	40

Reference soil concentrations: C_{is}: soil ingestion; C_{du}: dermal pathway; C_{id}: dust inhalation; C_{iv}: vapour inhalation; C_{iw}: intake drinking water; C_{ig}: vegetable consumption; C_{if}: fish consumption. Integrated human toxicological values: C_{KM}: land with sensitive land-use; $C_{MKM\ GV}$: land with less sensitive land-use and groundwater extraction; C_{MKM}: land with sensitive land-use). Ecotoxicological values for on-site effects: E_{KM}: land with sensitive land-use; E_{MKM}: land with less sensitive land-use. NL: not limited (> 1,000,000 mg/kg ds); ~~AWQC~~: no ambient water quality criteria for fish consumption available; A: value adjusted for background exposure; c: PAH considered carcinogenic

Dominant pathways in the derivation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM} are indicated by different cell fill patterns:

M	$C_{MKM\ GV}$	C_{MKM}	$C_{MKM\ GV}$ and C_{MKM}

Since additivity is assumed for the carcinogenic PAHs (marked c), the following rule applies for evaluation of soil contamination: if $\sum_i \dfrac{concentration\ PAH_i\ in\ soil}{C(M)KM\ (GV)_i} \le 1$ then there is no excess lifetime cancer risk of $1/10^5$.

Table 47: Calculated reference soil concentrations for different pathways and integrated values in S-RISK (concentrations in mg/kg dw

Scenario	Substance	KM									MKM							
		C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	C_{ig}	C_{if}	C_{KM}	E_{KM}	C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	$C_{MKM\ GV}$	C_{MKM}	E_{MKM}
Clayey loam	Acenaphthene[c]	16,923	56,410	24,000	30	96	42	AWQC	15	-	220,000	169,231	72,000	89	192	61	89	-
loam	Acenaphthylene[c]	1,692	5,641	2,400	1.2	18	4.3	32	0.87	-	22,000	16,923	7,200	3.6	36	3.3	3.6	-
	Benzo(a)anthr.[c]	169	564	240	593	182	72	3,232	30	20	2,200	1,692	720	1,778	365	174	334	40
Concrete floor	Benzo(a)pyr.[c]	17	56	24	182	80	8.9	14,110	4.0	20	220	169	72	546	159	31	38	40
	Benzo(b)fl.[c]	169	564	240	550	317	149	15,831	42	-	2,200	1,692	720	1,649	633	216	329	-
	Benzo(k)fl.[c]	169	564	240	179	121	89	12,289	27	20	2,200	1,692	720	538	242	119	233	40
	Chrysene[c]	1,692	5,641	2,400	933	55	894	2,452	46	-	22,000	16,923	7,200	2,798	110	103	1,664	40
	Dibenzo(a,h)anth.[c]	17	56	24	6,378	94	179	16,577	7.4	-	220	169	72	19,134	187	34	41	-
	Fluoranthene[c]	1,692	5,641	2,400	626	56	447	NL	44	20	22,000	16,923	7,200	1,879	112	103	1,289	40
	Ind.(1,2,3-cd)pyr.[c]	169	564	240	898,933	12,918	5,962	228,830	83	20	2,200	1,692	720	NL	25,836	404	411	40
	Phenanthrene[c]	16,923	56,410	24,000	531	254	2,981	263	99	20	220,000	169,231	72,000	1,594	509	382	1,535	40
	Pyrene[c]	16,923	56,410	24,000	3,955	814	4,472	1,121	368	-	220,000	169,231	72,000	11,864	1,627	1,383	9,205	-
	Anthracene	42,857	115,385	NL	NL	224	140,647	AWQC	222	20	1,000,000	329,670	NL	NL	447	446	165,746	40
	Benzo(g,h,i)per.	4,286	11,538	NL	NL	199	6,329	61,590	181	20	100,000	32,967	NL	NL	397	391	23,622	40
	Fluorene	5,714	15,385	NL	221,340	254	8,439	263	124	-	133,333	43,956	NL	664,040	509	501	30,526	-
	Naphthalene	2,857	7,692	600,000	116	13	16	AWQC	6.8	20	66,667	21,978	NL	349	27	25	342	40
Medium till fine sand	Acenaphthene[c]	16,923	56,410	24,000	47	96	42	AWQC	18	-	220,000	169,231	72,000	142	192	82	142	-
	Acenaphthylene[c]	1,692	5,641	2,400	2.0	18	4.3	32	1.2	-	22,000	16,923	7,200	6	36	5.1	6.0	-
	Benzo(a)anthr.[c]	169	564	240	946	182	72	3,232	31	20	2,200	1,692	720	2,838	365	181	359	40
Concrete basement	Benzo(a)pyr.[c]	17	56	24	291	80	8.9	14,110	4.1	20	220	169	72	872	159	32	39	40
	Benzo(b)fl.[c]	169	564	240	878	317	149	15,831	44	-	2,200	1,692	720	2,633	633	228	355	-
	Benzo(k)fl.[c]	169	564	240	286	121	89	12,289	29	20	2,200	1,692	720	858	242	129	278	40
	Chrysene[c]	1,692	5,641	2,400	1489	55	894	2,452	46	20	22,000	16,923	7,200	4,466	110	105	2140	40
	Dibenzo(a,h)anth.[c]	17	56	24	1,0180	94	179	16,577	7.4	-	220	169	72	30,540	187	34	41	-
	Fluoranthene[c]	1,692	5,641	2,400	1,000	56	447	NL	45	20	22,000	16,923	7,200	2,999	112	105	1,734	40
	Ind.(1,2,3-cd)pyr.[c]	169	564	240	NL	12,918	5,962	228,830	83	20	2,200	1,692	720	NL	25,836	404	411	40
	Phenanthrene[c]	16,923	56,410	24,000	848	254	2,981	263	107	20	220,000	169,231	72,000	2,543	509	420	2,395	40

Pyrene[c]	16,923	56,410	24,000	6,311	814	4,472	1,121	382	-	220,000	169,231	72,000	18,933	1,627	1,446	12,960	-
Anthracene	42,857	115,385	NL	NL	224	140,647	AWQC	222	20	1,000,000	329,670	NL	NL	447	446	247,934	40
Benzo(g,h,i)per.	4,286	11,538	NL	NL	199	6,329	61,590	181	20	100,000	32,967	NL	NL	397	391	24,793	40
Fluorene	5,714	15,385	NL	353,070	254	8,439	263	123	-	133,333	43,956	NL	NL	509	501	33,058	-
Naphthalene	2,857	7,692	600,000	185	13	16	AWQC	6.9	20	66,667	21,978	NL	554	27	26	536	40

Reference soil concentrations: C_{is}: soil ingestion; C_{du}: dermal pathway; C_{id}: dust inhalation; C_{iv}: vapour inhalation; C_{iw}: intake drinking water; C_{ig}: vegetable consumption; C_{if}: fish consumption. Integrated human toxicological values: C_{KM}: land with sensitive land-use; $C_{MKM\ GV}$: land with less sensitive land-use and groundwater extraction; C_{MKM}: land with sensitive land-use). Ecotoxicological values for on-site effects: E_{KM}: land with sensitive land-use; E_{MKM}: land with less sensitive land-use. NL: not limited (> 1,000,000 mg/kg ds); AWQC: no ambient water quality criteria for fish consumption available; A: value adjusted for background exposure; [c]: PAH considered carcinogenic.

Dominant pathways in the derivation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM} are indicated by different cell fill patterns:

M	$C_{MKM\ GV}$	C_{MKM}	$C_{MKM\ GV}$ and C_{MKM}

Since additivity is assumed for the carcinogenic PAHs (marked [c]), the following rule applies for evaluation of soil contamination: if $\sum_i \dfrac{concentration\ PAH_i\ in\ soil}{C_{(M)KM\ (GV)_i}} \leq 1$ then there is no excess lifetime cancer risk of $1/10^5$.

Table 47: Calculated reference soil concentrations for different pathways and integrated values in S-RISK (concentrations in mg/kg dw)

Scenario	Substance	KM									MKM							
		C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	C_{ig}	C_{if}	C_{KM}	E_{KM}	C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	$C_{MKM\ GV}$	C_{MKM}	E_{MKM}
Silty sand	Acenaphthene[c]	16,923	56,410	24,000	1774	96	42	AWQC	29	-	220,000	169,231	72,000	5,322	192	185	4,712	-
	Acenaphthylene[c]	1,692	5,641	2,400	72	18	4.3	32	3.0	-	22,000	16,923	7,200	216	36	31	205	-
	Benzo(a)anthr.[c]	169	564	240	35,489	182	72	3,232	32	20	2,200	1,692	720	106,468	365	193	409	40
Concrete basement	Benzo(a)pyr.[c]	17	56	24	10,903	80	8.9	14,110	4.1	20	220	169	72	32,710	159	33	41	40
	Benzo(b)fl.[c]	169	564	240	32,927	317	149	15,831	46	-	2,200	1,692	720	98,782	633	249	409	-
	Benzo(k)fl.[c]	169	564	240	10,735	121	89	12,289	32	20	2,200	1,692	720	32,205	242	152	406	40
	Chrysene[c]	1,692	5,641	2,400	55,858	55	894	2,452	48	20	22,000	16,923	7,200	167,574	110	107	4,010	40
	Dibenzo(a,h)anth.[c]	17	56	24	381,960	94	179	16,577	7.4	-	220	169	72	NL	187	34	41	-
	Fluoranthene[c]	1,692	5,641	2,400	37,504	56	447	NL	47	20	22,000	16,923	7,200	112,513	112	109	3,963	40
	Ind.(1,2,3-cd)pyr.[c]	169	564	240	NL	12,918	5,962	228,830	83	20	2,200	1,692	720	NL	25,836	404	411	40
	Phenanthrene[c]	16,923	56,410	24,000	31,816	254	2,981	263	122	20	220,000	169,231	72,000	95,447	509	500	28,719	40
	Pyrene[c]	16,923	56,410	24,000	236,811	814	4,472	1,121	406	-	220,000	169,231	72,000	710,432	1,627	1,562	38,833	-
	Anthracene	42,857	115,385	NL	NL	224	140,647	AWQC	222	20	1,000,000	329,670	NL	NL	447	446	247,934	40
	Benzo(g,h,i)per.	4,286	11,538	NL	NL	199	6,329	61,590	181	20	100,000	32,967	NL	NL	397	391	24,793	40
	Fluorene	5,714	15,385	NL	NL	254	8,439	263	123	-	133,333	43,956	NL	NL	509	501	33,058	-
	Naphthalene	2,857	7,692	600,000	6,944	13	16	AWQC	7.1	20	66,667	21,978	NL	20,833	27	27	9,217	40
Clayey loam	Acenaphthene[c]	16,923	56,410	24,000	4,690	96	42	AWQC	29	-	220,000	169,231	72,000	14,071	192	189	10,481	-
	Acenaphthylene[c]	1,692	5,641	2,400	191	18	4.3	32	3.1	-	22,000	16,923	7,200	573	36	34	503	-
	Benzo(a)anthr.[c]	169	564	240	93,689	182	72	3,232	32	20	2,200	1,692	720	281,066	365	193	410	40
Concrete basement	Benzo(a)pyr.[c]	17	56	24	28,783	80	8.9	14,110	4.1	20	220	169	72	86,350	159	33	41	40
	Benzo(b)fl.[c]	169	564	240	86,923	317	149	15,831	46	-	2,200	1,692	720	260,769	633	249	410	-
	Benzo(k)fl.[c]	169	564	240	28,339	121	89	12,289	32	20	2,200	1,692	720	85,017	242	152	409	40
	Chrysene[c]	1,692	5,641	2,400	147,462	55	894	2,452	48	20	22,000	16,923	7,200	442,385	110	107	4,070	40
	Dibenzo(a,h)anth.[c]	17	56	24	NL	94	179	16,577	7.4	-	220	169	72	NL	187	34	41	-
	Fluoranthene[c]	1,692	5,641	2,400	99,011	56	447	NL	47	20	22,000	16,923	7,200	297,032	112	109	4,052	40
	Ind.(1,2,3-cd)pyr.[c]	169	564	240	NL	12,918	5,962	228,830	83	20	2,200	1,692	720	NL	25,836	404	411	40
	Phenanthrene[c]	16,923	56,410	24,000	84,022	254	2,981	263	122	20	220,000	169,231	72,000	252,065	509	502	35,322	40
	Pyrene	16,923	56,410	24,000	625,198	814	4,472	1,121	406	-	220,000	169,231	72,000	NL	1,627	1,565	41,079	-
	Anthracene	42,857	115,385	NL	NL	224	140,647	AWQC	222	20	1,000,000	329,670	NL	NL	447	446	247934	40
	Benzo(g,h,i)per.	4,286	11,538	NL	NL	199	6,329	61,590	182	20	100,000	32,967	NL	NL	397	391	24,793	40
	Fluorene	5,714	15,385	NL	NL	254	8,439	263	124	-	133,333	43,956	NL	NL	509	501	33,058	-
	Naphthalene	2,857	7,692	600,000	18,401	13	16	AWQC	7.1	20	66,667	21,978	NL	55,204	27	27	12,720	40

Reference soil concentrations: C_{is}: soil ingestion; C_{du}: dermal pathway; C_{id}: dust inhalation; C_{iv}: vapour inhalation; C_{iw}: intake drinking water; C_{ig}: vegetable consumption; C_{if}: fish consumption. Integrated human toxicological values: C_{KM}: land with sensitive land-use; $C_{MKM\ GV}$: land with less sensitive land-use and groundwater extraction; C_{MKM}: land with sensitive land-use). Ecotoxicological values for on-site effects: E_{KM}: land with sensitive land-use; E_{MKM}: land with less sensitive land-use. NL: not limited (> 1,000,000 mg/kg ds); ~~AWQC~~: no ambient water quality criteria for fish consumption available; A: value adjusted for background exposure; [c]: PAH considered carcinogenic.

Dominant pathways in the derivation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM} are indicated by different cell fill patterns:

M	$C_{MKM\ GV}$	C_{MKM}	$C_{MKM\ GV}$ and C_{MKM}

Since additivity is assumed for the carcinogenic PAHs (marked [c]), the following rule applies for evaluation of soil contamination: if $\sum\limits_{i} \dfrac{concentration\ PAH_i\ in\ soil}{C_{(M)KM\ (GV)_i}} \leq 1$ then there is no excess lifetime cancer risk of $1/10^5$.

The dominant pathway in the calculation of C_{KM} and $C_{MKM\ GV}$ is the intake of drinking water (C_{iw}). Ingestion of soil (C_{is}) is the dominant pathway (after adjustment for background exposure) in the derivation of C_{MKM}.

The calculated reference soil concentration for the vegetable consumption (C_{ig}) in S-RISK is 14 mg/kg dw, or, adjusted for background exposure: 10 mg/kg dw. The $BCF_{overall}$ proposed in section 3.2.5.2 - 0.031 (mg/kg fw)/(mg/kg dw) - is derived for a soil concentration of 1.8 mg Cd/kg dw. It is known that BCF values for Cd decrease with increasing soil Cd concentrations. However, for the calculated human health based guideline value C_{KM} (3.1 mg/kg dw) the $BCF_{overall}$ is appropriate. For soil concentrations of 2, 3, and 5 mg Cd/kg dw, the $BCF_{overall}$ is 0.03, 0.028, and 0.027 (mg/kg fw)/(mg/kg dw) respectively. Also, since C_{iw} is the dominant route in the derivation for C_{KM} the latter will always approximate 4-3 mg/kg dw, regardless C_{ig}'s order of magnitude. Therefore the BCF values proposed in section 3.2.5.2 are not changed.

6.1.2   PAHs

- *Calculation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM}*

The importance of the pathways in the reference soil concentration is given in Table 48. Vapour inhalation is the dominant pathway for a majority of PAHs in case of a concrete floor and sandy soil for all land-uses. For dibenzo(a,h)anthracene and indeno(1,2,3-cd)pyrene soil ingestion (KM) or dust inhalation (MKM) dominate. For the less volatile PAHs either vegetable intake or water abstraction dominate in KM. In case of a basement, vapour intrusion becomes less important and is replaced by vegetable intake as a critical pathway in most of the cases in KM. For MKMGV and a basement the water pathway is rather important, whereas for MKM vapour intrusion is only dominant for the volatile PAHs, while dust inhalation and dermal uptake will dominate most of the reference soil concentrations.

The overall differences in calculated reference soil concentrations between the six scenarios can be attributed to the use of different soil air to indoor air dilution factors (DF_{ia}). If dilution is more pronounced, the reference soil concentration for the vapour inhalation pathway will increase so that the integrated value (C_{KM}, $C_{MKM\ GV}$ and C_{MKM}) in its turn will increase. This effect will be of greater importance if C_{iv} is the dominant route in the calculation of the integrated value. In short, the integrated value will increase in the order: scenario concrete floor < scenario basement, and in the order scenario medium/fine sand < scenario silty sand < scenario clayey loam.

Table 48: Dominant pathways in the calculated soil reference concentrations

	Substance	KM			MKMGV			MKM		
		MFS	SS	CL	MFS	SS	CL	MFS	SS	CL
concrete floor	Acenaphthene[c]	vapour	vapour	vapour	vapour	vapour	vapour	vapour	vapour	vapour
	Acenaphthylene[c]	vapour	vapour	vapour	vapour	vapour	vapour	vapour	vapour	vapour
	Benzo(a)anthr. [c]	vapour	veget	veget	vapour	water	water	vapour	vapour	dust inh.
	Benzo(a)pyr. [c]	vapour	veget	veget	vapour	dust inh.	dust inh.	vapour	dust inh.	dust inh.
	Benzo(b)fl. [c]	vapour	veget	veget	vapour	vapour	water	vapour	vapour	dust inh.
	Benzo(k)fl. [c]	vapour	vapour	veget	vapour	vapour	water	vapour	vapour	vapour
	Chrysene[c]	vapour	water	water	vapour	water	water	vapour	vapour	vapour
	Dibenzo(a,h)anth. [c]	soil	soil	soil	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.
	Fluoranthene[c]	vapour	water	water	vapour	water	water	vapour	vapour	vapour
	Ind.(1,2,3-cd)pyr. [c]	soil	soil	soil	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.
	Phenanthrene[c]	vapour	water	water	vapour	vapour	water	vapour	vapour	vapour
	Pyrene[c]	vapour	water	water	vapour	water	water	vapour	vapour	vapour
	Anthracene	water	water	water	water	water	water	vapour	dermal	dermal
	Benzo(g,h,i)per.	water	water	water	water	water	water	dermal	dermal	dermal
	Fluorene	water	water	water	water	water	water	vapour	dermal	dermal
	Naphthalene	vapour	water	water	vapour	water	water	vapour	vapour	vapour
basement	Acenaphthene[c]	vapour	veget	veget	vapour	water	water	vapour	vapour	vapour
	Acenaphthylene[c]	vapour	veget	veget	vapour	water	water	vapour	vapour	vapour
	Benzo(a)anthr. [c]	veget	veget	veget	water	water	water	dust inh.	dust inh.	dust inh.
	Benzo(a)pyr. [c]	veget	veget	veget	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.
	Benzo(b)fl. [c]	veget	veget	veget	water	water	water	dust inh.	dust inh.	dust inh.
	Benzo(k)fl. [c]	veget	veget	veget	water	water	water	dust inh.	dust inh.	dust inh.
	Chrysene[c]	water	water	water	water	water	water	vapour	dust inh.	dust inh.
	Dibenzo(a,h)anth. [c]	soil	soil	soil	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.
	Fluoranthene[c]	water	water	water	water	water	water	dust inh.	dust inh.	dust inh.
	Ind.(1,2,3-cd)pyr. [c]	soil	soil	soil	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.	dust inh.
	Phenanthrene[c]	water	water	water	water	water	water	vapour	dust inh.	dust inh.
	Pyrene	water	water	water	water	water	water	vapour	dust inh.	dust inh.
	Anthracene	water	water	water	water	water	water	dermal	dermal	dermal
	Benzo(g,h,i)per.	water	water	water	water	water	water	dermal	dermal	dermal
	Fluorene	water	water	water	water	water	water	dermal	dermal	dermal
	Naphthalene	water	water	water	water	water	water	vapour	vapour	dermal

MFS: medium till fine sand; SS: silty sand; CL: clayey loam

dominant pathways: vapour (vapour intrusion); veget (vegetable consumption); water: drinking water use; dust inh. (dust inhalation); dermal (dermal absorption)

- *Evaluation of carcinogenic PAHs in soil*

The cumulative risk of a combination of carcinogenic PAHs is assumed to be additive. Therefore, the following rule applies for soil contamination:

if $\sum_i \dfrac{concentration\ PAH_i\ in\ soil}{C_{(M)KM(GV)_i}} \le 1$ then there is no excess lifetime cancer risk of $1/10^5$.

In other words, for each carcinogenic PAH compound, the concentration in soil has to be weighed against its corresponding integrated human health based generic soil guideline (in case of land with sensitive land-use, this is C_{KM}, in case of land with less sensitive land-use with or without groundwater extraction, this is $C_{MKM\ GV}$ or C_{MKM} respectively). Then, the sum of all ratios may not exceed 1.

6.2 Comparison with current guideline values

Since the most common soil type used in JM construction is loamy sand (road filling Ludvig; see section 3.2.2), the current human health based guideline values (S-EPA) are compared with the calculated guideline values for silty sand (both buildings with concrete floor and concrete basement). The comparison is given in Table 49.

Table 49: Comparision of proposed human health based soil guideline values (S-RISK) with current (S-EPA) guideline values (mg/kg dw).

Substance	S-RISK		S-EPA
	Concrete floor	Concrete basement	

	KM	MKM GV	MKM	KM	MKM GV	MKM	KM	MKM GV	MKM
Cadmium**	3.0	9.0	905	3.0	9.0	905	0.4	1	200
carcinogenic PAHs***									
Acenaphthene	7.0	24	27	29	185	4,700	$\sum(PAH)_C$:	$\sum(PAH)_C$:	$\sum(PAH)_C$:
Acenaphthylene	0.33	1.1	1.1	3	31	205	0.3 *	7 *	7 *
Benzo(a)anthracene	27	143	235	32	193	409			
Benzo(a)pyrene	3.8	27	33	4.1	33	41			
Benzo(b)fluoranthene	36	167	227	46	249	409			
Benzo(k)fluoranthene	20	79	118	32	152	406			
Chrysene	41	95	710	48	107	4,000			
Dibenzo(a,h)anthracene	7.4	33	41	7.4	34	41			
Fluoranthene	38	92	507	47	109	3,960			
Indeno(1,2,3-cd)pyrene	83	404	411	83	404	411			
Phenanthrene	70	248	485	122	500	28,700			
Pyrene	304	1,095	3,352	406	1,562	38,900			
noncarcinogenic PAHs									
Anthracene	221	446	165,750	222	446	247,900	$\sum(PAH)_N$	$\sum(PAH)_N$	$\sum(PAH)_N$
Benzo(g,h,i)perylene	181	391	23,600	181	391	24,800	c: 25 *	c: 250 *	c: 3,000 *
Fluorene	123	500	27,670	123	500	33,000			
Naphthalene	6.0	21	106	7.1	27	9,200			

Note: Calculated soil guideline values apply to silty soils.

* Human health based guideline values for PAHs are as follows: (i) sum of carcinogenic PAHs: benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-cd)pyrene and dibenzo(a,h)anthracene: KM: 0.3 mg/kg dw, MKM GV and MKM: 7 mg/kg dw; (ii) sum of other PAHs: naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene and benzo(g,h,i)perylene: KM: 25 mg/kg dw, MKM GV: 250 mg/kg dw, and MKM: 3,000 mg/kg dw (Naturvårdsverket, 1996b).

**: Guideline values for the scenarios buildings with concrete floor and concrete basement are the same since C_a=0 for both scenarios.

**: if $\sum_i \frac{concentration\ PAH_i\ in\ soil}{C_{(M)KM\ (GV)_i}} \leq 1$ then there is no excess lifetime cancer risk of $1/10^5$

6.2.1 Cadmium

In Table 50, a detailed comparison is given between the reference soil concentrations for different pathways and the generic guideline values calculated by either S-EPA and S-RISK. Not all reference soil concentrations reported by S-EPA (Naturvårdsverket, 1996b) could be reproduced by input of the S-EPA methodology and S-EPA database in S-RISK Excel. From this table, it can be seen that in both methodologies, C_{iw} (consumption drinking water) is the dominant pathway in the calculation of C_{KM} and $C_{MKM\ GV}$. For C_{MKM}, the dominant pathway in S-EPA is C_{id} (inhalation dust); in S-RISK, this is C_{is} (ingestion soil).

The difference in calculated C_{iw} (dominant parameter for C_{KM} and $C_{MKM\ GV}$) between S-EPA and S-RISK is to a large extent due to the parameter values for the drinking water limit and the K_d. The reported drinking water limit in the S-EPA database is 0.001 mg/l (taken from SLV (1993); 10% of *TDI*); in the S-RISK database, the drinking water limit is 0.003 mg/l (WHO drinking water limit (1996); 10% of *PTWI*). The K_d values in the S-EPA and S-RISK databases are 30 l/kg and 102 l/kg respectively.

For C_{id}, the difference between S-EPA and S-RISK is largely due to the difference in parameter value used for C_{ad} (the annual average dust concentration in inhaled air): in the S-EPA methodology, the default value is 4.1×10^{-2} mg/m³, in S-RISK, C_{ad} is calculated as 5×10^{-3} mg/m³.

6.2.2 PAHs

In the S-EPA methodology, the PAH compounds are divided into two groups: carcinogenic PAHs and non-carcinogenic PAHs. Then, soil guideline values are calculated for the sum of each group. For the carcinogenic PAH compounds, the overall soil guideline value is based on benzo(a)pyrene. The soil guideline value for the non-carcinogenic PAHs is based on reference soil concentrations for pyrene (C_{is}, C_{du}) and fluorene (C_{iv}, C_{iw}, C_{ig} and C_{if}).

In the S-RISK methodology, by introduction of the TEF concept, for each PAH compound a soil guideline value is calculated. The applied TEF scheme was determined by consensus of opinion. A consequence of the introduction of TEFs is that some PAHs considered non-carcinogenic in S-EPA are to be considered carcinogenic (with no threshold for effects) in S-RISK. This is the case for acenaphthene, acenaphthylene, fluoranthene, phenanthrene and pyrene (see Table 46).

Since for carcinogenic PAHs additivity is assumed, the following rule applies for soil contamination:

if $\sum_i \frac{concentration\ PAH_i\ in\ soil}{C(M)KM\ (GV)_i} \leq 1$ then there is no excess lifetime cancer risk of $1/10^5$.

This approach is different from that applied for dioxins. In case of dioxins the TEF approach is applied to the measured concentrations and the TEQ value is compared to a single reference concentration based 2,3,7,8-TCDD. In case of PAHs, the difference in transfer properties of the individual PAHs are taken into account and the TEF approach is applied to the toxicological reference value, resulting in a series of reference concentrations. For benzo(a)pyrene, the reference soil concentrations calculated in S-RISK are compared with the values calculated following the S-EPA methodology. Results are given in Table 51. It must be remarked that not all reference soil concentrations reported in Naturvardsverket (1996b) could be reproduced with S-RISK Excel, although the input parameters from the S-EPA database were used.

From Table 51 it can be seen that in the calculation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM}, the same reference soil concentrations were the dominant pathways in both S-EPA and S-RISK. For C_{KM}, the dominant pathway is intake of vegetables (C_{ig}), for $C_{MKM\ GV}$ and C_{MKM}, this is the inhalation of dust (C_{id}).
For C_{KM}, the difference in C_{ig} between S-EPA and S-RISK is to a large extent attributable to the difference in parameter value used for K_{pl} (the plant-soil concentration ratio): in the S-EPA database, K_{pl} is 0.038 (mg/kg fw)/(mg/kg dw), in the S-RISK database, K_{pl} is 0.002 (mg/kg fw)/(mg/kg dw).
For $C_{MKM\ GV}$ and C_{MKM}, the difference in C_{id} between S-EPA and S-RISK can be attributed to the difference in parameter value used for C_{ad} (the annual average dust concentration in inhaled air): in the S-EPA methodology, the default value is 4.1×10^{-2} mg/m³, in S-RISK, C_{ad} is calculated as 5×10^{-3} mg/m³.

Table 50: Comparison of calculated reference soil concentrations for different pathways and integrated human health based values in S-EPA and S-RISK (silty sand) for cadmium (concentrations in [mg/kg dw]).

Scenario	Model	KM									MKM							
		C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	C_{ig}	C_{if}	C_{KM}	E_{KM}	C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	$C_{MKM\,GV}$	C_{MKM}	E_{MKM}
Default	S-RISK	143 107 A	1,250 938 A	1,000 1,000 A	C_a=0	4.6 4.6 A	14 10 A	AWQC	3.3 3.1 A	6	3,333 2,500 A	3,571 2,679 A	3,000 3,000 A	C_a=0	9.2 9.2 A	9.1 9.1 A	1,095 904 A	12
Default	S-EPA	100 75 A (? A)	357 268 A (? A)	122 (125) 122 A (? A)	C_a=0	0.45 0.45 A (? A)	4.4 3.3 A (? A)	AWQC	0.41 0.39 A (? A)	6	3,333 2,500 (? A)	1,020 765 A (? A)	366 (375) 366 A (? A)	C_a=0	0.91 0.91 A (? A)	0.90 0.90 A (1 A)	249 (254) 225 A (200 A)	12

Note: if reference soil concentrations following the S-EPA methodology and calculated in S-RISK Excel differs from the reported values in Naturvårdsverket (1996b), then the reported values are given between brackets.
A: value adjusted for background exposure.

Dominant pathways in the derivation of C_{KM}, $C_{MKM\,GV}$ and C_{MKM} are indicated by different cell fill patterns:

C_{KM}	$C_{MKM\,GV}$	C_{MKM}	$C_{MKM\,GV}$ *and* C_{MKM}

Table 51: Comparison of calculated reference soil concentrations for different pathways and integrated human health based values in S-EPA and S-RISK (silty sand) for benzo(a)pyrene (concentrations in [mg/kg dw]).

Scenario	Model	KM									MKM							
		C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	C_{ig}	C_{if}	C_{KM}	E_{KM}	C_{is}	C_{du}	C_{id}	C_{iv}	C_{iw}	$C_{MKM\,GV}$	C_{MKM}	E_{MKM}
Floor	S-RISK	17	56	24	56	80	8.9	14,110	3.8	20	220	169	72	168	159	27	33	40
Basement	S-RISK	17	56	24	10,903	80	8.9	14,110	4.0	20	220	169	72	32,710	159	33	41	40
Default	S-EPA	15	38 (7.7)	2.7 (2.8)	976 (969)	214	0.40 (0.41)	37,944 (38,067)	0.34 (0.33)	20	230	115	8.1 (8.3)	2,927 (2,911)	428	7.1 (7.3)	7.3 (7.4)	40

Note: if reference soil concentrations following the S-EPA methodology and calculated in S-RISK Excel differs from the reported values in Naturvårdsverket (1996b), then the reported values are given between brackets.

Dominant pathways in the derivation of C_{KM}, $C_{MKM\,GV}$ and C_{MKM} are indicated by different cell fill patterns:

C_{KM}	$C_{MKM\,GV}$	C_{MKM}	$C_{MKM\,GV}$ *and* C_{MKM}

7 GENERAL CONCLUSIONS

Revisions are proposed for the generic S-EPA methodology for the derivation of guideline values for soil contamination, for Cd and PAHs. These revisions are justified by advancing scientific insights in the modelling of transfer and exposure, risk calculations and toxicological aspects of soil contaminants. Also physico-chemical and biological properties are revised. Nevertheless the methodology is oriented towards regions and building practices for which the data were provided by JM. The results are thus not necessarily generally applicable to Sweden.

The revised S-EPA methodology is referred to as S-RISK. Incorporated in an Excel environment as a spreadsheet it is called S-RISK Excel. In S-RISK Excel, compound specific properties can be chosen from the S-EPA or S-RISK databases and both S-EPA and S-RISK models can be loaded.

The conclusions in the following sections apply to the comparison between calculated integrated human health based guideline values in S-RISK Excel for silty sand soils and the reported integrated human health based guideline values in S-EPA (Naturvårdsverket, 1996b). It must be emphasized that application of the revised generic guideline values is in fact a matter of policy.

7.1 Cadmium

For Cd, the proposed human health based guideline values for KM (land with sensitive land-use), C_{KM}, in S-RISK is approximately 8 times larger than the current one calculated with the S-EPA methodology and S-EPA database (3.1 vs. 0.4 mg/kg dw). For MKM GV (land with less sensitive land-use and groundwater extraction), the proposed guideline value, $C_{MKM\ GV}$, is approximately 9 times larger than the current one (9.1 vs. 1 mg/kg dw). The difference in guideline values between both methodologies is attributed to the difference in calculated C_{iw} (drinking water ingestion pathway), the dominant pathway for C_{KM} and $C_{MKM\ GV}$ in both S-RISK and S-EPA. C_{iw} in its turn, is to a large extent determined by the drinking water limit and the K_d value: in the S-EPA database the drinking water limit is 0.001 mg/l and the K_d is 30 l/kg while in the S-RISK database, the drinking water limit is 0.003 mg/l and the K_d is 102 l/kg.

The human health based guideline value for land with less sensitive land-use and without groundwater extraction, C_{MKM}, is approximately 4.5 times larger than the current one (904 vs. 200 mg/kg dw). For this land-use, the dominant pathway in S-EPA is C_{id} (inhalation dust) while in S-RISK, this is C_{is} (ingestion soil). For C_{id}, the difference between S-EPA and S-RISK is largely due to the difference in parameter value used for C_{ad} (the annual average dust concentration in inhaled air): in the S-EPA methodology, the default value is 4.1×10^{-2} mg/m³, in S-RISK, C_{ad} is calculated as 5×10^{-3} mg/m³.

7.2 PAHs

In the S-RISK methodology, by introduction of the TEF concept, for each PAH compound a soil guideline value is calculated. This is an improvement of the current methodology in S-EPA since the new approach facilitates the evaluation of a PAH soil contamination on an individual basis. For the evaluation of a carcinogenic PAH mixture in soil, the additivity rule comes into play. In the S-EPA methodology, the PAH compounds are divided into two groups: carcinogenic PAHs and non-carcinogenic PAHs. Then, soil guideline values are calculated for the sum of each group.

The applied TEF scheme was determined by consensus of opinion. A consequence of the introduction of TEFs is that some PAHs considered non-carcinogenic in S-EPA are to be considered carcinogenic (with no threshold for effects) in S-RISK. This is the case for acenaphthene, acenaphthylene, fluoranthene, phenanthrene and pyrene

For benzo(a)pyrene, the PAH compound on which the generic guideline value in S-EPA is based, the same reference soil concentrations were the dominant pathways in the calculation of C_{KM}, $C_{MKM\ GV}$ and C_{MKM} in both S-EPA and S-RISK. For C_{KM}, the dominant pathway is intake of vegetables (C_{ig}), for $C_{MKM\ GV}$ and C_{MKM}, this is the inhalation of dust (C_{id}). The difference in C_{ig} between S-EPA and S-RISK is to a large extent attributable to the difference in parameter value used for K_{pl} (the plant-soil concentration ratio): in the S-EPA database, K_{pl} is 0.038 (mg/kg fw)/(mg/kg dw), in the S-RISK database, K_{pl} is 0.002 (mg/kg fw)/(mg/kg dw). The difference in C_{id} between S-EPA and S-RISK can be attributed to the difference in parameter value used for C_{ad} (the annual average dust concentration in inhaled air): in the S-EPA methodology, the default value is 4.1×10^{-2} mg/m³, in S-RISK, C_{ad} is calculated as 5×10^{-3} mg/m³.

The proposed human health based guideline value for benzo(a)pyrene for KM calculated in S-RISK is more than ten times larger than the generic guideline value calculated in S-EPA. For MKM GV the calculated guideline values in S-RISK is approximately 4 times larger than the one calculated in S-EPA. For MKM, the generic guideline in S-RISK is approximately 5 times larger than the generic guideline value calculated in S-EPA.

7.3 Remarks

7.3.1 Choice of parameter values: mean values vs. percentiles

Within S-RISK, decisions were made concerning the use of either mean values or percentiles of certain parameter values. However, the ultimate choice of using either mean values or percentiles in the derivation of generic soil guideline values is a policy decision.

7.3.2 Ecotoxicology based guideline values

In the S-EPA framework for developing generic soil guideline values, the basic principle is to choose the lowest of the human health based value and the ecotoxicologically based value (Naturvårdsverket, 1996b). In this report, only the human health based soil guideline

values were revised. These revised guideline values were not integrated with their respective ecotoxicologically based values. Revision of these latter values would mean a significant improvement in the overall derivation of generic soil guideline values.

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APPENDIX A: DETAILS ON THE CONSUMPTION RATES AND THE CONSUMPTION PATTERN

TOTAL CONSUMPTION RATES

Based on the Dutch National Food Consumption Survey (DNFCS) from 1998 (Voedingscentrum, 1998) the (weighted) average lifetime consumption of vegetables was found to be 139 g per day fresh vegetables for adults and schoolgoing children (age 7-70). The (weighted) average lifetime consumption of vegetables for babies and pre-scholers (age 1-6) was 58.3 g per day fresh vegetables. For potatoes the average lifetime consumption was found to be 122 g per day for adults and schoolgoing children and 59.5 g per day for pre-scholers (1-6 year). These data have been summarised in Table C1.

Table C1: The average lifetime consumption [g fresh weight per day] for above-ground vegetables and potatoes (source: Dutch National Food Consumption Survey, DNFC, 1998).

Group	Time span	Aboveground vegetables	Potatoes
Babies and pre-scholars	1-6 year	58.3	59.5
Adults and schoolgoing children[1]	7-70 year	139	122

The presented data with respect to the general average consumption of potatoes and aboveground vegetables can be compared with other available data.

National food consumption surveys for all age groups have been performed in 1987-1988, 1992, 1997-1998 (WVC, 1988; Voorlichtingsbureau voor de voeding, 1993; Voedingscentrum 1998). A recent food consumption survey was performed in 2003 for the specific age-group of 19-30 years (Hulshof et al., 2004). The results of these surveys show a decrease in both the total consumption rate of potatoes and aboveground vegetables, starting from 1988 (Table C2).

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[1] About 6 % of the consumed amounts of vegetables and about 9 % of the consumed amount of potatoes are obtained outdoors. These amounts are not taken into account.

Table C2: Overview of daily consumption rates of potatoes and above-ground vegetables [g fresh weight per day] from Dutch food consumption surveys and for a specific study for kitchen gardeners (between brackets: data for a specific age-group; see text).

	General population				Kitchen gardeners
	Survey	*Survey*	*Survey*	*Survey*	*Survey*
Year	1988	1992	1998	(2003)	1988
Potatoes	132	119	114	(96)	147
Aboveground vegetables	144	128	123	(100)	255
Total	276	247	237	(196)	402

This trend seems to continue in 2003, although the 2003 data should be considered with care since they reflect a specific age-group and the methodology of the survey was different from earlier surveys. However, the average consumption of the age-group of 19-30 years, based on the 1988-1998 data, is expected to be higher than the average of all age groups. Therefore it is remarkable that the results for this age-group for 2003 are low compared with the results for all age groups of the previous years. The data of 2003 confirm the trend of decrease in consumption amounts (pers. comm. K. Hulshof, November 2005).

In theory, the most recent dataset must be preferred. However, the data form 2003 are less representative since they refer to a specific age-group (19-30) and relate to data collected in the fourth quarter of 2003 only. A new baseline survey for all age-groups is foreseen in 2007. Thus, at this moment the 1998 dataset is the most complete dataset actually available for total consumption rates in the Netherlands. It can be expected that the 1998 data give a slight overestimation of the actual vegetable consumption, considering the observed trend in decrease of the consumption of potatoes and aboveground vegetables.

KITCHEN GARDENERS

In a study using 154 households with kitchen gardens (Hulshof, 1988) the average consumption of aboveground vegetables and potatoes (both from own vegetable garden and from other sources) was 255 and 147 gram per day, respectively (all age groups, see Table C2). Thus, kitchen gardeners consume more vegetables than an average individual. Based on the 1988 data from both sources (DNFCS, 1988 and Hulshof, 1988) the factor of difference considering all age-groups for the consumption of potatoes is approximately 1.1. For aboveground vegetables this factor is approximately 1.8 for adults and schoolgoing children and 1.2 for babies and pre-scholars.

Although the data for kitchen gardeners refer to a situation in 1988, it is considered plausible that although the total consumption rates might have changed in time, these factors of difference still are representative for the difference between the total consumption rates of kitchen gardeners and general population. Applying these factors to the average lifetime consumption data (Table C1) results in the average lifetime consumption of aboveground vegetables and potatoes for kitchen gardeners, see Table C3.

Table C3: The average lifetime consumption [g fresh weight per day] for above-ground vegetables and potatoes corrected for kitchen gardeners.

Group		Aboveground	Potatoes

	time span	vegetables	
Babies and pre-scholars	0-6 year	58 x 1.2 = 70	60 x 1.1 = 66
Adults and schoolgoing children	7-70 year	139 x 1.8 = 250	122 x 1.1 = 134

CONSUMPTION PATTERN

Besides the total consumption rates also the contribution of different vegetables to the total consumption rate (consumption pattern) is of importance. Dooren-Flipsen (1996) used the data of DNFCS (1992) to transform the average consumption rates of foodstuffs into consumption rates of primary agricultural products. Based upon these data Versluijs and Otte (2001) considered 31 vegetables, including potatoes. Table C4 gives the average consumption pattern (average for all age-groups and both sexes). The consumption pattern, expressed in gram fresh product per day, is converted to the consumption pattern in gram dry weight per day[1]. The contribution of each crop and each group of crops in the average consumption is presented.

Note that these data from Dooren-Flipsen (1996) in Table C4 and DNFCS (1992) in Table C1 show differences in the total rates of consumed potatoes and vegetables. The reason for this is that the Dutch food consumption survey records in terms of prepared (cooked) actual amounts of foods, which includes composite products. The data in Table C4 are based on the fresh product (at harvesting; shrink and waste included). The latter is applicable for calculation of the generic plant-soil relationships. The first is applicable for the estimation of the total consumption rates. The relative contribution (%) in the average consumption is expected to be similar in both surveys

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[1] Calculation fresh weight - dry weight based upon water content data in EPA Exposure Factor Handbook 1997.

Table C4: Average consumption pattern in the Netherlands (Dooren-Flipsen et al., 1996).

no	Group	Crop	Average consumption	Water content	Average consumption	Contribution to average consumption pattern	
			g fresh weight per day	g/100 g product	g dry weight per day	% (crop)	% (group)
0	Potatoes	Potatoes	179.7	83.3	30.0	61.6	61.6
1	Roots and tubers	Beetroot	5.2	87.3	0.65	1.3	5.09
		Carrots	13.4	87.8	1.64	3.4	
		Celeriac	0.8	88.0	0.09	0.2	
		Turnip	0.8	91.9	0.07	0.1	
		Radish	0.4	94.8	0.02	0.05	
		Winter carrot	0.2	87.8	0.02	0.04	
2	Bulbous vegetables	Onions	17.0	90.8	1.56	3.2	7.7
		Leek	12.9	83.0	2.19	4.5	
3	Fruiting vegetables	Tomatoes	26.1	94.0	1.56	3.2	5.0
		Cucumber	8.0	96.1	0.31	0.6	
		Melon	2.2	89.7	0.23	0.5	
		Maize	1.4	76.0	0.34	0.7	
4	Cabbages	Cauliflower	16.0	92.3	1.23	2.5	7.6
		Brussels sprouts	4.7	86.0	0.65	1.3	
		White cabbage	7.0	95.3	0.33	1.6	
		Red cabbage	5.1	91.6	0.43		
		Ox heart cabbage	2.0	95.3	0.10	0.2	
		Curly kale	4.9	84.5	0.76	1.6	
		Broccoli	2.0	90.7	0.18	0.4	
5	Leaf vegetables (greens)	Lettuce (head)	8.5	95.4	0.39	0.8	4.4
		Endive	7.4	93.8	0.46	0.9	
		Spinach	10.4	91.6	0.88	1.8	
		Chicory	9.2	95.3	0.43	0.9	
6	Legumes (peas and beans)	Green bean	11.7	90.3	1.13	2.3	6.9
		String/bush bean	3.1	90.3	0.30	0.6	
		Broad/horse/fava bean	2.5	88.9	0.28	0.6	
		Garden pea	14.8	88.9	1.64	3.4	
7	Beans	Haricot bean	0.9	77.1	0.20	0.4	1.2
		Kidney bean	1.8	77.1	0.40	0.8	
8	Stem and stalk vegetables	Rhubarb	0.7	93.6	0.05	0.1	0.4
		Asparagus	1.7	92.3	0.13	0.3	

APPENDIX B: COMPARISON OF BCFS

In Table E1 the *BCF* values based on Versluijs and Otte (2001), Bockting and Van den Berg (1992) and several other researchers are listed. To this purpose the *BCF*s from the plant-soil relations for aboveground vegetables and potatoes have been integrated (integrated *BCF* = 0.33).

Table E1: Comparison of bioconcentration factors; among them the proposed BCF for S-RISK based on Versluijs and Otte (2001) and the BCF used in S-EPA from Bockting and Van den Berg (1992).

Versluijs and Otte, 2001	Bockting and Van den Berg, 1992	Van Driel et al., 1988	Baes, 1984	Bechtel and Jacobs, 1998	Sauerbeck and Lüben, 2001
consumption-averaged	geometric mean	consumption-averaged	median	median	Median
0.33	0.37	0.26	0.55	0.51	0.58

The *BCF* value from the proposed plant-soil relations from Versluijs and Otte (2001) for root vegetables and aboveground vegetables (respectively 0.170 and 0.70, with an overall value of 0.33) is of the same order of magnitude of the *BCF* values based on the other vegetable accumulation models. The Bockting and Van den Berg (1992) *BCF* is similar to the *BCF* value from the proposed plant-soil relations from Versluijs and Otte (2001), i.e. 0.37.

Three out of four of the *BCF*s from other four sources are slightly higher, Van Driel et al., 1988 slightly lower. The reason for this is that in the plant-soil relations based integrated *BCF* the lower *BCF* for potatoes gets a high weighting due to high contribution to total consumption (62%). At the other site the Versluijs and Otte (2001) data include more data from slightly contaminated soils then data from higher cadmium levels. For example, the median soil content of cadmium in the dataset of Versluijs (2001) is only 0.45 mg/kg cadmium (10 percentile is 0.12 and the 90 percentile is 3.2). It is known that *BCF*s for slightly contaminated soils are higher then *BCF* values taken from soils with higher cadmium levels.

APPENDIX C: AIR-TO-PLANT AND SOIL-TO-PLANT TRANSFER OF PAHs

1. INTRODUCTION

In the current S-EPA methodology, only uptake from soil is accounted for because it is assumed that uptake of contaminants by deposition of particles on above-ground plant parts is highly uncertain and the soil uptake route is conservative. As a parallel approach to experimentally determined bioconcentration factors, the model equations for uptake of organic contaminants are revised. Recent scientific publications are in favour of an update of the present equations.

For uptake from soil and air (gas phase), the simplified version of the PlantX model from Trapp & Matthies (1995) suitable for incorporation in a guideline development concept. It is also used in the EUSES model for risk assessment of new and existing substances and the CSOIL exposure model. Besides the conceptual PlantX model, also the findings of McLachlan and co-workers are incorporated in the final model equations of S-RISK. McLachlan (and co-workers) discussed a theoretical framework on both dry gaseous deposition and particle-bound deposition of semi-volatile organic compounds (McLachlan, 1995, 1999; McLachlan et al., 1995, 1999). To account for plant concentrations attributable to wet plus dry deposition, an additional model equation (Lorber et al., 1994) is fitted into the concept.

A schematic representation of the overall model is given in Figure F1. For the atmospheric pathways, only gas phase and particle phase concentrations originating from soil are considered in the model.

Literature data suggest that soil outgassing may be a potentially important factor in the change in PAH profile in the vegetable during different seasons. Cousins & Jones (1998) calculated soil-air fugacity quotients from typical UK soil and air concentration data and showed that the net gas phase flux for many low molecular weight PAHs is out of the soil (i.e. volatilization), especially during the summer.
Trapp et al. (1997) found that volatilization from soil and subsequent uptake into leaves could also contribute to the contamination of plants, in particular to the outer leaves. Only the 3- and 4-ring PAHs such as phenanthrene and fluoranthene have a small potential to be taken up via the roots (Matthies, 2003).

Soil contamination of the vegetable surface (i.e. following rain splash, wind blow, animal activity etcetera) can in normal background situations contribute up to approximately 30% of the herbage PAH burden (Smith & Jones, 2000). The effect of soil contamination on vegetables PAH concentrations will vary across the PAH compound class because soils are relatively enriched in the heavier molecular weight PAHs (Cousins et al., 1997).
Delschen et al. (1999) investigated the importance of different pollution sources for the PAH contamination of cultivated plants in a long-term field lysimeter experiment. Their results demonstrate that the PAH pollution may be caused by both the atmospheric deposition and the direct contamination of plant leaves with resuspended soil particles and subsequent PAH turnover by ad/desorption processes. Systemic PAH transfer via root uptake could generally not be observed. They concluded that the soil as well as the

deposition pathway must be integrated into a complex risk assessment of locations with food plant production, particularly in urban areas (Matthies, 2003).

In this appendix, first, the modelling equations to calculate the transfer of organic compounds from soil and air are discussed. Next, the calculation of the concentrations in the gaseous and particulate phase is elaborated. In the following sections, bioconcentration factors are put forward and the dominant process in air-to-plant transfer is discussed. Finally, recommendations for use in S-RISK are given.
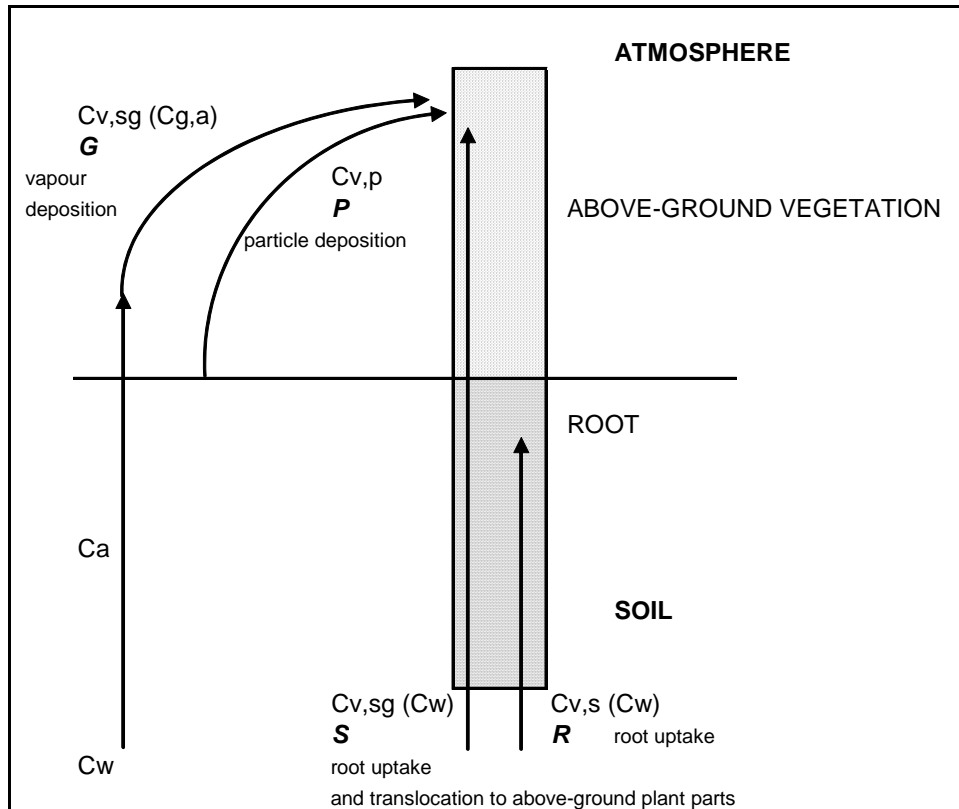
Figure F1: Schematic representation of transfer to vegetables via different pathways (S, G, R, P – see text).

2. ABOVE-GROUND VEGETABLES

2.1 SOIL-GAS PHASE TRANSFER TO VEGETABLES

Transfer of organic compounds from soil (via pore water) and air (via gas phase) to above-ground vegetables is calculated on the basis of the conceptual PlantX model from Trapp & Matthies (1995) and accounting for the findings of McLachlan (1999) and McLachlan et al. (1995, 1999):

$$C_{v,sg}(t) = C_{v,sg}(0) \times e^{-a \times t} + \frac{b}{a.\rho}\left(1 - e^{-a \times t}\right)$$

where:

$C_{v,sg}$: the plant concentration due to gaseous deposition and transfer from soil to above-ground vegetables [mg/kg fresh weight (fw)]; at $t = 0$, $C_{v,sg} = 0$;

t: time [d];

a: sink term accounting elimination of the substance in the plant [d^{-1}];

b: source term, including transfer from soil and uptake by gaseous deposition [mg/m³.d];

ρ: the plant wet density [kg fresh weight/m³]; the default value in EUSES is 700 kg fw/m³.

2.1.1 Sink term

The sink term a, is the sum of the losses by metabolism, photodegradation, and volatilization, and dilution by growth:

$$a = \sum a_i = a_{metabolism} + a_{photo\,deg\,radation} + a_{volatilization} + a_{growth}$$

It is assumed that no metabolization or photodegradation occurs (i.e. $a_{metabolism} = a_{photodegradation} = 0$). The default value for a_{growth} is 0.035 d^{-1} (EUSES). Losses by volatilization are calculated as:

$$a_{volatilization} = \frac{A \times g}{V \times K_{VG}}$$

where:

A: the plant surface area [m²];

V: the plant volume [m³];

g: the leaf conductance [m/d];

K_{VG}: the gas-plant partition coefficient [m³/m³].

The EUSES-default values for A and V are 5 m² and 0.002 m³ respectively.

The use of v_G, the gas deposition velocity [m/d], in the sink term (as well as in the source term, see below) in stead of the conductance can be debated. However, in this modelling approach, the conductance is used as default. Yet, it can be replaced by v_G if more information is available on this factor. The gas deposition velocity can be calculated as the harmonic mean of the transfer rate from atmosphere to the plant surface and the transfer rate from plant surface to plant reservoir.

$$v_G = \frac{1}{\left(\frac{1}{v_{GG}} + \frac{1}{\left(K_{VG} \times v_{GV}\right)} \right)}$$

where:

v_{GG}: the mass transfer rate from atmosphere to plant surface [m/d];
v_{GV}: the mass transfer rate from plant surface to plant reservoir [m/d].

Estimates of average values for the conductance are as follows (Trapp & Matthies, 1995):
Lower boundary: cuticle is comparatively impermeable; uptake mainly via stomata (vapours, approximate when log K_{OW} - log K_{AW} < 5; where K_{AW} is the air-water partition coefficient [m³/m³]); conductance is approximately 86.4-8.64 m/d, depending on plant species and environmental conditions (Riederer, 1994);
Upper boundary: cuticle is relatively permeable (very lipophilic compounds, approximate when log K_{OW} - log K_{AW} > 10); the main resistance is from the atmospheric boundary layer, g is approximately 432 m/d (Thompson, 1983).
For the PAHs under consideration, log K_{OW} - log K_{AW} is apprimately 5-11 so that 86.4 m/d, the EUSES-default value for g, is a good estimate.

The gas-plant partition coefficient K_{VG} is given by an empirical relationship with the octanol-air partition coefficient:

$$K_{VG} = m \times K_{OA}^{n}$$

where m and n are plant specific regression constants. In literature, values for m and n are reported for PCDD/Fs in rye-grass (Böhme et al., 1999) and PCBs in rye-grass, clover, plantain, Hawk's beard and yarrow (Kömp & McLachlan, 1997a,b). The default parameter values for m and n in S-RISK, $10^{-2.53}$ and 1.09 respectively, are chosen from the study of Kömp & McLachlan (1997b) and apply to PCBs in rye-grass.

Maddalena et al. (2002) measured gas-plant (bell peper) partition coefficients for anthracene, fluoranthene, phenanthrene and pyrene in a continuous stirred flow-through exposure chamber. In general, the measured K_{VG}s for PAHs are in close agreement with the modelled ones using Kömp & McLachlan's (1997b) parameter values for PCBs (Table F1).

Table F1: Measured and calculated log K_{VG}s for some PAH compounds.

	Anthracene	Fluoranthene	Phenanthrene	Pyrene
Measured (Maddalena et al., 2002)	5.7	6.0	5.7	6.2
Modelled (Kömp & McLachlan, 1997b)	4.8	6.4	5.4	6.3

2.1.2 Source term

The source term b is defined as:

$$b = C_w \frac{TSCF \times Q_{transp}}{V} + C_{g,a} \frac{g \times A}{V}$$

where:

C_w: the soil pore water concentration [mg/m³];
Q_{transp}:   the transpiration rate [m³/d]; the EUSES-default value is 0.001 m³/d;
$TSCF$:   the transpiration stream concentration factor [-]; it is the ratio between the concentration in the transpiration stream and the concentration in the pore water;
$C_{g,a}$:   the gas phase concentration in air [mg/m³].

The transpiration stream concentration factor $TSCF$ is given by Briggs et al. (1982) and was derived for a small group of pesticides in barley. It is calculated from the octanol-water partition coefficient K_{OW}:

$$TSCF = 0.784 \times e^{-\frac{(\log K_{OW} - 1.78)^2}{2.44}} \qquad \text{for } \log K_{OW} \leq 4.5$$

For $\log K_{OW}$ values above 4.5, $TSCF = 0.038$ and chemicals are almost not taken up from soil.

A calculation method to estimate the gas phase concentration in air ($C_{g,a}$) at the vegetables height and *solely* due to the contamination of soil is discussed in chapter 4.

2.2 PARTICLE PHASE TRANSFER TO VEGETABLES

Plant concentrations resulting from the wet and dry deposition (either measured as F_P or estimated from air particle concentration $C_{p,a}$), are given by:

$$C_{v,p} = \frac{F_P \times I_V \times \left(1 - e^{-k_w \times t}\right)}{k_w \times Y_V} \times dw = \frac{C_{p,a} \times I_V \times \left((V_d) + (R_n \times R_w \times W_p)\right) \times \left(1 - e^{-k_w \times t}\right)}{k_w \times Y_V} \times dw$$

where:

$C_{v,p}$: the plant concentration due to particle deposition [mg/kg fresh weight];
F_P: the contaminant particle deposition flux [mg/m².d)];
$C_{p,a}$: the air particle concentration [mg/m³];
I_V: the fraction of particles intercepted [-];
k_w: the plant weathering constant [d⁻¹];
dw: fresh to dry weight conversion factor [kg dry weigh/kg fresh weight];
Y_V: the plant yield [kg dry weight/m²];
V_d: the dry particle deposition rate [m/d];
R_n: the annual rainfall [m/d];

R_w: the fraction retained after rainfall [-];
W_p: the washout factor [-].

The fraction of particles intercepted by the vegetables, I_V, can be estimated by (Baes et al., 1984):

$$I_V = 1 - e^{-2.88 \times Y_V} \qquad \text{gras/hay}$$

$$I_V = 1 - e^{-0.769 \times Y_v} \qquad \text{corn silage}$$

$$I_V = 1 - e^{-0.0846 \times Y_{V,fresh\ weight}} \qquad \text{leafy vegetables } (Y_{V,fresh\ weight}: [\text{kg fw/m}^2])$$

The plant weathering constant k_w, reported by Lorber et al. (1994) and Douben et al. (1997) is 0.049 d^{-1}. Y_V (0.38 kg dry weight/m²) and t (100 d) are taken from ECETOC (1992) and apply to foliar crops. I_V is 0.4 (default value; ECETOC, 1992). The default value for R_w, the fraction retained after rainfall is 0.3 (Douben et al., 1997; Lorber et al., 1994). Default values for the volumetric washout factor W_p and the dry deposition velocity V_d are 10^5 and 43.2 m/d respectively (Kaupp & McLachlan, 1999). Data on rainfall are: Stockholm: 1.48x10^{-3} m/d, Göteborg: 2.05x10^{-3} m/d, Malmö: 1.64x10^{-3} m/d (arithmetic mean: 1.88x10^{-3} m/d; Meteorological Service Sweden). The fresh to dry weight conversion factor, dw, is 0.12 for above-ground vegetables.

A calculation method to estimate the particle phase concentration in air ($C_{p,a}$) at the vegetables height and *solely* due to the contamination of soil is discussed in chapter 4.

2.3 ATMOSPHERE-SOIL-PLANT TRANSFER

The total above-ground plant concentration $C_{v,sa}$ [m/kg fresh weight] due to atmosphere-soil-plant transfer is given by:

$$C_{v,sa} = C_{v,sg} + C_{v,p}$$

3. ROOT CROPS

The concentration in root tissue $C_{v,s}$ [mg/kg fresh weight] is governed mainly by physical sorption and is given by (EUSES):

$$C_{v,s} = C_w \frac{K_{pl,w}}{\rho_r}$$

where:

$K_{pl,w}$: the partition coefficient between plant tissue and water [m³/m³];

ρ_r: the wet root density [kg fresh weight/m³]; the default value is the same as ρ (i.e. 700 kg fw/m³).

The plant-water partition coefficient is calculated from the octanol-water partition coefficient:

$$K_{pl,w} = \theta_{w,v} + \left(\theta_{l,v} \times K_{OW}^{b_{cf}} \right)$$

where:

$\theta_{w,v}$: the volumetric plant water content [m³/m³]; the EUSES-default value is 0.65 m³/m³;
$\theta_{l,v}$: the volumetric plant lipid content [m³/m³]; the EUSES-default value is 0.01 m³/m³;
b_{cf}: the octanol-lipid correction factor [-]; the EUSES-default value is 0.95.

4. CALCULATION OF $C_{g,a}$ AND $C_{p,a}$

For the calculation of the concentrations in the vapour and particle phases, only the contaminant load originating from the soil contamination is considered, i.e. background concentrations in air (due to road traffic etcetera) are not accounted for.

4.1 CALCULATON OF THE CONCENTRATION IN THE VAPOUR PHASE

The on-site concentration in the vapour phase at the vegetables height due to contamination in the soil is calculated as:

$$C_{g,a} = C_{g,a,0} + \frac{J_{oa,g}}{V_f}$$

where

$C_{g,a,0}$: initial concentration in the vapour phase [mg/m³];
$J_{oa,g}$: diffusion flux vapour phase soil to atmosphere [mg/d.m²];
V_f: dilution velocity [m/d].

The initial concentration in the vapour phase is assumed to equal zero.

To estimate the volatile emissions of a chemical from soil, the model of Farmer et al. (1980) is applied. This model treats vapor loss/emissions from soil as a diffusion controlled process that is quantified using the Fick's law for steady state diffusion. It is incorporated in the American Petroleum Institute's (API) Decision Support System for Exposure and Risk Assessment version 2.0 (API, 1999).

$$J_{oa,g} = \frac{D_e}{d_{sc}} \times \left(C_a - C_{g,a,ss} \right)$$

where

D_e: effective diffusion coefficient of the chemical in air [m²/d];
d_{sc}:   depth of soil cover [m]; assumed to be 1.5 m;
C_a: vapour concentration in pore air [mg/m³]
$C_{g,a,ss}$:   air concentration of the chemical at the soil surface [mg/m³].

It is assumed that $C_{g,a,ss}$ is significantly less than the soil vapour concentration so that $J_{oa,g}$ can be written as:

$$J_{oa,g} = \frac{D_e}{d_{sc}} \times C_a$$

The effective diffusion coefficient of the chemical can be calculated as (Millington & Quirk, 1961):

$$D_e = \left(D_{air} \times \frac{\theta_a^{10/3}}{\theta_t^2} \right) + \left(\frac{D_{water}}{H} \times \frac{\theta_w^{10/3}}{\theta_t^2} \right)$$

where

D_{air}:   diffusion coefficient for the chemical in air [m²/d];
D_{water}:   diffusion coefficient for the chemical in water [m²/d];
θ_a: air-filled porosity of soil [-]; in S-EPA, $\theta_a = 0.2$;
θ_w: water-filled porosity of soil [-]; in S-EPA, $\theta_w = 0.3$;
θ_t: total porosity of soil [-].

In the Johnson & Ettinger (2004) model, the equation of Millington & Quirk (1961) is used to calculate the effective diffusion coefficient across the capillary zone, as well as the effective diffusion coefficient within the saturated zone.

The diffusion coefficients D_{air} [m²/d] and D_{water} [m²/d] are estimated as:

$$D_{air} = 24 \times 0.036 \times \sqrt{\frac{76}{M}}$$

$$D_{water} = 24 \times 0.0000036 \times \sqrt{\frac{76}{M}}$$

where M [g/mol] is the molecular weight of the compound.

In Table F2, estimated parameter values for θ_a, θ_w, and θ_t for different soil textures relevant for the considered region in Sweden are given.

Table F2: Estimation of θ_t, θ_w, and θ_a for different soil textures.

Texture	θ_t		θ_w		θ_a
Medium till fine sand (S)	U (0.340:0.375)	**0.358**	U (0.04:0.076)	**0.058**	0.3
Silty sand (SS)	U (0.375:0.399)	**0.387**	U (0.076:0.146)	**0.111**	0.276
Clay loam (CL)	U (0.399:0.489)	**0.444**	U (0.146:0.216)	**0.181**	0.263

U: uniform distribution (cfr. calculation DF_{ia}); $\theta_a = \theta_t - \theta_w$; values in **bold** are used in the calculation of D_e.

In Table F3, D_{air}, D_{water} and D_e are calculated for the PAHs under consideration.

Table F3: Calculation of D_{air}, D_{water} and D_e for PAHs for different soil textures.

PAH	M	D_{air}	D_{water}	D_e [m²/d]		
	[g/mol]	[m²/d]	[m²/d]	S	SS	CL
Acenaphthene	154.21	0.6065	6.07x10⁻⁵	0.0855	0.0555	0.0360
Acenaphthylene	152.2	0.6105	6.11x10⁻⁵	0.0861	0.0558	0.0362
Anthracene	178.23	0.5642	5.64x10⁻⁵	0.0796	0.0516	0.0335
Benzo(a)anthracene	228.22	0.4986	4.99x10⁻⁵	0.0705	0.0468	0.0341
Benzo(a)pyrene	252.56	0.4740	4.74x10⁻⁵	0.0669	0.0441	0.0311
Benzo(b)fluoranthene	252.24	0.4743	4.74x10⁻⁵	0.0669	0.0436	0.0289
Benzo(g,h,i)perylene	276.34	0.4531	4.53x10⁻⁵	0.0651	0.0506	0.0623
Benzo(k)fluoranthene	252.24	0.4743	4.74x10⁻⁵	0.0669	0.0434	0.0284
Chrysene	228.28	0.4985	4.99x10⁻⁵	0.0703	0.0458	0.0304
Dibenzo(a,h)anthracene	278.36	0.4515	4.51x10⁻⁵	0.0667	0.0640	0.1148
Fluoranthene	202.2	0.5297	5.3x10⁻⁵	0.0747	0.0487	0.0323
Fluorene	166.22	0.5842	5.84x10⁻⁵	0.0824	0.0535	0.0348
Indeno(1,2,3-cd)pyrene	276.33	0.4531	4.53x10⁻⁵	0.0670	0.0647	0.1171
Naphthalene	128.18	0.6653	6.65x10⁻⁵	0.0938	0.0608	0.0394
Phenantrene	178.23	0.5642	5.64x10⁻⁵	0.0796	0.0517	0.0339
Pyrene	202.27	0.5296	5.3x10⁻⁵	0.0747	0.0487	0.0323

S: medium till fine sand; SS: silty sand; CL: clay loam.

The dilution velocity V_f is calculated by applying the Box model as described in Appendix I (also used in the Vlier-Humaan 2.0 model, Flanders). The same parameter values are used as in the calculation of the average dust concentration, except for $J_{oa,p}$ and Y. $J_{oa,p}$ is replaced by $J_{oa,g}$, calculated by the Farmer's model. Y, the receptor height is set to 0.2 m, i.e. a value considered representative for the average height of the vegetables.

For Stockholm, Malmö and Göteborg, the following parameter values were used to calculate V_f:

C_o: 9.06;
A: 100x100 m²;
B: 100 m;
L_{bl}:   100 m;
S_z: 60 m;
Y: 0.2 m (vegetables);
V_h: Stockholm: 273,024 m/d; Malmö: 315,360 m/d; Göteborg: 254,880 m/d;
h: 10 m;
k: 0.4;
S_r: 0.6 m.

The calculated dilution velocities V_f are: Stockholm: 11,650 m/d, Malmö: 13,457 m/d, and Göteborg: 10,876 m/d. The default input value in S-RISK is set to 12,000 m/d (arithmethic mean of the three values: 11,994 m/d).

4.2 CALCULATON OF THE CONCENTRATION IN THE PARTICLE PHASE

Concentrations of PAH compounds in the particle phase are calculated on the basis of the calculated annual average outdoor concentration of inhaled dust (Appendix I). The proposed dust concentration ($C_{dust\ in\ air}$, 5×10^{-3} mg/m³) takes into account wind-induced dust emissions and (by the introduction of a safety factor) also additional (mechanical) dust emissions due to activities on the site (e.g. children playing, agricultural activities, …). Only deposition of dust particles originating from the site is considered.

The air particle concentration $C_{p,a}$ is calculated as:

$$C_{p,a} = \frac{C_s}{C_{dust\ in\ air}}$$

5. BIOCONCENTRATION FACTORS

The following bioconcentration factors [(mg/kg fw plant)/(mg/kg soil)] can be defined:

$$BCF_{above-ground,fw/dw} = \frac{C_{v,sa}}{C_s}$$

$$BCF_{root,fw/dw} = \frac{C_{v,s}}{C_s}$$

$$K_{pl} = \left(BCF_{above-ground,fw/dw} \times f_{leaf}\right) + \left(BCF_{root,fw/dw} \times f_{root}\right)$$

Since C_w, C_a, $C_{g,a}$ and $C_{p,a}$ can be written in terms of C_s, these bioconcentration factors can be simplified. Therefore, the following variables are introduced:

$$b_1 = \frac{TSCF \times Q_{transp}}{V} \qquad b_2 = \frac{g \times A}{V} \qquad b_3 = \frac{1}{K_d + \frac{(\theta_w + \theta_a \times H)}{\rho_b}}$$

$$c_1 = \frac{1}{V_f} \times \frac{D_e}{d_{sc}} \qquad c_2 = \frac{I_V \times \left((V_d) + \left(R_n \times R_w \times W_p\right)\right) \times \left(1 - e^{-k_w \times t}\right)}{k_w \times Y_V} \times dw$$

$$B_1 = b_1 \times b_3 \qquad B_2 = H \times b_2 \times b_3 \times c_1$$

$$r_1 = \frac{\theta_{w,v} + \left(\theta_{l,v} \times K_{OW}^{b_{cf}}\right)}{\rho_r} \qquad R = r_1 \times b_3 \qquad P = \frac{c_2}{C_{dust\,in\,air} \times 1000000}$$

The source term b can then be written as:

$$b = C_s \times \left(B_1 + B_2\right)$$

By the introduction of S and G:

$$S = \left(B_1\right) \times \left(\frac{1}{a \times \rho}\right) \times \left(1 - e^{-a \times t}\right)$$

$$G = \left(B_2\right) \times \left(\frac{1}{a \times \rho}\right) \times \left(1 - e^{-a \times t}\right)$$

the concentration in the vegetables $C_{v,sg}$ due to the soil-gas phase transfer can then be written as:

$$C_{v,sg} = C_s \times \left(S + G\right)$$

Further,

$$C_{v,p} = C_s \times P$$

Finally:

$$BCF_{above-ground,fw/dw} = \frac{C_{v,sa}}{C_s} = S + G + P \qquad BCF_{root,fw/dw} = \frac{C_{v,s}}{C_s} = R$$

$$K_{pl} = \left(\left(S + G + P\right) \times f_{leaf}\right) + \left(R \times f_{root}\right)$$

where S, G, P, and R are the bioconcentration factors for the soil to above-ground vegetables, gas to above-ground vegetables, particle phase to above-ground vegetables, and soil to root pathway respectively. They all have the dimension: (mg/kg fw plant)/(mg/kg dw). In Table F4, as an example, calculated bioconcentration factors S, G, P, R [(mg/kg fw)/(mg/kg dw)] and S/dw, G/dw, P/dw and R/dw [(mg/kg dw)/(mg/kg dw)] for silty soil are compared with measured $BCFs$ (values used in Vlier-Humaan). From this table, it can be seen that the modelled concentration in the above-ground vegetables is mainly governed by the calculated particle bound deposition bioconcentration factor (P).

Table F4: Calculated S, G, P, R (mg/kg fw)/(mg/kg dw), S/dw, G/dw, P/dw and R/dw (mg/kg dw)/(mg/kg dw) for PAHs for silty soil, and comparison with measured BCFs (mg/kg dw)/(mg/kg dw).

PAH	S	G	P	(S+G+P)	R	S/dw	G/dw	P/dw	R/dw	BCF_{above}	BCF_{root}
Acenaphthene	$1,40 \times 10^{-7}$	$1,47 \times 10^{-8}$	0.051	0.051	0,00285	$1,17 \times 10^{-6}$	$1,23 \times 10^{-7}$	0.425	0,0141	2.32	2.32
Acenaphthylene	$2,26 \times 10^{-7}$	$2,31 \times 10^{-8}$	0.051	0.051	0,00469	$1,88 \times 10^{-6}$	$1,92 \times 10^{-7}$	0.425	0,0232	2.32	2.32
Anthracene	$3,70 \times 10^{-8}$	$6,03 \times 10^{-9}$	0.051	0.051	0,00108	$3,08 \times 10^{-7}$	$5,02 \times 10^{-8}$	0.425	0,0054	0.022	0.002
Benzo(a)anthracene	$4,04 \times 10^{-7}$	$2,19 \times 10^{-9}$	0.051	0.051	0,00284	$3,37 \times 10^{-6}$	$1,82 \times 10^{-8}$	0.425	0,0141	0.007	0.015
Benzo(a)pyrene	$9,59 \times 10^{-8}$	$6,95 \times 10^{-10}$	0.051	0.051	0,00170	$7,99 \times 10^{-7}$	$5,79 \times 10^{-9}$	0.425	0,0084	0.002	0.012
Benzo(b)fluoranthene	$7,85 \times 10^{-8}$	$2,09 \times 10^{-9}$	0.051	0.051	0,00169	$6,54 \times 10^{-7}$	$1,74 \times 10^{-8}$	0.425	0,0084	0.014	0.005
Benzo(g,h,i)perylene	$2,27 \times 10^{-8}$	$1,57 \times 10^{-11}$	0.051	0.051	0,00158	$1,89 \times 10^{-7}$	$1,31 \times 10^{-10}$	0.425	0,0078	0.004	0.011
Benzo(k)fluoranthene	$9,62 \times 10^{-8}$	$6,07 \times 10^{-9}$	0.051	0.051	0,00361	$8,02 \times 10^{-7}$	$5,06 \times 10^{-8}$	0.425	0,0179	0.003	0.015
Chrysene	$3,92 \times 10^{-7}$	$1,00 \times 10^{-8}$	0.051	0.051	0,00336	$3,27 \times 10^{-6}$	$8,35 \times 10^{-8}$	0.425	0,0166	0.008	0.013
Dibenzo(a,h)anthracene	$8,43 \times 10^{-8}$	$2,97 \times 10^{-11}$	0.051	0.051	0,00262	$7,03 \times 10^{-7}$	$2,48 \times 10^{-10}$	0.425	0,0130	0.0003	0.0005
Fluoranthene	$2,53 \times 10^{-7}$	$7,23 \times 10^{-9}$	0.051	0.051	0,00131	$2,11 \times 10^{-6}$	$6,02 \times 10^{-8}$	0.425	0,0065	0.029	0.023
Fluorene	$6,41 \times 10^{-8}$	$5,27 \times 10^{-9}$	0.051	0.051	0,00097	$5,34 \times 10^{-7}$	$4,39 \times 10^{-8}$	0.425	0,0048	0.005	0.009
Indeno(1,2,3-cd)pyrene	$6,11 \times 10^{-9}$	$2,13 \times 10^{-12}$	0.051	0.051	0,00011	$5,09 \times 10^{-8}$	$1,78 \times 10^{-11}$	0.425	0,0005	0.0001	0.0002
Naphthalene	$6,19 \times 10^{-8}$	$6,98 \times 10^{-9}$	0.051	0.051	0,00162	$5,16 \times 10^{-7}$	$5,82 \times 10^{-8}$	0.425	0,0080	2.92	2.92
Phenantrene	$2,16 \times 10^{-7}$	$1,18 \times 10^{-8}$	0.051	0.051	0,00191	$1,80 \times 10^{-6}$	$9,81 \times 10^{-8}$	0.425	0,0095	0.041	0.031
Pyrene	$2,97 \times 10^{-7}$	$8,69 \times 10^{-9}$	0.051	0.051	0,00149	$2,48 \times 10^{-6}$	$7,24 \times 10^{-8}$	0.425	0,0074	0.011	0.021

6. DOMINANT PROCESS IN AIR-TO-PLANT TRANSFER

6.1 GAS-PARTICLE PARTITIONING

At usual ambient temperatures, PAHs exists in the atmosphere as a gas, or adsorbed onto solid particles, or as particles (Finlayson-Pitts & Pitss, 2000). Lighter PAHs (lower molecular weight, fewer rings) are more likely to be in the vapour phase rather than in the particular phase. As a rule of thumb, it can be stated that naphthalene, a two-ring species is found in the vapour phase, PAHs with three to five rings are present both in the gas phase and onto particles, and PAHs with five or more rings are almost exclusively present in the particulate phase. In Table F5 the PAHs under consideration are presented with their respective partitioning behaviour at ambient temperature.

Table F5: Partitioning behaviour of PAHs at usual ambient temperature and dominant route in air-to-plant transfer.

Chemical	# rings	Partitioning behaviour in air*	log K_{OA}	Dominant route in air-to-plant transfer**
Acenaphthene	3	G	6.18	Equilibrium partitioning
Acenaphthylene	3	G	5.99	Equilibrium partitioning
Anthracene	3	G	6.69	Equilibrium partitioning
Benzo(a)anthracene	4	P	9.67	Kinetically limited gaseous deposition
Benzo(a)pyrene	5	P	9.85	Kinetically limited gaseous deposition
Benzo(b)fluoranthene	5	P	9.33	Kinetically limited gaseous deposition
Benzo(g,h,i)perylene	6	P	11.57	Particle bound deposition
Benzo(k)fluoranthene	5	P	9.19	Kinetically limited gaseous deposition
Chrysene	4	P	8.83	Kinetically limited gaseous deposition
Dibenzo(a,h)anthracene	5	P	11.6	Particle bound deposition
Fluoranthene	4	G	8.22	Equilibrium partitioning
Fluorene	3	G	6.52	Equilibrium partitioning
Indeno(1,2,3-cd)pyrene	6	P	11.35	Particle bound deposition
Naphthalene	2	G	5.08	Equilibrium partitioning
Phenantrene	3	G	7.28	Equilibrium partitioning
Pyrene	4	G	8.07	Equilibrium partitioning

*: on the basis of measurements of PAHs in air in Australia (Department of Environment and Conservation, 2004), the UK (September-July; Smith et al., 2001) and Flanders (summer, winter; Van De Weghe et al., 2005); compounds found totally/almost exclusively in the gas phase (G) or particle phase (P); remark: the partitioning behaviour is influenced by the ambient temperature and the total concentration of suspended particles in air; the partitioning behaviour as presented in the table is indicative and mainly corresponds with the partitioning behaviour during the (warmer) growing period of the vegetation (vegetables).
**: implicated by McLachlan et al. (1995); McLachlan et al. (1999).

It must be remarked that gas-particle partitioning of individual PAHs is influenced by several factors such as ambient temperature and total particle concentration in air.

6.2 ESTIMATING GAS-PARTICLE PARTITIONING (IMMISSION)

If only the sum of the gas and particle phase concentrations in air (immission) as well as the *TSP* (total suspended particles in air) are known, the individual components ($C_{g,a}$ and $C_{p,a}$) can be estimated by using following equations:

$$\phi = \frac{C_{p,a}}{C_{p,a} + C_{g,a}}$$

$$K_P = \frac{C_{p,a}/TSP}{C_{g,a}}$$

$$\phi = \frac{C_{p,a}}{C_{p,a} + C_{g,a}} = \frac{K_P \times TSP}{(K_P \times TSP) + 1}$$

where:

Φ: fraction of the compound adsorbed to aerosol particles [-]
K_P: temperature-dependent gas-particle partition coefficient [m³/µg];
TSP: total suspended particles in air [µg/m³].

For PAHs Finizio et al. (1997) derived following correlation between K_P and K_{OA}:

$\log K_P = 0.79 \log K_{OA} - 10.01$ (R²=0.97)

6.3 AIR-PLANT PARTITIONING

To determine the dominant route in the gas/particle partitioning of PAHs for use in the air-to-plant transfer model, the framework of McLachlan (1999) can be used, together with literature data on both measured concentrations of PAHs in air and vegetables.

The framework of McLachan is based on the rationale that both dry gaseous deposition and particle-bound (wet and dry) contribute to the deposition of semi-volatile organic compounds. Which one is actually controlling the deposition, depends on the K_{OA} of the compound, the plant species and the prevailing temperature and wind. Under regular environmental conditions, the deposition pathway to a given plant species is governed by the K_{OA}, in which the controlling process goes from equilibrium partitioning to kinetically limited dry gaseous deposition to particle-bound deposition with increasing K_{OA}.

Compounds with low K_{OA} values are relatively volatile and do not sorb to atmospheric particles to meaningful extent. Hence they are deposited primarily via gaseous diffusion. These compounds also have comparatively low vegetation/air partition coefficients and tend to approach an equilibrium rapidly.

For compounds with intermediate K_{OA} values, there is tendency to partition out of the gas phase onto particles, but the vegetation/air coefficient becomes so large that a partitioning equilibrium between the vegetables and the gas phase is not approached during the lifetime of the vegetables. This is the case of kinetically limited dry gaseous deposition.

For compounds with high K_{OA} values, there is a strong tendency to partition out of the gas phase onto particles, and particle-bound deposition (both wet and dry) becomes the

dominant process. This means that the levels in vegetables are dominant by particle-bound deposition.

Experimental data from Böhme et al. (1999) and McLachlan et al. (1999) corroborate the mechanisms underlying the interpretative framework. For compounds with log K_{OA} < 8.8 (e.g. phenanthrene, fluoranthene, and pyrene), equilibrium partitioning was the determining process determining plant uptake. For compounds with 8.8 < log K_{OA} < 11 (e.g. chrysene), plant uptake was determined by kinetically limited gaseous deposition. Finally, for compounds with log K_{OA} > 11 (e.g. the 5-6 ring PAHs), particle-bound deposition dominated.

6.4 CONSEQUENCES FOR USING THE MODELLED BIOCONCENTRATION FACTORS IN S-RISK

Calculated *BCFs* (Table F4) for the different soil-to-plant and air-to-plant pathways suggest that the concentration of any given PAH in the above-ground vegetables is mainly due to particle bound deposition of that compound. For the heavier PAHs which - according to the framework of McLachlan (1999) - the dominant route in the air-to-plant transfer is particle bound deposition, this will most likely be the case (Table F5: benzo(g,h,i)perylene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene).

However, for the lighter PAH compounds who are totally or almost exclusively found in the gas phase and for which the dominant route in the air-to-plant is equilibrium partitioning, the modelled concentration in the above-ground vegetables will be overestimated if particle bound deposition is included. This will be the case for acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, naphthalene, phenanthrene and pyrene (Table F5).

For the intermediate weight PAHs, another factor complicates the unrestricted use of the modelled bioconcentration factor *P* in the overall estimation of the concentration in above-ground vegetables. Following McLachlan's framework, the dominant route in air-to-plant transfer of PAH compounds with 8.8 < log K_{OA} < 11 is kinetically limited gaseous deposition. This means that dry gaseous deposition is still the dominant uptake process but the storage capacity of the vegetables for the chemical is so high that an equilibrium is not approached over the time of exposure. This means that *P* would theoretically approach zero (or at least: *G* >> *P*). However, measured data of gas and particle phase concentrations of these PAHs (benzo(a)antracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene and chrysene) suggest that these compounds are almost exclusively found in the particle phase.

Another remark is that in literature volatilization of (semi) volatile organic compounds from soil is generally not accounted for: concentrations of these substances are mainly related to ambient gas/particle phase concentrations (immission data). It is assumed that ambient gas phase concentrations of PAHs, especially in urban regions will probably be higher than the gas phase concentrations originating from the contamination in soil.

Given the above mentioned remarks, it is concluded that in S-RISK the use of measured bioconcentration factors in the estimation of the concentration in vegetables should be favoured.

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APPENDIX D: DERMAL ABSORPTION FACTORS FOR PAHS AND CADMIUM – ADJUSTMENT OF TOXICITY FACTORS

1. Introduction

In this appendix, some aspects on dermal exposure to soil and dust are elaborated. On the basis of this elaboration, recommendations for S-RISK are given.

There are various ways to calculate dermal exposure in risk assessment methodologies. For example, mass transfer or permeability coefficients, percent absorbed values or relative absorption factors can be used. The choice of the approach is to a large extent determined by the exposure pathway (aquous media, air, soil, etc.).

2. US-EPA

Dermal contact with contaminants can result in direct toxicity at the site of application and/or contribute to systemic toxicity via percutaneous absorption. In the absence of dermal toxicity factors, US-EPA has devised a simplified paradigm for making route-to-route (oral-to-dermal) extrapolations for systemic effects. Primarily, it accounts for the fact that most oral reference doses and slope factors are expressed as the amount of substance administered per unit time and body weight, whereas exposure estimates for the dermal pathway are expressed as absorbed dose. The process utilizes the dose-response relationship obtained from oral administration studies and makes an adjustment for absorption efficiency to represent the toxicity factor in terms of absorbed dose.

To characterize risk from the dermal exposure pathway, adjustment of the oral toxicity factor to represent an absorbed rather than administered dose is necessary. This adjustment accounts for the absorption efficiency in the critical study, which forms the basis of the *RfD*. The magnitude of toxicity factor adjustment is inversely proportional to the absorption fraction in the critical study. In practice, an adjustment in oral toxicity factor is recommended when the following conditions are met: (i) the toxicity value derived from the critical study is based on an administered dose (e.g. delivery in the diet or by gavage) in its study design; (2) a scientifically defensible database demonstrates that the gastrointestinal (GI) absorption of the chemical in question, from a medium (e.g. water, feed) similar to the one employed in the critical study, is significantly less than 100% (e.g. <50%). A cutoff of 50% GI absorption is recommended to reflect the intrinsic variability in the analysis of absorption studies. If these conditions are not met, a default value of complete (i.e. 100%) oral absorption may be assumed, thereby eliminating the need for oral toxicity-value adjustment. Once the criteria for adjustment have been met and a specific ABS_{GI} value has been identified, a toxicity factor that reflects the absorbed dose can be calculated from the oral toxicity values.

$$SF_{ABS} = \frac{SF_o}{ABS_{GI}}$$

where:

SF_{ABS}:   absorbed slope factor [(mg/kg.d)$^{-1}$];
SF_o: oral slope factor [(mg/kg.d)$^{-1}$];
ABS_{GI}:   fraction of contaminant absorbed in GI tract [-] in the critical toxicity study.

$$RfD_{ABS} = RfD_o \times ABS_{GI}$$

where

RfD_{ABS}: absorbed reference dose [mg/kg.d];
RfD_o: reference dose, oral [mg/kg.d];
ABS_{GI}:   fraction of contaminant absorbed in GI tract [-] in the critical toxicity study.

For PAH-compounds, no adjustment of the slope factor is recommended by US-EPA. On the other hand, the *RfD* of Cd should be adjusted (US-EPA, 2004).

The default dermal absorption assumptions based on US-EPA Region 4 (US-EPA, 2000c) guidance recommend a value of 0.01 for organics and 0.001 for inorganics. The technical basis for these values is not explained in the guidance other than to state that they include considerations of reduced dermal absorption of chemicals from a soil matrix. There is evidence to indicate that the dermal absorption of some chemicals may exceed these defaults, and specific examples are provided in the US-EPA Dermal Assessment guidance (US-EPA, 2000b). However, based on the wide range of absorption factors indicated by the studies of Wester et al. (1990) and Yang et al. (1989), and at that time (1992) the need of further research on the bioavailability of the compound in soil, the US-EPA did not attempt to recommend a range of values for the percutaneous absorption of benzo(a)pyrene in its Dermal Exposure Assessment handbook (US-EPA, 1992).

In the Risk Assessment Guidance for Superfund (US-EPA, 2004), the dermal absorption fraction from soil for benzo(a)pyrene and other PAHs is 0.13, based on the experimental mean value reported by Wester et al. (1990). This value has been determined to be applicable using the Superfund default human exposure assumptions, and are average absorption values.

For inorganics, the speciation of the compound is critical to the dermal absorption and there are too little data to extrapolate a reasonable default value (US-EPA, 2004). However, for Cd, a recommended dermal absorption fraction of 0.001 is given, on the basis of US-EPA (1992) and the study of Wester et al. (1992a). Since the gastrointestinal absorption is significantly less than 100% (US-EPA reports an ABS_{GI} of 2.5% and 5% for Cd in diet and water respectively), the *RfD* should be adjusted.

3. RAIS

The dermal absorption factors, noted in RAIS, the Risk Assessment Information System, are derived from US-EPA (1995, 2004a). A dermal absorption factor of 0.001 is reported for

Cd. The PAHs under consideration in this document all have an dermal absorption factor of 0.13, except acenaphthylene and phenanthrene; their dermal absorption factor is 0.01.

4. MDEP

The relative absorption factors (*RAFs*) of the Massachusetts Department of Environmental Protection (MDEP) address two major issues (MDEP, 1994): (i) the absorption efficiency for the chemical via the route and medium of exposure for which the standard is being developed, and (ii) the absorption efficiency for the route and medium of exposure in the experimental study which is the basis of the Reference Dose or Cancer Slope Factor for the chemical in question. Or, in other words, the relative absorption factor is used to account for difference in the absorption of a compound under assumed exposure conditions at the site (exposure route and matrix) relative to the absorption of the chemical under experimental conditions upon which the dose-response value is based. *RAFs* are used in stead of absorption efficiencies to ensure that the exposures evaluated at the disposal site are comparable to the toxicity information identified in the literature. The *RAF* is calculated as (MDEP, 1992, 1995):

$$RAF = \frac{Absorption\ Efficiency_{SITE\ route\,/\,medium\ of\ exposure}}{Absorption\ Efficiency_{STUDY\ route\,/\,medium\ of\ exposure}}$$

For example, the RAF specific to the cancer risk evaluation of benzo(a)pyrene for soil dermal contact exposures is determined by the ratio:

$$RAF = \frac{Absorption\ Efficiency_{benzo(a)pyrene\ from\ soil\ via\ dermal\ contact}}{Absorption\ Efficiency_{benzo(a)pyrene\ via\ oral\ exposure}} = \frac{0.18}{0.91} = 0.2$$

An absorption efficiency (or absorption factor) which does not consider derivation of the toxicity values is not considered an *RAF*.

In the absence of readily available site-specific and chemical-specific information, default absorption efficiencies are used. For PAHs (semi-volatile organics), the default absorption efficiency for the soil-dermal site route is 0.17. The absorption efficiencies that correspond to the various ways in which the chemical was administrated in the study on which the toxicity value is based are: food: 0.95, gavage (oil): 0.91, injection: 1, dermal contact: 0.14 (MDEP, 1995).

5. S-EPA

The model for exposure due to dermal contact with soil and dust is based on the model used in CSOIL.

The reference soil concentration for the dermal pathway, C_{du} [mg/kg] is calculated as:

$$C_{du} = \frac{TRV}{f_{du} \times R_{du}} \times 10^6$$

where

TRV: toxicological reference value [mg/kg.d];

f_{du}:   substance specific relative absorption factor for dermal uptake [-];

R_{du}:   average daily dermal exposure [mg/kg.d], i.e. long-term dermal exposure for chronic exposure for non-genotoxic substances and integrated lifetime dermal exposure for genotoxic substances.

Substance specific relative absorption factors for dermal uptake, f_{du}, were taken from MDEP's RAFs. A soil exposure of 0.51 mg/cm² is used for children as well as adults. No rationale for this choice is given. The value 0.51 mg/cm² is the amount of soil or dust on the skin of children during outdoor activities (ECETOC, 1992), implicating that indoor exposure to soil or dust is omitted. However, in MDEP (1994), this value is presented -on the basis of Hawley (1985)- as the amount soil in contact with the skin for both children and adults, for the days when the receptor is exposed both indoors and outdoors. Exposure to adults is quantified here as it is assumed that all ages have the opportunity for contact with the soil through playing or gardening (MDEP, 1994).

The exposure time on land with less sensitive land-use is assumed to be a third of the exposure time on land with sensitive land-use. Input values for R_{du} are given in Table G1. No rationale is given for the deviation from the ECETOC/CSOIL methodology.

Table G1: Dermal contact parameters used by S-EPA.

Parameter	Land with sensitive land-use (KM)	Land with less sensitive land-use (MKM)
Long-term dermal soil exposure per unit body weight – child [mg/kd/d]	20	7
Integrated lifetime dermal soil exposure [mg/kg/d]	3	1

6. Recommendations for S-RISK

In the risk assessment procedure, it is necessary to take into account the possible differences in expression of the site exposure estimates and the toxicity values for comparison. In case of dermal exposure assessment, most procedures result in an estimate of the absorbed dose. Since dermal toxicity values are mostly lacking, the use of oral toxicity values (on the basis of intake of food, drinking water, etc. contaminated with chemicals) as a surrogate is widespread. However, since most *TDI*s or *RfD*s, and some slope factors are expressed as the amount of substance administered per unit time and unit body weight, whereas exposure estimates for the dermal route of exposure are eventually expressed as absorbed doses, adjustment of the oral toxicity value from an administered to an absorbed dose may be necessary.

This adjustment can be achieved by defining a *RAF* (MDEP), i.e. adjusting the absorption efficiency, or by adjusting the *TDI*/*RfD* or slope factor itself (US-EPA). However, in the ultimate risk assessment, both procedures have the same result.

US-EPA recommends not to adjust the slope factor for PAH-compounds since their gastrointestinal absorption is not significantly less than 100%. The *RfD* of Cd however should be adjusted (US-EPA, 2004).

Taking the recommendations of US-EPA into account, the following procedure for the dermal risk assessment in S-RISK is proposed:
- for PAHs: f_{du} should be replaced by the US-EPA dermal absorption factors (Table G2), i.e. no adjustment for absorption efficiency should be made;
- for cadmium: US-EPA reports a ABS_{GI} of 2.5% and 5% for Cd in diet and water respectively; since the oral *RfD* used in S-RISK is based upon the intake of contaminated food, f_{du} can be defined as the ratio 0.001/0.025, i.e. 0.04.

Table G2: Dermal (relative) absorption factors of PAHs (and Cd).

Substance	Dermal absorption factor		Relative absorption factor			Recommended value for f_{du}
	US-EPA	RAIS	MDEP		S-EPA	S-RISK
			Non-carc.	Carc.		
Cadmium	0.001	0.001 (R1)	0.14	NC	0.14 (S1)	0.04
Acenaphtene	0.13	0.13 (R1)	0.20	NC	ND	0.13
Acenaphtylene	0.13	0.01 (R2)	0.18	NC	0.18 (S1)	0.13
Anthracene	0.13	0.13 (R1)	0.29	NC	ND	0.13
Benzo(a)anthracene	0.13	0.13 (R1)	0.18	0.20	0.20 (S1)	0.13
Benzo(a)pyrene	0.13	0.13 (R1)	0.18	0.20	0.20 (S1)	0.13
Benzo(b)fluoranthene	0.13	0.13 (R1)	0.18	0.20	0.20 (S1)	0.13
Benzo(g,h,i)perylene	0.13	0.13 (R1)	0.18	NC	0.18 (S1)	0.13
Benzo(k)fluoranthene	0.13	0.13 (R1)	0.18	0.20	0.20 (S1)	0.13
Chrysene	0.13	0.13 (R1)	0.18	0.20	0.20 (S1)	0.13
Dibenzo(a,h)anthracene	0.13	0.13 (R1)	0.08	0.09	0.09 (S1)	0.13
Fluoranthene	0.13	0.13 (R1)	0.20	NC	0.20 (S1)	0.13
Fluorene	0.13	0.13 (R1)	0.20	NC	0.20 (S1)	0.13
Indeno(1,2,3-cd)pyrene	0.13	0.13 (R1)	0.18	0.20	0.18 (S1)	0.13
Naphthalene	0.13	0.13 (R1)	0.10	NC	0.10 (S1)	0.13
Phenanthrene	0.13	0.01 (R2)	0.18	NC	0.18 (S1)	0.13
Pyrene	0.13	0.13 (R1)	0.20	NC	0.20 (S1)	0.13

ND: no data is given for this substance in Naturvårdsverket (1996b);
(R1): US-EPA (2004a);
(R2): US-EPA (1995);
(S1): MDEP (1994);
NC: not calculated.

APPENDIX E: ESTIMATING WIND-BLOWN SOIL DUST EMISSIONS

1. Introduction

Wind-blown emissions of fine dust (particle size < 10 µm) from soils is essentially caused by the bombardment of the soil surface by coarse sand particles having a size in the range 50-500 µm. At sufficiently high wind speeds, sand particles are extracted from the surface, and follow a more or less ballistic trajectory, extracting finer particles from the soil on impaction. Owing to their higher friction (relative to their mass), the fine particles remain airborne for a relatively long time.

The simulation of soil dust generally is done based on a two-step procedure (Shao, 2000; Raupach and Lu, 2004), as follows:
- firstly, the saltation flux is calculated, i.e., the flux of sand particles following the flow (hence mainly horizontally), integrated over the vertical direction;
- subsequently, the vertical flux of fine soil dust is calculated as a function of the saltation flux, and of certain soil properties.

Since the saltation flux depends on (1) wind speed and (2) soil moisture in a strongly non-linear fashion, it is not feasible to employ annual average values for these quantities in the calculation of the emission fluxes. Daily and even sub-daily (for the wind) fluctuations have to be accounted for.

In the following sections, a relatively simple method is presented for the calculation of wind-blown soil dust emissions. Particular attention is devoted to represent the effect of built-up areas (residential areas, industrial sites) on these emissions. The method relies on daily meteorological data and on site characteristics such as soil texture and obstacle density and height. Meteorological data are taken from the GLOBALSOD database (*Global Summary of the Day*, available from ftp://ftp.ncdc.noaa.gov/pub/data/globalsod/), which contains daily average observed values of wind speed, temperature, precipitation, among other meteorological parameters, for thousands of stations world-wide, for the period 1994-now.

2. The model

Several models exist in the literature to calculate saltation fluxes (Q, units of $m^{-1} s^{-1}$). Shao (2000) provides an overview of parametrisations in his Table 6.1. Here, the formulation by White (1979) is chosen, as one of the most recent and widely used,

$$Q = c \frac{\rho}{g} u_*^3 \left(1 - \frac{u_{*t}}{u_*}\right)\left(1 + \frac{u_{*t}}{u_*}\right)^2 \qquad \begin{array}{l} u_* > u_{*t} \\[8pt] u_* < u_{*t} \end{array} \qquad (1)$$
$$= 0$$

where u_* is the friction velocity (a scale for the turbulent velocity fluctuations in the atmosphere near the ground, see below) and u_{*t} a threshold value below which no saltation occurs. Furthermore, $c \approx 2.6$ is a unitless constant, ρ is air density, and $g = 9.81$ m s^{-2} is the

gravitational acceleration at the Earth's surface. [Remark that (1) differs from what is sometimes used in the literature. Indeed, the original paper by White (1979) contained a typographic error, as mentioned by Baas (2005), which appears to have propagated in certain models.]

The vertical flux of soil dust (F, units of kg m^{-2} s^{-1}) is calculated as a (most of the time linear) function of Q. One of the most recent formulations, also with a firm physical basis, is the model by Lu and Shao (1999). Unfortunately, this model is currently not a practical option owing to the large number of (often unknown) parameters. Therefore, the empirical scheme by Marticorena and Bergametti (1995) is used here:

$$F = \gamma\, Q\, e^{0.308 f_c} , \tag{2}$$

where f_c represents the percentage by mass of clay particles (size < 2 µm) in the soil (see Table H1), and $\gamma = 9.96 \times 10^{-5}$ m^{-1}. Note that, following Marticorena and Bergametti (1995), $f_c < 20$ % is imposed.

Table H1. Soil particle size distribution (by mass) used here for sand, loam, and clay. Pure clay particles are those with a size below 2 µm, PM10 is the fraction of soil particles with a diameter below 10 µm, and dust is considered here to consist of particles with a diameter less than 50 µm, following Marticorena and Bergametti (1995).

			size class	mass fraction (in %)		
				sand	loam	clay
dust	**PM₁₀**	**clay**	**< 2 µm**	2.61	13.70	26.04
			2-10 µm	1.14	9.54	7.95
			10-20 µm	1.24	15.65	7.94
			20-50 µm	5.01	52.12	22.84
			50-100 µm	15.37	5.20	16.68
			100-200 µm	47.69	2.08	14.30
			200-500 µm	24.98	1.14	4.17
			500-2,000 µm	1.74	0.68	0.68

It should be noted that there exists a variety of saltation and dust emission models, and that each of these models may yield quite different results for a given situation, even when detailed site-descriptive and meteorological data are available. Therefore, the method described below can, at best, yield an order-of-magnitude estimate of wind-blown soil dust emissions.

The formulae (1)-(2) are fairly straightforward. The challenge consists rather of:
1. Correctly calculating u_*, accounting for sub-daily variability. Indeed, the daily maximum friction velocity value may substantially exceed the threshold, even when the daily average value does not. This is all the more important when considering that Q displays a very large sensitivity to u_* (cubic dependence).
2. The correct calculation of u_{*t}, which depends on several factors including soil moisture and obstacles (buildings, vegetation, rocks) shielding the underlying soil surface.

In the remainder of this section more details are provided regarding the calculation of u_* en u_{*t}, as well as the precise way the saltation and dust fluxes are calculated.

2.1 Friction velocity

2.1.1 Vertical wind profile

According to Prandtl's theory of the atmospheric surface layer (see, e.g., Garrat, 1992), the variation of the wind speed with height is logarithmic, following

$$u(z) = \frac{u_*}{k} \ln\left(\frac{z - d}{z_0}\right), \tag{3}$$

with $u(z)$ the wind speed at a heigth z above the ground, $k \approx 0.4$ von Kàrmàn's constant, d the displacement height, and z_0 the roughness length. The latter is a measure for the roughness of the surface, and generally has a value of the order of 10 % of the average height of the roughness elements. Typical values range from 0.1-1 mm for a relatively flat bare soil, up to several metres for forests and cities. The displacement height d is of the order of 70 % of the heigth of the roughness elements, and accounts for the uplift of the logarithmic profile over rough terrain. The friction velocity can be obtained by inversion of (3), assuming that the roughness length and wind speed (typically at 10 m height) are known.

It should be noted that (3) is applicable only to situations of neutral hydrodynamic atmospheric stability. No stability effects are taken into consideration here, as this would entail a considerate increase of the complexity of the method, and as the required information (e.g., surface turbulent heat fluxes) are generally not available in routine meteorological observations. Furthermore, most dust emissions only occur at high wind speeds, i.e., low magnitudes of the Richardson number (Garratt, 1992) hence close to neutral stability. For instance, atmospheric stability classes used in Gaussian dispersion models become are classified as neutral whenever the 10-m wind speed exceeds 6 m s^{-1}, except for strong insolation, in which case the stability is classified as slightly unstable (see, e.g., Godish, 2004).

For most types of surface extensive information is available regarding the roughness length values. For built-up areas, however, roughness is calculated using geometric parameters describing the site (e.g., building height), so as to allow some flexibility in the definition of the site characteristics, and also to ensure consistency with the parametrisation of the building shielding effect (see below). The roughness length is calculated according the expressions developed by Raupach (1995), also see Mestayer and Bottema (2002), which are based on the *frontal area index* λ. This quantity is defined as the area of the roughness elements in a direction perpendicular to the flow, divided by the surface area occupied by the elements. The roughness formulation then reads as follows:

$$z_0 = (h - d)\exp\left[-\frac{ku(h)}{u_*} - \psi_r\right], \tag{4}$$

with h the building height, $\psi_r = 0.193$ a correction for the urban roughness sublayer, and

$$\frac{d}{h} = 1 - \frac{1 - \exp\left(-\sqrt{2C_{d1}\lambda}\right)}{\sqrt{2C_{d1}\lambda}}$$

$$\frac{u_*}{u(h)} = \sqrt{C_{ds} + 0.5\lambda C_{dh}}, \tag{5}$$

where $C_{d1} = 7.5$, and C_{ds} and C_{dh} are, respectively, the substrate surface drag and the unit obstacle drag coefficients estimated at level $z = h$. For buildings, $C_{dh} = 0.6$ is a representative value. The drag coefficient for the substrate surface (i.e., in between the buildings) is given by

$$C_{ds} = \left[k/\ln\left(h/z_{0g}\right)\right]^2, \tag{6}$$

with z_{0g} the aerodynamic roughness length of the substrate surface without the building obstacles, typically having a value of 0.1-1 mm for flat soil surfaces, and 1-10 cm for low vegetation.

For residential areas, a value of $\lambda \sim 0.12$ is used here as, for low enough values of the substrate surface roughness, it yields $z_0 \sim 0.6$ m for buildings 10 m high (also a value representative for residential areas), which is the value advocated by Wieringa (1993) for residential areas.

2.1.2 At-station versus site-specific friction velocity

Unless site-specific data (e.g., from a detailed experimental campaign) are available, use has to be made of meteorological data from routine meteorological stations. For most areas in Europe, the GLOBALSOD database contains data from stations at distances of a few tens of kilometres or less from any site of interest. However, the terrain characteristics at the meteorological station may very well be completely unrepresentative for the conditions at the site of interest. For instance, meteorological stations that are operated according the guidelines of the World Meteorological Organisation (WMO) are most of the time installed on terrain with a roughness length of the order of a centimetre. When the site for which a dust emission estimate is required has a different roughness length, e.g., in case of an industrial or urban site, it is not correct to simply apply the station values of wind speed to the site of interest.

Instead, use is made of theoretical-empirical knowledge regarding the effect of roughness transitions on turbulence, more specifically on the friction velocity. It can be shown (see, e.g., Kaimal and Finnigan, 1994) that the ratio of the friction velocities over smooth versus rough terrain is given by

$$\frac{u_*}{u_{*,stn}} \approx 1 - \frac{M}{\ln\left(\delta_i/z_0\right)}. \tag{7}$$

In this expression, $u_{*,stn}$ is the friction velocity for the meteorological station. This quantity can be derived from (3), using measured wind speed at 10 m and assuming a roughness length of $z_{0,stn} \approx 0.01$ m (unless of course detailed information regarding the roughness at the meteorological station is available, in which case a more precise value may be used). The quantity z_0 is the roughness length of the study site, and δ_i is the depth of the so-called internal boundary layer, which develops whenever an air mass encounters a surface with different properties, all the air below the height δ_i (in principle) being in equilibrium with the surface. An often used empirical formula is

$$\frac{\delta_i}{z_0} \approx A_1 \left(\frac{x}{z_0} \right)^n ,$$ (8)

with $n \approx 0.8$ an empirical constant and $A_1 \approx 0.75 + 0.03M$, and $M \equiv \ln(z_{0,stn} / z_0)$. The variable x is the fetch, i.e., the upwind distance to the roughness transition. Note that for very small fetch values, the ratio defined in (7) may become unrealistically large, tending to $+\infty$ for $x \rightarrow 0$. In order to avoid this situation, the fetch is limited so as not to exceed the maximum friction-velocity ratio obtained from experiments, which suggest an upper bound of the order of 2.5 for a smooth-rough transition involving roughness lengths differing by two orders of magnitude.

2.1.3 Sub-daily wind speed variability

In the method described here use is made of the daily average wind speed, which is available in the GLOBALSOD database. However, as mentioned above, within the course of a day significant wind speed fluctuations occur which, given the sensitivity of (1) to wind speed, has to be accounted for in some way. Fortunately, the GLOBALSOD database also contains the maximum sustained wind speed, which is the highest 10-minute average wind speed of the day.

In order to exploit this additional information to the best extent, a statistical analysis was made of detailed time series of wind speed observed at the instrumented 114-m meteorological tower at the Vito-SCK/CEN domain. The tower is located at a longitude of 5.0887° and a latitude of 51.2186° (decimal degrees). The terrain around the tower is mainly characterised by the presence of pine trees (height around 20 m) and medium-size buildings (most are lower than the trees). Wind speed is measured at 24, 48, 69, 78, and 114 m (these measurement heights are with respect to the forest floor). The measurements are averaged and archived over 10-minute periods. Data of the 48-m level were used to estimate the daily wind speed distribution function, using the mean and maximum sustained wind speed values.

Wind speed (denoted V) data were collected for one month (May 2004), and the cumulative distribution function was plotted for every individual day of this month, with obviously a rather large scatter on the displayed data. In order to reduce the variability between the different days, the goal being to derive a 'universal' wind distribution function, the wind speeds of a given day were normalised with respect to their daily mean values. However, after this operation there was still a large variability, caused by daily differing ratio's of

maximum (V_{max}) and mean (V_0) wind speed values. In order to tackle this, the following transformation was applied to the observed wind speed values:

$$\frac{V}{V_0} \rightarrow \left(\frac{V}{V_0}\right)^{\alpha},$$ (9)

with the requirement that $(V_{max}/V_0)^{\alpha} = 2$ (forcing all transformed values to have a common maximum), thus implying

$$\alpha = \frac{\ln 2}{\ln(V_{max}/V_0)}.$$ (10)

Daily cumulative probability distribution functions (PDF's) for wind speed were then plotted as a function of $(V_{max}/V_0)^{\alpha}$ (Figure H1), showing that the daily cumulative PDF's appear to collapse onto a single curve, though with quite some remaining scatter. It was subsequently found that the common curve (thick line in Figure H1) could be well represented by a Weibull distribution function, as follows:

$$P_W = 1 - \exp\left[-\left(\frac{V}{c_W}\right)^{k_W}\right],$$ (11)

with $k_W = 2.8\ \alpha$ (the coefficient '2.8' was obtained by fitting (11) by eye to the data in Figure H1), and

$$c_W = \frac{V_0}{(\ln 2)^{1/k_W}}$$ (12)

where the factor 'ln 2' in the denominator ensures that the PDF is symmetric with respect to the median value, as is apparent from Figure H1..
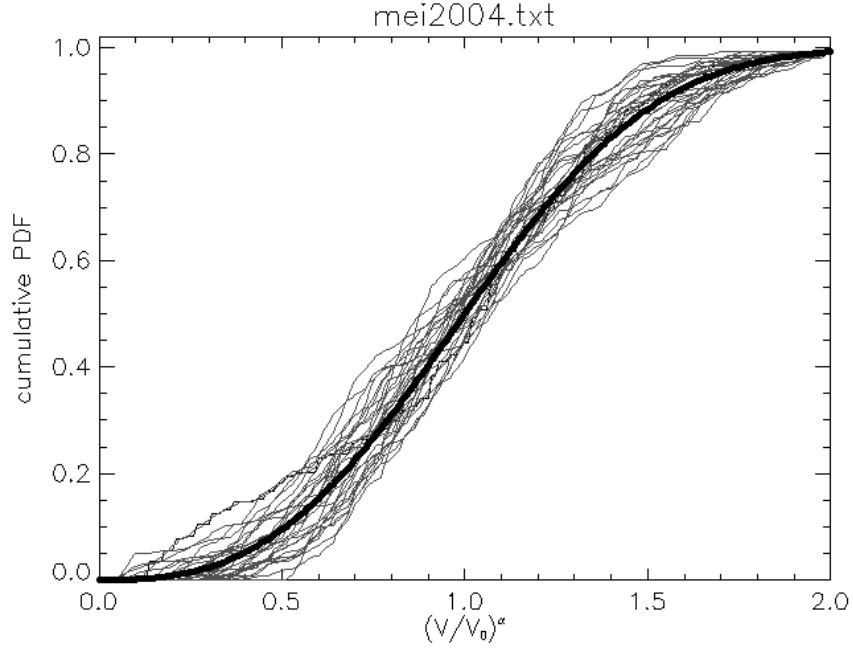
Figure H1. Cumulative probablility distribution function of the observed 10-minute averaged wind speed values from the meteorlogical tower at Mol, for all days of May 2004, each thin curve representing another day. The thick solid line corresponds to the function defined in (11).

It is clear from Figure H1 that (11) exhibits quite a bit of scatter. However, it is also clear that the fitted curve displays both under- and overestimations, so that compensations occur. When repeating the fitting procedure for data from other months from the same site, the PDF fitted those data equally well, thus confirming the universal nature of (11). Nevertheless, care should be taken to extrapolate these data to other sites.

2.2 Threshold friction velocity (u_{*t})

The threshold value of the friction velocity, u_{*t}, which is required in (1), is customarily calculated as

$$u_{*t} = u_{*t0} f_w(\theta_s) f_s(\lambda) \mathrm{K} . \qquad (13)$$

In this expression u_{*t0} represents the minimal threshold value, which applies to an 'ideal' dry soil surface with no obstacles. The functions f (> 1) account for processes that increase the threshold friction velocity above this minimal value, such as soil moisture, shielding of the soil surface by buildings or vegetation, salt content of the soil, among others. The sole functions retained here are those that account for the effects of surface soil moisture (denoted θ_s) and shielding.

2.2.1 Minimal threshold friction velocity (u_{*t0})

Shao en Lu (2000) derived the following expression for the lower limit of the threshold value for saltation of the friction velocity, as a function of saltation particle size:

$$u_{*t0}(d) = \sqrt{a_1\left(\sigma_p g d + \frac{a_2}{\rho d}\right)},$$
(14)

with d the particle size, $a_1 \approx 0.0123$ a unitless constant, $a_2 \approx 3 \times 10^{-4}$ kg s^{-2}, $\sigma_p \approx 2200$ the ratio of the densities of soil particles versus air, and g en ρ having been defined above, see (1).

Expression (14) exhibits a minimum at a certain intermediate particle size (Figure H2). The reason is that very small particles are subject to large cohesion forces, while very large particles are to heavy for saltation. At an intermediate or optimal particle size of the order d_0 ≈ 100 µm the resistance to being extracted from the soil by the wind is at a minimum.
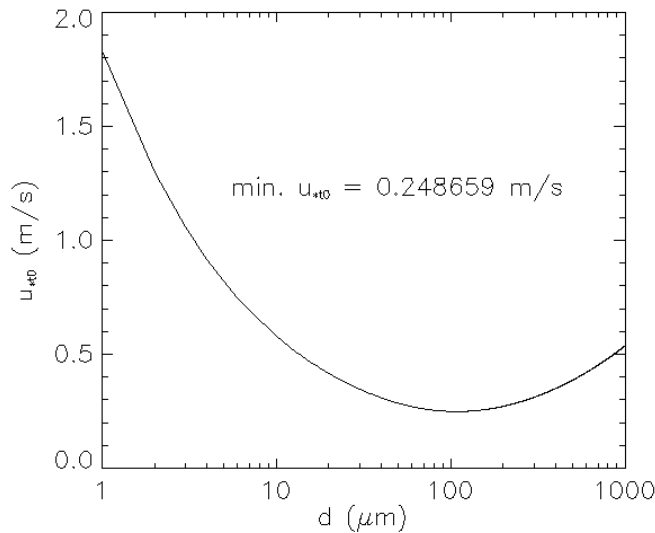
Figure H2. Lower limit of the threshold friction velocity for saltation, as a function of particle size.

In a later section, a saltation flux $Q(d)$ will be calculated per particle size class, using (14) to obtain u_{*t0} as a function of particle size. The total saltation flux will then be calculated as a weighted average, using the particle size distribution function of the soil to determine the weights per size class.

2.2.2 Soil moisture effects (f_w)

In a moist soil the cohesion forces between particles are larger than in a dry soil. As a consequence, a higher friction velocity is required for the particles to get extracted from the soil matrix. Initially, the method used the parametrisation by Fécan *et al.* (1999) to represent moisture effects, but it was found that with this formulation rather wet soils exhibit

relatively low f_w values (~ 3), thus could sustain saltation fluxes even for soils at field capacity, which does not appear very realistic. On the other hand, theoretical considerations suggest that

$$f_w = \left(1 + \frac{h_w}{|\psi|}\right)^{1/2}$$

(15)

[see, e.g., Shao (2000), his Eq. (9.48)], with ψ the matric potential (expressed here as the height of a water column, units of metres), which depends on soil moisture content and soil texture, and h_w an empirical soil-texture dependent coefficient. Since the magnitude of the matric potential of a soil is very much smaller for a moist compared to a dry soil, the effect of soil moisture should be very significant, and yield very high f_w values at field capacity, as required here.

Expression (15) is rather convenient as the matric potential can be calculated whence soil moisture content is known, e.g., using the empirical relations by Clapp end Hornberger (1978):

$$|\psi| = |\psi_s| \left(\frac{\theta_s}{\theta}\right)^b,$$

(16)

with ψ_s the matric potential at saturation, θ volumetric soil water content (units of m^3 m^{-3}), θ_s volumetric soil water content at saturation, and b an empirical texture-dependent coefficient. The coefficient h_w that appears in (15) was determined as a function of soil textural type using observational data presented in Figure 5 of Fécan *et al.* (1999), which represents measured f_w as a function of gravimetric soil moisture. This was done by identifying, from their data, at which value of gravitational soil moisture content (this value is denoted w_0) the function f_w reached a value of $f_{w0} = 2$. Using (15) and (16), one finds by inversion that

$$h_w = (f_{w0}^2 - 1) |\psi_s| \left(\frac{\theta_s}{\theta_0}\right)^b,$$

(17)

where the relation

$$\theta_0 = \frac{\rho_p}{\rho_w} (1 - \theta_s) w_0$$

(18)

allows to convert from gravimetric to volumetric soil moisture content, with $\rho_p \approx 2500$ kg m^{-3} and $\rho_w \approx 1000$ kg m^{-3} representing, respectively, the density of the soil particles and of water. Using all the above, the w_0 values yielding $f_{w0} = 2$ were estimated from Figure 5 of Fécan *et al.* (1999). The resulting soil-type dependent values are given in Table H2.

Table H2. Coefficients representing textural characteristics of certain soils, based on the USDA classification.

textur	$|\psi_s|$	θ_s	K_s	b	a	p	C_{1sat}	C_{2ref}	θ_{fc}	w_0

e	(m)	(-)	$(10^{-6}\,\mathrm{m\,s^{-1}})$	(-)	(-)				(-)	(-)
sand	0.121	0.395	176.0	4.05	0.387	4.	0.082	3.9	0.135	0.03
loam	0.478	0.451	7.0	5.39	0.148	6.	0.191	0.8	0.240	0.08
clay	0.405	0.482	1.3	11.40	0.083	12.	0.342	0.3	0.367	0.15

The difficulty now is to specify the surface soil moisture content. This is problematic indeed as routine measurements of this quantity are relatively rare, and generally not available as station data together with the meteorological parameters. Since, however, soil moisture has a large impact on saltation and dust emissions, a soil water balance was included in the methodology. Models simulating soil hydrology come in different categories, ranging from simple 'bucket'-type models to models that numerically resolve the 3-D Richard's equations of soil water flow. Here, the 'force-restore' method developed by Noilhan and Planton (1989) is used, as it offers a good trade-off between accuracy and computational efficiency. Furthermore, in contrast to a bucket model, it also simulates the water content at the soil surface, which is of paramount importance in the context of wind erosion modelling. The force-restore method consists of prognostic equations for the upper and deep soil water content, as follows:

$$\frac{d\theta_g}{dt} = \frac{C_1}{\rho_w d_1}\left(P - E_g\right) - \frac{C_2}{\tau}\left(\theta_g - \theta_{geq}\right)$$
$$\frac{d\theta_2}{dt} = \frac{1}{\rho_w d_2}\left(P - E_g - E_{tr}\right) - \frac{K_2}{d_2}.$$

(19)

The quantities θ and θ_g represent the volumetric soil moisture content of, respectively, the upper soil and of a 1-m deep layer. P is the precipitation, E_g is evaporation of water at the soil surface, and E_{tr} is evapotranspiration, i.e., water that is extracted by plant roots from the soil column. The coefficients d_1 and d_2 have values of 0.1 m and 1 m, respectively, and τ is the length of a day (86,400 s).

The remaining factors in (19) are defined as follows:

$$\frac{\theta_{geq}}{\theta_s} = \frac{\theta_2}{\theta_s} - a\left(\frac{\theta_2}{\theta_s}\right)^p\left[1 - \left(\frac{\theta_2}{\theta_s}\right)^{8p}\right]$$

$$C_1 = C_{1sat}\left(\frac{\theta_s}{\theta}\right)^{\frac{b}{2}+1}$$

(20)

$$C_2 = C_{2ref}\frac{\theta_2}{\theta_s - \theta_2 + \varepsilon}.$$

The values of the empirical coeffients a, p, C_{1sat} and C_{2ref} are given in Table H2, and $\varepsilon \approx 0.001$ is a small coefficient to ensure that C_2 remains limited at saturation. Finally, the last term of (19) represents drainage at the bottom of the soil model. This is parameterised assuming that the vertikal soil moisture gradient at the bottom disappears, leaving only

gravitational drainage. The latter is expressed using the empirical scheme proposed by Clapp and Hornberger (1978):

$$K_2 = K_s \left(\frac{\theta_2}{\theta_s} \right)^{2b+3}, \tag{21}$$

with values for K_s also being provided in Tabl H2.

The pair of prognostic equations (19) is solved with a simple forward-in-time numerical scheme. For the deep layer a time step of a day is used. The surface soil moisture, however, fluctuating at much shorter time scales, is resolved using a time step of one hour. It was found that such a short time step is required to ensure the numerical stability of the solution.

The evaporation of water from the soil surface, required in (19), is taken proportional to the potential evaporation E_p:

$$E_g = \frac{\theta_g}{\theta_{fc}} E_p, \tag{22}$$

where the coefficient of proportionality is a linear function of the surface moisture content scaled to moisture content at field capacity (θ_{fc}, see Table H2 for values). It is recalled that potential evaporation is (somewhat loosely) defined as the evaporation that would take place if there were no restraints on the water supply. However, the problem of specifying daily surface soil moisture has now been shifted to the problem of specifying the daily potential evaporation, which again is a quantity that is measured routinely only at a very limited number of meteorological stations.

Fortunately, several (semi-)empirical formulas exist in the literature that allow to express E_p as a function of standard meteorological parameters, e.g., using the Penmann formula (see, e.g., Guyot, 1997). Here we employ the still simpler Hargreaves formula (see, e.g., Allen et al., 1998), which was shown by Droogers and Allen (2002) to yield as good results as the more complex Penmann formula:

$$E_p = 0.0023 \times (T_0 + 17.8) \sqrt{T_{max} - T_{min}} \, \frac{R_{TOA}}{L_v}, \tag{23}$$

with T_0 the daily average temperature (in °C), T_{max} and T_{min} the daily maxima and minima temperatures, and $L_v \approx 2.5 \times 10^6$ J kg^{-1} K^{-1} the latent heat of vaporisation for water. R_{TOA} (in MJ day^{-1}) is the daily average insolation at the top of the atmosphere, which can easily and accurately be calculated as a function of latitude and the Julian day (see, e.g., Oke, 1987), as

$$R_{TOA} = \frac{S_0}{\pi} d_r \left[h_0 \sin\phi \sin\delta + \cos\phi \cos\delta \sin h_0 \right], \tag{24}$$

with S_0 = 118.08 MJ day^{-1} the solar constant, δ = 0.409 × sin(2πJ/365-1.39) the solar declination given as a function of the Julian day J, and d_r = 1 + 0.033 × cos(2πJ/365) the relative Sun-Earth distance. The hour angle of sunset is given by h_0 = arccos(-tanϕ tanδ).

2.2.3 Shielding effects (f_s)

Whenever a soil is covered by obstacles such as buildings or vegetation, a shielding effect occurs inhibiting saltation and hence dust emissions. This implies that a higher friction velocity is required to initiate saltation. As was the case for soil moisture, shielding effects are acounted for by introducing a coefficient that depends on the degree of shielding and that increases the threshold friction velocity. In this section, shielding by large obstacles (buildings) is treated first, and the shielding effect of small-scale obstacles (vegetation, pebbles) on the ground is dealt with suvsequently

The function chose heren to represent shielding from buildings stems from Raupach *et al.* (1993), also see Shao (2000), and is expressed as follows:

$$f_h = \sqrt{(1 - m\sigma\lambda)(1 + m\beta\lambda)}, \tag{25}$$

with λ the frontal area index (defined above), σ the 'basal-element to frontal area ratio', β = C_{dh} / C_{dl} the ratio of the aerodynamic drag coefficients of the roughness elements (C_{dh}) versus the underlying soil (C_{dl}) (defined above), and m a tuning coefficient that was estimated by Raupach *et al.* (1993) at $m \approx 0.5$. The parameters appearing in (25) are essentially determined by the geometry and the position of the roughness elements (buildings) on the ground. The parameters σ and λ depend on the geometry of the obstacles. For built-up areas (Plate et al., 2004) one counters typical values of $\sigma \sim 2$ and $\lambda \sim 0.12$.

The shielding effect of the low obstacles (pebbles, short vegetation) on the soil surface in between buildings can, in principle, also be calculated using (25). However, the specification of the frontal area index is then a problem as this is a largely unknown quantity for any given surface. Therefore, use is made of the firmulation by Marticorena and Bergametti (1995), as follows:

$$f_l = \left\{ 1 - \frac{\ln\left(z_{0g}/z_{0s}\right)}{\ln\left[0.35\left(x_0/z_{0s}\right)^{0.8}\right]} \right\}^{-1}, \tag{26}$$

with $x_0 \approx 0.1$ m, and $z_{0s} \approx 10^{-5}$ m the roughness of the soil surface free of 'non-erodible' elements (Marticorena and Bergametti, 1995). Note that the shielding functions for the buildings and for the lower roughness obstacles are multiplicative (Shao, 2000), hence $f_s = f_h f_l$.

2.3 Dust emissions

2.3.1 Saltation

In principle, the saltation flux is calculated according to (1). However, that expression is valid only for a soil containing a single particle size. In reality, though, soil particle sizes are distributed over different size classes (Table H1). Therefore, for a given value of u_*, the saltation flux is calculated as a weighted average of the saltation fluxes calculated for each size class separately (hence using a threshold value u_{*t} depending on the mean particle size of the considered class), as follows:

$$\overline{Q} = \sum_i \widetilde{Q}[u_*, u_{*t}(D_i)]\, p(D_i),\tag{27}$$

where the sum is over the size classes shown in Table H1, with $p(D_i)$ the mass fraction of the class with mean size D_i.

For given values of u_* and $u_{*t}(D_i)$, (27) gives the instantaneous saltation flux. However, since the method described here is based on daily meteorological data, the effect of sub-daily varying wind speed (hence friction velocity) is to be accounted for. To do so, use is made of the wind statistics developed above, and per size class i the daily average saltation flux required in (27) is calculated as follows:

$$\widetilde{Q} = c\,\frac{\rho}{g}\left[\frac{k}{\ln(z/z_0)}\right]^3 \int_{V_t}^{\infty} Q(V,V_t)\exp\left[-\left(\frac{V}{c_W}\right)^{k_W}\right] d\left(\frac{V}{c_W}\right)^{k_W},\tag{28}$$

which is a daily average value, weighted using the Weibull distribution function, for which daily coefficients were derived above as a function of daily average and maximum sustained wind speed. The factor in front of the integral arises from (1) and (3). The wind speed V and the threshold wind speed V_t that occur in the integration are related to the friction velocity and the threshold value of the latter by means of (3). Again using (1) and (3), (28) can be developed as

$$\widetilde{Q} = c\,\frac{\rho}{g}\left[\frac{k}{\ln(z/z_0)}\right]^3 \left(I_3 + V_t I_2 - V_t^2 I_1 - V_t^3 I_0\right),\tag{29}$$

with

$$I_n \equiv c_W^n \Gamma_{x_t}\left(1+\frac{n}{k_W}\right).\tag{30}$$

In the last expression use is made of the incomplete Gamma function (see, e.g., Abramowitz and Stegun, 1969), given by

$$\Gamma_x(a) \equiv \int_x^{\infty} e^{-t} t^{a-1}\, dt,\tag{31}$$

and employing

$$x_t \equiv \left(\frac{V}{c_W}\right)^{k_W}.$$ (32)

2.3.2 Dust fluxes

The final step of the dust calculation method consists of the application of (2) which, when using daily average saltation fluxes, calculates the daily average dust flux as follows:

$$\overline{F} = \gamma\,\overline{Q}\,e^{0.308\,f_c}.$$ (33)

The thus calculated dust is composed of particles with a size up to 50 μm. As we are mainly interested here in PM$_{10}$, the dust fluxes calculated with (33) are multiplied by the proportion of dust that is composed of particles smaller than 10 μm, as can be derived from Table H1.

3. Results

Figure H3 gives an overview of the output that is typically produced by the method described here, using meteorological data from the station SKAVASTA/ STOCKHOLM. The soil type is loam, and the terain consists of an upwind urban edge containing residential buildings of 10 m high, with a frotal area index of 10. This corresponds to a situation where a parcel of terrain of 100×100 m^2 contains 10 houses of 10 m wide by 10 m high. The soil between the buildings is without vegetation or else with very sparse vegetation, characterised by $z_{0g} = 0.01$ cm, which is very smooth.
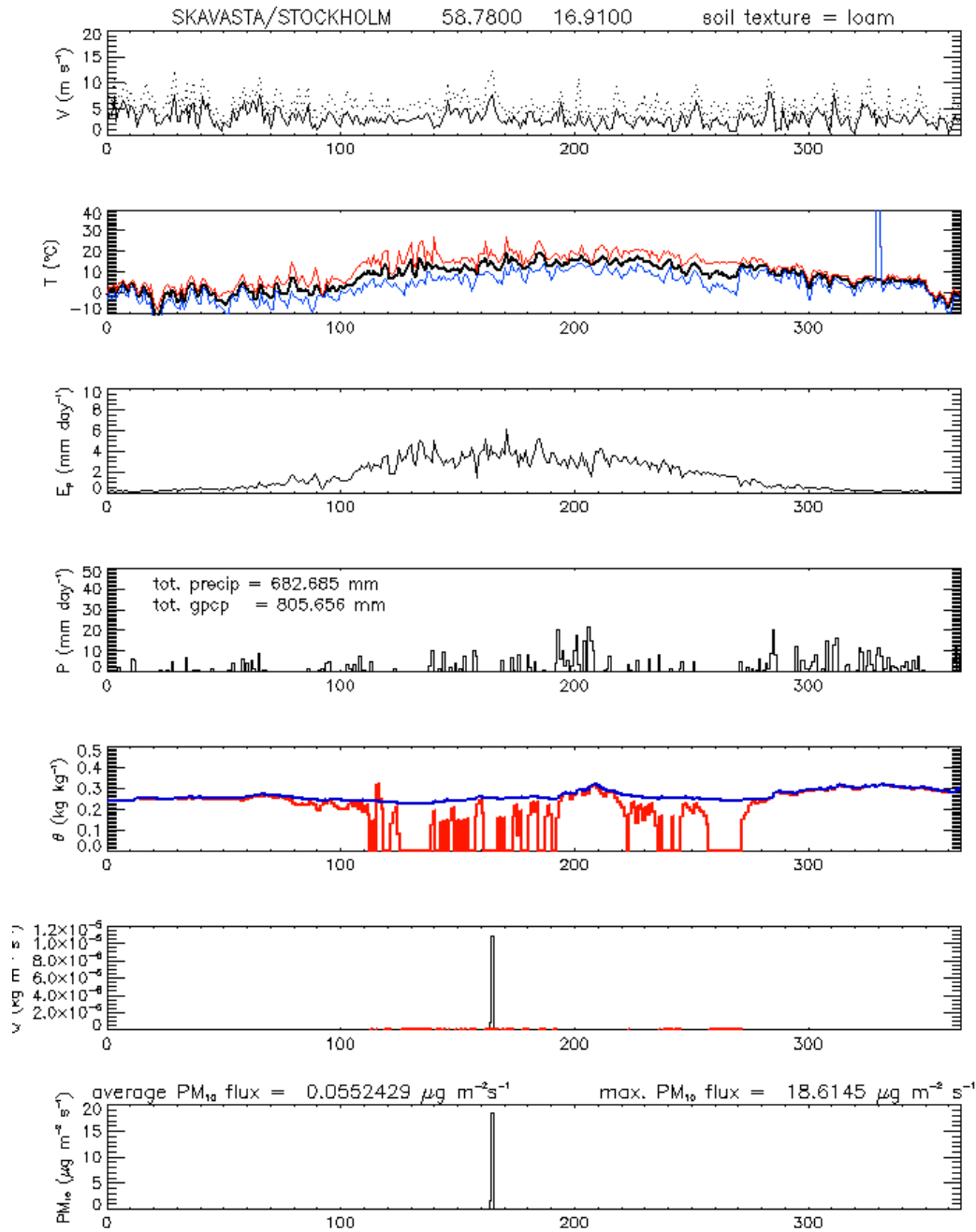
Figure H3. Results of a calculation of PM10 fluxes using the method described in the text. All panels show time series of quantities with a resolution of one day, as follows. The upper panel shows wind speed (solid line) and maximum sustained wind speed (dotted line) calculated at the site. The second panel shows average (black), maximum (red) and minimum (blue) temperatures. The third panel gives daily potential evaporation, and the fourth panel daily precipitation. The fifth panel displays deep (blue) and superficial (red) soil moisture. The two lower panels show, respectively, the saltation flux and the PM10 flux.

In Table H3 some results are shown using meteorological data from different Swedisch stations, for residential areas composed of 10-m high buildings. The fetch (upwind distance) to the city edge (assumed to be located next to a smooth plain) was taken as 10 m, though the 'turbulence enhancement factor' was imposed an upper limit of 2.5. The soil in between the buildings was assumed extremely flat, with $z_{0g} = 10^{-5}$ m.

*Table H3. Calulated PM10 fluxes using meteorological data from different Swedish stations, for a very flat soil between buildings located at a city edge next to a smooth plain, for different soil types (values in **bold** are used in calculations).*

z_{0g} (m)	soil	PM$_{10}$ flux (μg m^{-2} s^{-1})							
		Skavasta/ Stockholm		Stockholm/ Bromma		Malmö/ Sturup		Göteborg/ Landvetter	
		mean	max	mean	max	mean	max	mean	max
10^{-5}	sand	6.4	1,672	-	-	3.1	1,094	0.92	333.7
	loam	15.8	4,248	-	-	7.1	2,555	3.4	1,231
	clay	389	102,024	-	-	184.4	66,010	58.3	21,120
10^{-4}	sand	0.026	8.6	-	-	0.002	0.69	0.05	17.5
	loam	**0.05**	18	-	-	**0.004**	1.4	**0.12**	43.7
	clay	1.5	501	-	-	0.11	38.9	2.9	1,060

The data in Table H3 shown for $z_{0g} = 10^{-5}$ m represents a worse case scenario in terms of PM$_{10}$ fluxes. In fact, such a smooth surface is not very realistic except perhaps in the smoothest deserts. A value of $z_{0g} = 10^{-4}$ m is more realistic, though still very smooth. In the model fluxes were reduced to zero by increasing the roughness above $z_{0g} = 0.001$ m, meaning that 'normally rough' soil surfaces or vegetated surfaces can inhibit the dust emissions completely.

Main uncertainties:
- Clay dependence of dust yield function at high clay fractions (also stated as problematic by Bergametti and marticorena, 1995);
- high turbulence levels at city edge – what is maximum possible (currently turbulence enhancement factor is calculated and limited to 2.5 at most, but using, e.g., 2 as an upper limit drastically alters the results...).

APPENDIX E: CALCULATION OF THE AVERAGE CONCENTRATION OF CONTAMINATED DUST IN INHALED AIR

On the basis of the calculated PM_{10} fluxes (Appendix G), annual average concentrations of contaminated dust in inhaled air are calculated by using a Box model.

The following PM_{10} fluxes are calculated (z_{0g}: 10^{-4} m; Appendix H):
- Stockholm/Skavasta: 0.05 µg/m².s   (i.e. 4.32 mg/m².d);
- Malmö/Sturup: 0.004 µg/m².s   (i.e. 0.346 mg/m².d);
- Göteborg/Landsvetter: 0.12. µg/m².s   (i.e. 10.37 mg/m².d).

These fluxes apply to a very flat soil between buildings located at a city edge next to a smooth plain, for a loam soil.

The Box model used in S-RISK is the same as applied in the Vlier-Humaan model (Flanders) for the calculation of on-site concentrations in the gas phase. Following model equations are employed:

$$C_{oa} = C_{oa,0} + \frac{J_{oa,p}}{V_f}$$

$$S_z = C_o \times 0.2 \times L_{bl}^{0.76}$$

$$V* = (k \times V_h) / \ln(h / s_r)$$

If $s_r > Y$
Then $V_Y = 0$
Else $V_Y = \ln(Y/s_r) \times V*/k$

$$V_g = (V_Y + V*) / 2$$

$$V_f = \frac{(V_g \times S_z)}{L_{bl}}$$

where

C_{oa}:   concentration in outdoor dust [mg/m³];
$C_{oa,0}$: incoming concentration in outdoor dust [mg/m³];
$J_{oa,p}$:   diffusion flux soil to atmoshere [mg/m².d];
C_o: correction factor for roughness length [-];
S_z: Pasquill dispersion coefficient, vertical direction, stability class D [m];
V_g: average wind velocity [m/d];
V_Y: wind velocity on height Y [m/d];
V_f: dilution velocity [m/d]
Y: inhalation height child or adult [m];

V^*: friction velocity [m/d];
V_h: wind velocity on height h [m/d];
h: height [m];
k: Karman constant [-];
S_r: roughness [m].

For Stockholm, Malmö and Göteborg, concentrations in dust are calculated by using following parameter values:

$C_{oa,0}$: 0 mg/m³;
$J_{oa,p}$: Stockholm: 4.32 mg/m².d; Malmö: 0.346 mg/m².d; Göteborg: 10.37 mg/m².d;
C_o: 9.06;
S_z: 60 m;
Y: 1 m (child);
V_h: Stockholm: 273,024 m/d; Malmö: 315,360 m/d; Göteborg: 254,880 m/d;
h: 10 m;
k: 0.4;
S_r: 0.6 m.

The following concentrations of inhaled dust in outdoor air are calculated:
  - Stockholm: 1.63×10^{-4} mg/m³;
  - Malmö: 1.13×10^{-5} mg/m³;
  - Göteborg: 4.19×10^{-4} mg/m³.

Since these calculated concentrations only consider wind-induced dust emissions originating from the site, and additional (mechanical) dust emissions due to activities on the site (e.g. children playing, agricultural activities,…) are not taken into account, the calculated concentrations of inhaled dust were multiplied with an additional safety factor of 10. Also, it is assumed that the concentration of inhaled dust in indoor air is the same as in outdoor air.

To derive an overall applicable annual average concentration of inhaled dust in the entire specified region, the calculated outdoor dust concentration in Göteborg (4.19×10^{-4} mg/m³) was multiplied with a safty factor of 10 and round up to 5×10^{-3} mg/m³ (value used in S-RISK).

At present, the average concentration of contaminated dust in inhaled air in S-EPA is estimated to be 4.1×10^{-2} mg/m³ (based on data from CSOIL).

APPENDIX F: TOXICOLOGY OF CADMIUM

This appendix is to a large extent excerpted from WHO (1992a,b, 1996). The goal of this appendix is to give a rationale for the recommended toxicological reference values for the oral and inhalation exposure pathways in S-RISK.

1. Kinetics and metabolism

In mammals, Cd is virtually absent at birth. Data from experimental animals and humans have shown that pulmonary absorption is higher than gastrointestinal absorption. Depending on chemical speciation, particle size, and solubility in biological fluids, up to 50% of the inhaled cadmium compound may be absorbed. The gastrointestinal absorption of cadmium is influenced by the type of diet and nutritional status. The nutritional iron status appears to be of particular importance. On average, 5% of the total oral intake of cadmium is absorbed, but individual values range from less than 1% to more than 20%. There is a maternal-fetal gradient of cadmium. Although cadmium accumulates in the placenta, transfer to the fetus is low. Cadmium absorbed from the lungs or the gastrointestinal tract is mainly stored in the liver and kidneys, where more than half of the body burden will be deposited. With increasing exposure intensity, an increasing proportion of the absorbed cadmium is stored in the liver. Excretion is normally slow, and the biological half-time is very long (decades) in the muscles, kidneys, liver, and whole body of humans. The cadmium concentrations in most tissues increase with age. Highest concentrations are generally found in the renal cortex, but excessive exposures may lead to higher concentrations in the liver. In exposed people with renal damage, urinary excretion of cadmium increases and so the whole body half-time is shortened. The renal damage leads to losses of cadmium from the kidney, and the renal concentrations of cadmium will eventually be lower than in people with similar exposure but without renal damage (JECFA, 2001; WHO, 1992a,b).

Metallothionein is an important transport and storage protein for cadmium and other metals. Cadmium can induce metallothionein synthesis in many organs including the liver and kidney. The binding of intracellular cadmium to metallothionein in tissues protects against the toxicity of cadmium. Cadmium not bound to metallothionein may therefore play a role in the pathogenesis of cadmium-related tissue injury. The speciation of other cadmium complexes in tissues or biological fluids is unknown (WHO, 1992a,b).

Urinary excretion of cadmium is related to body burden, recent exposure, and renal damage. In people with low exposure, the urine cadmium level is mainly related to the body burden. When cadmium-induced renal damage has occurred, or even without renal damage if exposure is excessive, urinary excretion increases. Cadmium-exposed people with proteinuria generally have higher cadmium excretion than such people without proteinuria. After high exposure ceases, the urine cadmium level will decrease even though renal damage persists. The interpretation of urinary cadmium is thus dependent on a number of factors. Gastrointestinal excretion is approximately equal to urinary excretion but cannot be easily measured. Other excretory routes such as lactation, sweating or placental transfer are insignificant (WHO, 1992a,b).

The level of cadmium in faeces is a good indicator of recent daily intake from food in the absence of inhalation exposure. Cadmium in blood is found mainly in the red blood cells, and the plasma concentrations are very low. There are at least two compartments in blood, one related to recent exposure with a half-time of about 2-3 months, and one which is probably related to body burden with a half-time of several years (WHO, 1992a,b).

2. Effects on humans

2.1 Overview

The estimated lethal oral dose for humans is 350-3,500 mg Cd. A dose of 3 mg Cd has no effect on adults. With chronic oral exposure, the kidney appears to be the most sensitive organ. Cadmium affects the resorption function of the proximal tubules, the first symptom being an increase in the urinary excretion of low-molecular-weight proteins, known as tubular proteinuria. Intakes of 140-255 µg of cadmium per day have been associated with low-molecular-weight proteinuria in the elderly; the minimum (critical) level of cadmium in the human renal cortex, related to the first sign of tubular dysfunction, varied from 100 to 450 mg/kg wet weight. The estimated critical concentration in the renal cortex at which the prevalence of low-molecular-weight proteinuria would reach 10% in the general population is about 200 mg/kg; this would be reached after a daily dietary intake of about 75 µg per person for 50 years, as calculated by regression analysis of cadmium intake and mean kidney cadmium concentration in various countries. It was estimated that a daily intake of 100 µg of cadmium per person would lead to the critical cadmium concentration in the renal cortex being exceeded in 2% of the population. More severe cadmium damage may also involve the glomeruli, giving rise to increased inulin clearance. Other possible effects include aminoaciduria, glucosuria, and phosphaturia. Disturbances in renal handling of phosphorus and calcium may cause resorption of minerals from bone, which can result in the development of kidney stones and osteomalacia. Many cases of itai-itai disease (osteomalacia -softening of the bones- with various grades of osteoporosis accompanied by severe renal tubular disease) and low-molecular-weight proteinuria have been reported among people living in contaminated areas in Japan and exposed to cadmium via food and drinking-water. The daily intake of cadmium in the most heavily contaminated areas amounted to 600-2000 µg/day; in other less heavily contaminated areas, daily intakes of 100-390 µg/day have been found. A relationship between chronic occupational exposure to cadmium or chronic oral exposure to cadmium via the diet in contaminated areas and hypertension could not be demonstrated. Epidemiological studies of people chronically exposed to cadmium via the diet as a result of environmental contamination have not shown an increased cancer risk. The results of studies of chromosomal aberrations in the peripheral lymphocytes of patients with itai-itai disease exposed chronically to cadmium via the diet were contradictory. No reliable studies on reproductive, teratogenic, or embryotoxic effects in humans are available (WHO, 1992a,b, 1996).

High inhalation exposure to cadmium oxide fume results in acute pneumonitis with pulmonary oedema, which may be lethal. Following high occupational exposure, lung changes are primarily characterized by chronic obstructive airway disease. Early minor changes in ventilatory function tests may progress, with continued cadmium exposure, to respiratory insufficiency. Epidemiological studies of humans exposed by inhalation to

relatively high cadmium concentrations in the workplace revealed some evidence of an increased lung cancer risk, but a definite conclusion could not be reached, due to confounding factors (WHO, 1992a,b, 1996).

2.2 Conclusions

The kidney is considered the critical target organ for the general population as well as for occupationally exposed populations. Chronic obstructive airway disease is associated with long-term high-level occupational exposure by inhalation. There is some evidence that such exposure to cadmium may contribute to the development of cancer of the lung but observations from exposed workers have been difficult to interpret because of confounding factors (WHO, 1992a,b, 1996).

3. Toxicological reference values of cadmium

3.1 WHO/IARC/JECFA

The IARC has classified Cd and Cd compounds in Group 1. They concluded that there is sufficient evidence in humans for the carcinogenicity of cadmium and cadmium compounds, sufficient evidence in experimental animals for the carcinogenicity of cadmium compounds, and limited evidence in experimental animals for the carcinogenicity of cadmium metal. In making the overall evaluation, the Working Group took into consideration the evidence that ionic cadmium causes genotoxic effects in a variety of types of eukaryotic cells, including human cells (IARC, 1993).

3.1.1 Oral exposure

There is no evidence of carcinogenicity by the oral route (WHO, 1996).

On the assumption of an absorption rate for dietary cadmium of 5% and a daily excretion rate of 0.005% of body burden, the JECFA (Joint WHO/FAO Expert Committee on Food Additives) concluded that, if levels of cadmium in the renal are not to exceed 50 mg/kg, the total intake of cadmium should not exceed 1 µg/lg of body weight per day. The provisional tolerable weekly intake (*PTWI*) was therefore set at 7 µg/kg of body weight in 1988 and reconfirmed in 1993 and 2003. It is recognized that the margin between the *PTWI* and the actual weekly intake of cadmium by the general population is small, namely less than 10-fold, and that this margin may be even smaller in smokers.

JECFA re-evaluated Cd in 2001 because analysis of new population-based data indicated that the early renal effects of Cd were prevalent after lower intakes than those indicated by the model used to establish the *PTWI*. It was concluded that the risk for tubular dysfunction begins to increase when urinary excretion exceeds 2.5 µg/g of creatinine. Using a theoretical model, is was then calculated which cadmium (oral) intake would produce excess prevalence of renal tubular dysfunction (i.e. if urinary excretion exceeds 2.5 µg/g). Three scenarios were considered as reasonable (Table J1).

Table J1: Relationschip between predicted cadmium intake, urinary cadmium excretion of 2.5 µg/g creatinine at steady state, and predicted no-excess prevalence (i.e. equals 0) of renal tubular dysfunction (JECFA, 2001).

Scenario	Predicted intake of Cd	
	[mg/d]	**[mg/kg.d] (60 kg)**
Ratio of dietary Cd intake to urinary excretion: 12; Bioavailability of Cd in diet: 10%; Absorbed Cd excreted in urine: 100%.	3×10^{-2}	5×10^{-4}
Ratio of dietary Cd intake to urinary excretion: 24; Bioavailability of Cd in diet: 10%; Absorbed Cd excreted in urine: 50%.	6×10^{-2}	1×10^{-3}
Ratio of dietary Cd intake to urinary excretion: 48; Bioavailability of Cd in diet: 5%; Absorbed Cd excreted in urine: 50%.	1.2×10^{-1}	2×10^{-3}

These estimates indicate that a proportion of the general population may be at increased risk for tubular dysfunction when exposed at the *PTWI* of 7 µg/kg.week. However, the Committee maintained this value because of lack of precision in the risk estimates (JECFA, 2001). The Committee made several recommendations regarding the data that would be needed in order to reduce the uncertainty in the prevalence estimates.

Recently (2004), the JECFA Committee considered an extensive amount of new information, particularly from a series of Japanese environmental epidemiological studies, that addressed issues identified as research needs at it fifthy-fifth meeting in 2001. However, The Committee concluded that the new data do not provide a sufficient basis for revising the *PTWI*, and therefore maintained the current *PTWI* of 7 µg/kg body weight. No excess prevalence of renal tubular dysfunction would be predicted to occur at the current *PTWI* under the most appropriate assumptions about the fractional bioavailability of cadmium and the percentage of the absorbed cadmium that is excreted in urine (JECFA, 2004).

WHO established a guideline value for cadmium of 3.10^{-3} mg/l on the basis of an allocation of 10% of the *PTWI* to drinking water (WHO, 1996).

3.1.1 Inhalation exposure

Cadmium, whether absorbed by inhalation or via contaminated food, may give rise to various renal alterations. The lowest estimate of the cumulative exposure to airborne cadmium in industrial workers leading to an increased risk of renal dysfunction (low-molecular-weight proteinuria) or lung cancer is 100 µg/m³ x years for an 8-hour exposure. Extrapolation to a continuous lifetime exposure gives a value of around 3×10^{-4} mg/m³. Since the identified and controversial influence of concomitant exposure to arsenic in the epidemiological study, no reliable unit risk could be derived to estimate the excess lifetime risk for lung cancer. Therefore the derived value should be considered a *TCL*. The corresponding *TDI* for inhalation exposure (20 m³/d; 70 kg) is 8.57×10^{-5} mg/kg.d.

Existing levels of cadmium in the air of most urban or industrial areas are around one-fiftieth of this value.The finding of renal effects in areas contaminated by past emissions of cadmium indicates that the cadmium body burden of the general population in some parts of

Europe cannot be further increased without endangering renal function. To prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations, a guideline of 5 ng/m³ is established (WHO, 2000).

3.2 US-EPA/IRIS

The US-EPA (1985) has classified Cd as a B2 (probable human) carcinogen on the basis of limited evidence from occupational epidemiological studies and sufficient evidence from animal studies.

3.2.1 Oral exposure

There is no quantitative estimate of carcinogenic risk from oral exposure available since there are no positive studies of orally ingested cadmium suitable for quantification (US-EPA, 2005).

The US-EPA recommends two oral reference doses for cadmium: one for cadmium exposure from food and one for cadmium exposure from water. Both *RfD*s recognize that a concentration of 200 µg/g (wet weight) in the human kidney cortex is the highest renal level not associated with significant proteinuria. A toxicokinetic model was used to determine the level of chronic human oral exposure (NOAEL) which results in 200 µg Cd/g wet human renal cortex (the model assumes that 0.01% day of the Cd body burden is eliminated per day). Assuming 2.5% absorption of Cd from food or 5% from water, the toxicokinetic model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg.d from water and food, respectively. Thus, based on an estimated NOAEL of 0.005 mg Cd/kg.d for Cd in drinking water and an uncertainty factor of 10, an *RfD* of 0.0005 mg/kg (water) was calculated; an equivalent *RfD* for Cd in food is 0.001 mg/kg.d (US-EPA, 2005).

The MCLG (Maximum Contaminant Level Goal) and the MCL (Maximum Contaminant Level) in drinking water both have been set at $5x10^{-3}$ mg/l (US-EPA, 2005).

3.2.1 Inhalation exposure

An inhalation unit risk of $1.8x10^{-3}$ per µg/m³ is derived, using a two stage extrapolation method on results of an inhalation exposure study in the workplace. The corresponding air concentration and dose at risk level 10^{-5} is $6x10^{-6}$ mg/m³ and $1.7x10^{-6}$ mg/kg.d respectively (US-EPA, 2005).

At present, there is no *RfC* for chronic inhalation exposure available (for non-cancer effects; US-EPA, 2005).

3.3 EC

The European Commission has classified cadmium chloride, cadmium oxide, and cadmium sulfate classified as substances which should be regarded as if they are carcinogenic to man (Group 2). Cadmium sulfide is classified as a substance which cause concern for man owing to possible carcinogenic effects, but in respect of which the available information is not adequate for making a satisfactory assessment (Group 3).

3.3.1 Oral exposure

No toxicological reference values are derived for the oral exposure pathway.

3.3.1 Inhalation exposure

As cadmium and cadmium compounds may be -at least in part- genotoxic, the Working Group believes that it is prudent to extrapolate linearly from the available unit risk estimates quoted by WHO, US-EPA and NIOSH (National Institute for Occupational Safety and Health): 1.8 or 2.45 or 4.15×10^{-3}, to a cadmium concentration associated with an excess lifetime risk of lung cancer of one-in-a-million. This leads to a concentration range of 0.24-0.55 ng/m³. It should be noted that this range is significantly below currently measured concentrations in non-rural areas of Europe. However, they also note that the uncertainties concerning qualitative and quantitative aspects of possible cancerogenicity of cadmium, and the uncertainty associated with the extrapolation from (the unit risk reference point of) 1 µg/m³ to considerably lower concentrations at the nanogram/m³ level means that the 0.24-0.55 range based on the unit-risk approach is likely to be overprotective (EC, 2001a).

The European Environmental Bureau is of the opinion that with respect to the genotoxicity of cadmium and as lower-dose-linear models may be more appropriate when a carcinogen acts in concert with other exposures and processes that cause a background incidence of cancer, a proposal of 2.5 ng/m³ corresponding to the upper bound of the urban background values in Europe can be regarded as one first step to a further reduction, by a future revision of this limit value (EC, 2001a).

The EC Working Group considers the cumulative dose of 100 µg/m³ x years as a threshold for an increased excretion of markers of renal function changes (as derived by WHO) as a starting point, from which a LOAEL can be extracted and used to derive a limit value. This occupational LOAEL is extrapolated from 8 hours to 24 hours, from 225 working days to 365 days and distributed over an average human lifetime of 75 years. The overall conversion factor is 0.0027 (8/24 x 225/365 x 1/75). Consequently, by applying this factor, the occupational LOAEL of 100 µg/m³ x years can be converted to a LOAEL (continuous) of 270 ng/m³. Applying an uncertainty factor of 5 for extrapolation from LOAEL to NOAEL, and an intraspecies uncertainty factor of 10 leads to a limit value of 5 ng/m³, which is likely to prevent renal damage due to inhalation exposure.

In summary, the Working Group believes that an annual mean concentration level of 5 ng/m³ as derived from non-cancer effects provides also an appropriate level of protection from cancer risk due to exposure to cadmium (EC, 2001a). WHO also recommends 5 ng/m³

in order to prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations. However, WHO has derived this value on different considerations.

Recently, the European Directive 2004/107/EC (European Parliament and the Council, 2005) has set a target value for Cd in ambient air. This target value is not to be considered as an environmental quality standard but means a concentration in ambient air fixed with the aim of avoiding, preventing or reducing harmful effects on human health and the environment as a whole, to be attained where possible over a given period. The Member States of the EU therefore need to take all necessary measures not entailing disproportionate costs to ensure that, as from 31/12/2012, the concentration of Cd in ambient air does not exceed 5 ng/m³ (for the total content in the PM_{10} fraction averaged over a calendar year).

3.4 Summary of toxicological reference values

A summary of the toxicological reference values for cadmium reported by different agencies is given in Table J2.

Table J2: Summary toxicological reference values for cadmium.

Agency	Non-carc. effects			Carc. effects		
	Oral [mg/kg.d]	Inhalation ([mg/m³])	Drinking water limit [mg/l]	Oral [mg/kg.d]	Inhalation ([mg/m³])	Drinking water limit [mg/l]
JECFA, 2001, 2003, 2004 WHO, 1989, 1993, 1998b, 2000, 2001	1×10^{-3}	8.57×10^{-5} (3×10^{-4}) (5×10^{-6}) *	3×10^{-3}	-		-
US-EPA, 1985, 2005	5×10^{-4} (water) 1×10^{-3} (food)	-	5×10^{-3}	-	1.7×10^{-6} (6×10^{-6})	-
EC, 2001a	-	1.43×10^{-6} (5×10^{-6}) #	-	-	$0.69\text{-}1.57 \times 10^{-6}$ ($2.4\text{-}5.5 \times 10^{-6}$) **	-

*: The finding of renal effects in areas contaminated by past emissions of cadmium indicates that the body burden of the general population in some parts of Europe cannot be further increased without endangering renal functions. To prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations, a guideline of 5 ng/m³ is established.

**: Derived from the range $2.4\text{-}5.5 \times 10^{-7}$ mg/m³ corresponding to an excess lifetime cancer risk of 10^{-6}.

#: Set as target value according to Directive 2004/107/EC.

4. Recommendations for S-RISK

4.1 Oral exposure

An oral *TDI* of $1x10^{-3}$ mg/kg.d is selected on the basis of the *PTWI* of $7x10^{-3}$ mg/kg.week. This value is the same as the *RfD* (for food) reported by US-EPA. Also, JECFA has recently re-evaluated Cd and maintained this value.

The WHO-drinking water guideline value of $3x10^{-3}$ mg/l is recommended. It is established based on an allocation of 10% of the *PTWI* to drinking water (WHO, 1996).

4.2 Inhalation exposure

The *TCL* of EC (i.e. $5x10^{-6}$ mg/m³) is recommended for use in S-RISK. According to the Working Group of the EC, this value derived from non-cancer effects, provides also an appropriate level of protection from cancer risk due to exposure to cadmium. Also, this value is recommended by WHO to prevent any further increase of cadmium in agricultural soils likely to increase the dietary intake of future generations.

APPENDIX G: TOXICOLOGY OF PAH COMPOUNDS

This appendix provides an overall picture of kinetic and metabolic aspects of PAHs, effects on laboratory animals (and *in vitro*) and humans, a brief outline on the carcinogenicity of PAH compounds and toxicological reference values, as derived by different authorities. The information in this chapter is to a large extent compiled from data from Boström et al. (2002), EC (2001b), Ramesh et al. (2002), WHO (1996, 1998a, 2000) and the IARC-, US-EPA(IRIS)-, and WHO-websites.

The goal of this appendix is to give a rationale for the recommended toxicological reference values for the oral and inhalation exposure pathways in S-RISK.

1. Kinetics and metabolism

Polycyclic aromatic hydrocarbons are lipophilic compounds that can be absorbed through the lungs, the gastrointestinal tract, and the skin. Irrespective of the route of administration PAHs are rapidly and widely distributed in the organism. The pattern of distribution of benzo(a)pyrene was found to be similar after subcutaneous, intravenous and intratracheal administration to mice and rats. Detectable levels of benzo(a)pyrene can be observed in most internal organs from minutes to hours after administration. Highest levels are obtained in the liver. Mammary and other fatty tisssues are significant storage depots for PAHS, but owing to the rapid metabolism no significant accumulation seems to take place (WHO, 1998a, 2000).

Absorption from the gastrointestinal tract occurs rapidly in rodents, but metabolites return to the intestine via biliary excretion. Therefore, the gastrointestinal tract contains relatively high levels of metabolites. For pyrene, the distribution to the tissues was highest in the perirenal fat, intermediate in the liver, kidneys and lungs, and lowest in the heart, testes, spleen and brain. Benzo(a)pyrene can readily cross the placental barrier of rats and mice, consistent with the fetal and developmental toxicity of the substance (WHO, 1998a, 2000).

The rate of absorption from the lungs depends on the type of PAH, the size of the particles on which they are absorbed, and the composition of the adsorbent. PAHs adsorbed onto particulate matter are cleared from the lungs more slowly than free hydrocarbons (WHO, 1998a).

Studies with ^{32}P-postlabelling of percutaneous absorption of mixtures of PAHs in rodents showed that components of the mixtures reach the lungs, where they become bound to DNA, the rate of percutaneous absorption in mice according to the compound. All aspects of the absorption, metabolism, activation, and excretion of benzo(a)pyrene have been covered exhaustively in the published literature, but there is a dearth of information on many of the other PAHs (WHO, 1998a, 2000).

The metabolism of PAHs to more water-soluble derivatives, which is a prerequisite for their excretion, is complex. Generally, the process involves epoxidation of double bonds, a reaction catalysed by cytochrome P450-dependent mono-oxygenases, rearrangement or

hydration of the epoxides to yield phenols or diols, respectively, and conjugation of the hydroxylated derivatives. Metabolism of PAHs occurs in all tissues. The metabolic process involves several possible pathways with varying degrees of enzyme activities. The activities and affinities of the enzymes in a given tissue determine which metabolic route will prevail. Therefore, the reaction rates vary widely: interindividual variations of up to 75-fold have been observed, for example, with human macrophages, mammary epithelial cells, and bronchial explants from different donors. Most metabolism results in detoxification, but some PAHs are activated to DNA-binding species, principally diol epoxides, which can initiate tumors (HSDB, 2004; WHO, 1998a).

PAH metabolites and their conjugates are excreted via the urine and faeces, but conjugates excreted in the bile can be hydrolysed by enzymes of the gut flora and reabsorbed. It can be inferred from the available information on the total human body burden that PAHs do not persist in the body and that turnover is rapid. This inference excludes those PAH moieties that become covalently bound to tissue constituents, in particular nucleic acids, and are not removed by repair (WHO, 1998a).

2. Effects on laboratory animals and *in vitro*

2.1 Acute exposure

The oral LD_{50}-values for various PAHs are reported to range between 490 and 18,000 mg/kg body weight. Effects induced in animals following acute exposure induce inflammation, hyperplasia, hyperkeratosis and ulceration of the skin, pneumonitis, damage to the haematopoietic and lymphoid systems, immunosuppresion, adrenal necrosis, ovotoxicity, and antispermatogenic effects (WHO, 1996).

2.2 Short-term exposure

Short-term studies showed adverse haematological effects, expressed as myelotoxicity with benzo(a)pyrene, haemolymphatic changes with dibenzo(a,h)anthracene, and anaemia with naphthalene. However, in a seven-day study by oral and intraperitoneal administration in mice, tolerance to the effect of naphthalene was observed (WHO, 1998a).

Other reported effects after short-term exposure to various PAHs include: increased alanine aminotransferase levels, kidney and liver changes, and clinical and haematological changes with fluoranthene; haematological effects with fluorene, effects on the kidney (renal tubular pathology, decreased kidney weight) with pyrene; loss of body weight and mild pathological changes in the liver and kidney in rats given oral doses of acenaphthene; and depression of body weight gain, elevated liver weight, and lowered spleen weight with fluorene (WHO, 1996).

2.3 Long-term exposure

Systemic effects caused by long-term treatment with PAHs have been described only rarely, because the end-point of most studies has been carcinogenicity. Significant toxic effects are manifested at doses at which carcinogenic responses are also triggered (WHO, 1996).

2.4 Reproductive toxicity, embryotoxicity, and teratogenicity

Benz(a)anthracene, benzo(a)pyrene, dibenzo(a,h)anthracene, and naphthalene are embrotoxic to mice and rats. Benzo(a)pyrene also has teratogenic and reproductive effects. Intensive efforts have been made to elucidate the genetic basis of the embryotoxic effect of benzo(a)pyrene. Fetal death and malformations are observed only if the cytochrome P450 mono-oxygenase system is inducible, either in the mother (with placental permigration) or in the embryo. Not all of the effects observed can be explained by genetic predisposition, however: in mice and rabbits, benzo(a)pyrene had transplacental carcinogenic activity, resulting in pulmonary adenomas and skin papillomas in the progeny. Reduced fertility and oocyte destruction were also observed (WHO, 1998a).

2.5 Mutagenicity and related end-points

Benzo(a)pyrene was found to be mutagenic in *Salmonella typhimurium* strain TA1538 after metabolic activation by a preparation of microsomal enzymes from a liver homogenate, fraction S9, obtained from rats. It has also induced mutations in cultured human lymphoblastoid cells. The diol-epoxide metabolites of benzo(a)pyrene are considerably more mutagenic than the parent compound. Induction of sister chromatid exchanges in Chinese hamsters following intraperitoneal administration of benzo(a)pyrene has been reported, and a correlation has been observed between sister chromatid exchange and the production of benzo(a)pyrene metabolites in two variant mouse hepatoma cell lines (WHO, 1996).

3. Effects on humans

Because of the complex profile of PAHs in the environment and in workplaces, human exposure to pure, individual PAH has been limited to scientific experiments with volunteers, except in the case of naphthalene which is used as a moth-repellant for clothing (WHO, 1998a).

After dermal application, anthracene, fluoranthene, and phenanthrene induced specific skin reactions, and benzo(a)pyrene induced reversible, regressive verrucae which were classified as neoplastic proliferations. The systemic effects of naphthalene are known from numerous cases of accidental intake, particularly by children. The lethal oral dose is 5000-15,000 mg for adults and 2,000 mg taken over two days for a child. The typical effect after dermal or oral exposure is acute haemolytic anaemia, which can also affect fetuses transplacentally (WHO, 199a).

4. Carcinogenicity

4.1 Introduction

Evidence that mixtures of PAHs are carcinogenic in humans comes primarily from occupational studies of workers. Cancer associated with exposure to PAH-containing mixtures in humans occurs predominantly in the lung and skin following inhalation and dermal exposure, respectively. There are no data available for humans for the oral route. There are only a few animal carcinogenicity studies on oral administration. Further information is available on the carcinogenicity of single PAHs from experiments with dermal application (WHO, 1998b).

In summary, it is not possible to assess directly the risk of PAHs to humans for the oral route owing to a lack of human data. One must rely on animal data to estimate the risk of exposure to individual PAHs, not forgetting that humans are exposed to mixtures of PAHs and not to pure individual PAHs. The extrapolation of risk to humans from animal data is complicated: the relevance of forestomach tumors in rodents when considering extrapolation to humans is not clear. However, it is anticipated that PAHs are carcinogenic by the oral pathway as well (WHO, 1998b).

Tobacco smoking is the most important single factor in the induction of lung tumors and also for increased incidences of tumors of the urinary bladder, renal pelvis, mouth, harynx, larynx, and oesophagus. The contribution of PAH in the diet to the development of human cancer is not considered to be high. In highly industrialized areas, increased body burdens of PAH due to polluted ambient air were detected. Psoriasis patients treated with coal-tar are also exposed to PAH. Occupational exposure to soot as a cause of scrotal cancer was noted for the first time in 1775. Later, occupational exposure to tars and paraffins was reported to induce skin cancer. The lung is now the main site of PAH-induced cancer, whereas skin tumors have become more rare because of better personal hygiene (WHO, 1998a).

4.2 Mechanism of carcinogenicity

Extensive mechanistic studies have shown that many PAH compounds -including some that occur in ambient air- are complete carcinogens, i.e. they can both induce cancer by producing mutations in DNA and promote cancer by affecting the proliferative capacity of affected cells (e.g. Baird et al., 2005). These effects are rendered to as genotoxic and epigenetic effects, respectively (EC, 2001b).

Genotoxic effects depend on intracellular conversion of PAH compounds to diol-epoxides. This essential step is part of the process by which PAH compounds are converted to forms that can be conjugated with, for example, glucuronic acid and glutamic acid, rendered water soluble and excreted by the kidney (EC, 2001b).

The epigenetic effects of PAH compounds involve binding to a cytosolic receptor, aryl hydroxylase (Ah), translocation of the PAH-Ah complex into the nucleus, binding to a

nuclear transcription factor and activation of genes that regulate the expression of factors that control the cell growth and differentiation. This epigenetic effect does not seem to be dependent on initial conversion to diol-epoxides (EC, 2001b).

PAH compounds may also affect the production of cancer by triggering an inflammatory response and generating intracellular oxidative stress by free-radical production (EC, 2001b).

A more elaborated review of the mechanisms of carcinogenesis of PAHs can be found in Boström et al. (2002) and Harvey (1996).

4.3 Dose-response relationships for carcinogenicity

There is some evidence that a multiplicative model for cancer incidence, P_{cancer}, is the one best adaptable to experimental data on PAH carcinogenesis (Boström et al., 2002). This can be expressed as:

$$P_{cancer} = P_{initiation} \times P_{promotion}$$

In this approach, the probability of initiation, $P_{initiation}$, is modelled by a linear, non-treshold curve, and the probability (and intensivity) of promotion, $P_{promotion}$, by the S-shaped cumulative function. Therefore, the possibility that a threshold exists, arises. This can be explained by the hypothesis that initiation can occur at very low doses but this will not lead to cancer unless promotion occurs and this promotion component is characterized by a threshold. The problem is complicated by the fact that the steps involved in the promoter activity of PAH compounds (beginning with binding to the Ah receptor) lead to up-regulation of the enzymes involved in production of the diol-epoxides from PAH compounds, and these mediate the genotoxic effects of PAH compound (EC, 2001b).

However, the WHO Air Quality Guidelines report recommends use of the US-EPA default model, i.e. the linearized multistage model, although it acknowledges that this will be likely to produce a conservative estimate of risk, and despite the non-linear response often seen for high doses in animal tests (WHO, 2000; EC, 2001b).

4.4 Biomarkers for PAH exposure

A number of techniques have been developed for the biological monitoring of human exposures to PAHs. Benzo(a)pyrene has often been used as an indicator for the carcinogenic PAHs present in the environment. The methods most commonly used have been determination of PAHs and their metabolites in blood and urine (e.g. urinary 1-hydroxypyrene), measurement of mutagenicity in urine and faeces, chromosome aberrations and sister chromatid exchanges in peripheral blood lymphocytes, and DNA and protein adduct formation in the latter and in other tissues.

4.5 Classification of PAH compounds

Exposure to single PAH compounds does not occur in man and thus it has been impossible to classify individual PAH compounds as proven human carcinogens. The International Agency for Research in Cancer (IARC) has classified a number of individual PAH compounds as probable human carcinogens (category 2A) and a number of common mixtures of substances that include PAH compounds as carcinogenic to humans (category 1).

A summary of results of tests for genotoxicity and carcinogenicity for the PAHs under consideration and their respective US-EPA and IARC classification is shown in Table K1. A compilation of IARC evaluations of certain complex mixtures and occupational exposures involving exposures to PAH compounds is given in Table K2. On the IARC-website (IARC, 2005) a future meeting is announced in October 2005 on the basis of which a new document regarding PAHs will probably be produced (IARC, Vol. 92: Air Pollution, Part 1, Polycyclic Aromatic Hydrocarbons, 11-18 October 2005).

Table K1: Degree of evidence for carcinogenicity of PAH in experimental animals and overall evaluations of carcinogenicity to humans (IARC, 1983, 1987, 2005; US-EPA, 1993, 2005; WHO, 1998a).

PAH compound	US-EPA, 1993, 2005	IARC, 1983, 1987, 2005		WHO, 1998a	
		Humans	**Animals**	**Genotoxicity**	**Carcinogenicity**
Acenaphthene	D*	not assessed		(?)	(?)
Acenaphthylene	D	not assessed		(?)	No studies
Anthracene	D	3	I	-	-
Benzo(a)anthracene	B2	2A	S	+	+
Benzo(a)pyrene	B2	2A	S	+	+
Benzo(b)fluoranthene	B2	2B	S	+	+
Benzo(g,h,i)perylene	D	3	I	+	-
Benzo(k)fluoranthene	B2	2B	S	+	+
Chrysene	B2	3	L	+	+
Dibenzo(a,h)anthracene	B2	2A	S	+	+
Fluoranthene	D	3	I	+	(+)
Fluorene	D	3	I	-	-
Indeno(1,2,3-cd)pyrene	B2	2B	S	+	+
Naphthalene	D/C**	not assessed		-	(?)
Phenantrene	D	3	I	(?)	(?)
Pyrene	D	3	I	(?)	(?)

+: positive; -: negative; ?: questionable; ( ): results derived from small database;
I: inadequate evidence; L: limited evidence, S: sufficient evidence;
*: the IRIS database (April, 2005) does not provide a carcinogenicity assessment for this compound;
**: the IRIS database (April, 2005) classifies this compound as possible human carcinogen (C);
US-EPA classification: A: human carcinogen; B1: probable human carcinogen, limited human data available; B2: probable human carcinogen, sufficient evidence in animals but inadequate or no evidence in humans; C: possible human carcinogen; D: not classifiable as to human carcinogenicity; E: evidence of non-carcinogenicity for human;
IARC classification: 1: carcinogenic to humans; 2A: probably carcinogenic to humans; 2B: possibly carcinogenic to humans; 3: not classifiable as to its carcinogenicity to humans; 4: probably not carcinogenic to humans.

Table K2: IARC evaluations of certain complex mixtures and occupational exposures involving exposure to PAH compounds (IARC, 1987, 1987, 1989a,b,c, 1996, 1997, 2002, 2005).

Mixture / Exposure	IARC	Mixture / Exposure	IARC
Bitumen	3	Mineral oils	
Bitumen extracts	2B	Mineral oils, untreated	1
Carbon black	2B	Mineral oils, mildly treated	1
Coal dust	3	Mineral oils, highly refined	3
Coal tar pitches	1	Petroleum solvents	3
Coal tars	1	Shale oils	1
Creosotes	2A	Soots	1
Crude oil	3	Diesel exhausts	2A
Diesel fuels		Gasoline exhausts	2B
Diesel fuels, light	3	Tobacco smoke and smoking	1
Diesel fuels, marine	2B	Involuntary smoking	1
Fuel oils		Aluminium production	1
Fuel oils, heavy	2B	Coal gasification	1
Fuel oils, light	3	Coke production	1
Gasoline	2B	Petroleum refining	2A
Jet fuel	3		

On the basis of data derived from WHO/IARC and US-EPA/IRIS, the following contaminants are considered carcinogenic to humans: benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene.

Fluoranthene is suspected of human carcinogenicity (WHO, 1998). However, according to the IARC and US-EPA, this compound is not classifiable as to human carcinogenicity.

Anthracene, fluorene and naphthalene can be considered non-genotoxic compounds. With the exception of naphthalene, for which carcinogenicity is questionable due to the small database, these compounds are also considerd as non-carcinogenic by WHO (1998a). This is in agreement with the conclusion of US-EPA who classifies naphthalene as a possible human carcinogen. WHO classifies benzo(g,h,i)perylene als as a non-carcinogenic PAH, according to US-EPA, this compound is not classifiable as to human carcinogenicity; the IARC did not provide a carcinogenicity assessment.

It is presently unclear if acenaphthene, acenaphtylene, phenanthrene and pyrene are human carcinogens or not.

4.6 Comparative cancer potency

Most of the toxicity data for environmental chemicals are available for individual components. Hence, risks are calculated for individual compounds. Because PAHs occur in the environment as complex mixtures of varying composition, there is a need to develop reliable estimates of toxicity for these chemicals. Developing such estimates requires using toxicity data derived from experiments with the mixture of interest. However, PAHs are handicapped by the lack of mixture-specific toxicity data and thus necessitate the use of approximations to predict toxicity (Ramesh et al., 2004; Reeves et al., 2001).

The WHO-monograph on PAHs (WHO, 1998a) and the Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures (US-EPA, 2000a) describe three approaches commonly used to asses dose-response relationships for PAHs as a mixture:

(i) use of ***toxic equivalence factors** (TEFs)*:
This approach provides a cancer risk estimate for the whole mixture by summing the carcinogenic potential of individual PAHs relative to an index compound (e.g. benzo(a)pyrene).

(ii) the ***comparative potency*** approach:
The comparative potency approach assumes that toxicological modes of actions are the same form similar mixtures and that the potency of both mixtures in *in vivo* and *in vitro* bioassays is directly proportional to the potency of human.

(iii) the benzo(a)pyrene ***surrogate*** approach:
The surrogate approach estimates the potency of the PAH fraction of the a mixture of concern, based on the assumption that the cancer risk of this fraction is proportional to the level of an indicator PAH (in this case: benzo(a)pyrene) in the mixture. An assumption must be made that the composition of the PAH mixture of concern is sufficiently similar to a surrogate PAH mixture.

In the WHO-monograph, main advantages and disadvantages for each approach are given. They are summarized in Table K3. However, no definite recommendation is given. In the WHO Guidelines for drinking water quality (WHO, 1996, 1998b), the WHO states that it is assumed that the relative carcinogenicity of PAH compounds is similar for the oral and other routes of application. However, they also come to the conclusion that it cannot be assumed that the carcinogenic effects of individual PAHs are additive or that PAHs present in a mixture (e.g. coal tar) act in the same way as each PAH individually. Also, there is a considerable amount of evidence for enhancement or inhibition of carcinogenicity by other PAHs (WHO, 1998b). Considering this argumentation, they derived a guideline value for benzo(a)pyrene (and fluoranthene; cf. paragraph Toxicological reference values of PAHs). In the Air Quality Guidelines, the WHO reports that there are doubts about the scientific justification for the assumption that carcinogenic effects of PAHs to be additive. And although they recognized that the use of benzo(a)pyrene alone will probably underestimate the carcinogenic potential of airborne PAH mixtures, they chose this compound as an indicator (WHO, 2000).

Approach	Main advantages	Main disadvantages
Individual PAH approach	Clearly defined chemical species are assessed.	May underestimate risk due to all PAHs by considering only a few compounds.
	A good body of scientific literature is available to evaluate it.	Depends on extrapolation from animal models to humans.
	Not affected by variability in the composition of mixtures.	Resource-intensive, as monitored and analysis are required.
	Relative easy to apply in ambient environments affected by many sources.	
	Regulatory experience exists.	
Comparative potency approach	The risk of whole mixtures, rather than only a few compounds, is estimated.	Does not define the contribution of PAH to estimated overall risk.
	A good body of scientific literature is available to evaluate it.	Difficult to use for assessing speciated components of a mixture.
	Takes advantage of existing data on human carcinogenicity.	Risk estimates require estimates of the contributions of individual sources to the levels of organic compounds in the ambient environment.
	Simple and requires inexpensive monitoring.	The assumption that mixtures from the same source are associated with similar risks may not be supported by the available data.
		The levels of compounds extractable in organic solvents are not usually reported, and the analytical methods are not standardized.
Benzo(a)pyrene surrogate approach	Can be used to estimate risk of entire PAH components of a mixture.	May result in overestimate of the risk of PAHs within a mixture.
	Simple and based on a few testable assumptions.	Some PAHs, such as substituted ones, are not well represented by benzo(a)pyrene and must be considered separately.
	Well supported by the available data.	
	Relatively easy and inexpensive to apply for regulatory purposes.	
	Regulatory experience exists.	

In the Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures (US-EPA, 2000a), US-EPA also describes procedures for risk assessment using data on the mixture of concern (comparative potency approach), data on a toxicologically similar mixture (surrogate approach), and data on the mixture component chemicals (relative potency approach). It is observed that the state of the science varies dramatically for these three approaches. The whole-mixture procedures are most advanced for assessing carcinogenic risk, mainly because of the long use of *in vitro* mutagenicity tests to indicate carcinogenic potency. *In vitro* test procedures for non-cancer endpoints are still in the

pioneering stage. In contrast, the component-based procedures, particularly those that incorporate information on toxicologic interactions, are most advanced for non-carcinogenic toxicity. In the report and by analogy with the WHO, no single approach is recommended. Instead, guidance is given for the use of several approaches depending on the nature and quality of the data.

Use of TEF schemes in risk assessments is emerging. However, additivity of toxic effects (in this case carcinogenesis) of different PAH compounds, one of the assumptions of the TEF approach, is still debated. Studies suggest that in general, the carcinogenicity of a known PAH mixture (as a whole) is in good agreement with the sum of the respective potencies of its components, i.e. that additivity can be assumed (e.g. McClure & Schoeny, 1995), and that use of a TEF scheme would not be alter significantly the outcome of risk assessments. Nevertheless, one of the major objections of use of TEFs is that complex environmental mixtures differ from defined synthetic mixtures in that they contain not only PAHs of known carcinogenicity but also hundreds of PAHs and other potentially carcinogenic non-PAH compounds for which carcinogenicity has not been established. In this way, the overall carcinogenic risk of the mixture as a whole will be underestimated. Including other (especially highly potent) PAHs in the TEF scheme could considerably enhance the outcome of the risk assessment.

Another drawback of the TEF approach is that attempts to define the toxicities of PAH relative to benzo(a)pyrene are complicated by the fact that some carcinogenic PAHs are capable to initiate and promote tumors. PAH risk assessment could then be improved by mechanistic studies providing a better understanding of complex mixture interactions (Ramesh et al., 2002; Reeves et al., 2001).

Also, evaluation of several studies with various PAH mixtures revealed that the potency ratio between pure benzo(a)pyrene and the PAH mixture in the same assay is highly dependent on the exposure pathway and the target organ. The analysis of Schneider et al. (2002; Forschungs- und Beratungsinstitut Gefahrstoffe, Germany) led to the conclusion that the contribution of benzo(a)pyrene to the carcinogenic potency of the mixture depends on the exposure pathway and type of cancer observed, but is relatively constant for various PAH mixtures from industrial sources. They derived a cancer slope factor for oral PAH exposure, based on data from a recent animal feeding study with coal tar mixtures (Culp et al., 1998). By using incidence data for all exposure-related tumors, a slope factor for humans of 11.5 (human excess risk per oral lifetime exposure with 1 mg benzo(a)pyrene/kg.d in a PAH mixture) was obtained. They recommend the use of the derived oral slope factor for the risk assessment of PAH-contaminated soils (Schneider et al., 2002).

In Table K4 relative potencies of the PAH compounds under consideration compared with benzo(a)pyrene are given according to different authors. To calculate the relative contribution of the individual PAH to the carcinogenicity of various air mixtures, different TEF schemes were used by different countries (and authors). For example, in Swiss and French studies, the TEFs proposed by Nisbet & LaGoy (1992) were used. In Sweden, the TEF scheme by Larsen & Larsen (1998) was applied. In Canada, the PAHs were assigned TEF values of Meek et al. (1994). A more elaborated review of the derivation of the different TEF schemes can be found in Boström et al. (2002), WHO (1998a) or the primary literature. More aspects on TEFs and their use in risk assessment are discussed in Kameda

et al. (2005), Matsumoto et al. (1998), Pufulete et al. (2004), Reeves et al. (2001), Schneider et al. (2002), and Weyand et al. (1995).

5. Toxicological reference values of PAHs

5.1 WHO

5.1.1 Oral exposure

The 1993 WHO Guidelines for drinking-water quality recommend a guideline value for benzo(a)pyrene in drinking water of 0.7 µg/l, corresponding to an excess lifetime cancer risk of 10^{-5}. This guideline was based on an increased incidence of forestomach tumors in mice fed benzo(a)pyrene in the diet, quantified using the two-stage birth-death mutation model. The unit risk calculated was 0.46 per mg/kg.d, or in other words, a dose of 2.2×10^{-5} mg/kg.d corresponds to an excess lifetime cancer risk of 10^{-5}. There were insufficient data available to allow the derivation of drinking-water guideline values for other PAHs.

Evidence that mixtures of PAHs are carcinogenic in humans comes primarily from occupational studies of workers. Cancer associated with exposure to PAH-containing mixtures in humans occurs predominantly in the lung and skin following inhalation and dermal exposure, respectively. There are no data available for humans for the oral route. There are only a few animal carcinogenicity studies on oral administration. Further information is available on the carcinogenicity of single PAHs from experiments with dermal application (WHO, 1998b).

Summarizing, it is not possible to assess directly the risk of PAHs to humans for the oral route owing to a lack of human data. One must rely on animal data to estimate the risk of exposure to individual PAHs, not forgetting that humans are exposed to mixtures of PAHs and not to pure individual PAHs. The extrapolation of risk to humans from animal data is complicated: the relevance of forestomach tumors in rodents when considering extrapolation to humans is not clear. There is some indication that there are interspecies differences in the enzymes that activate PAHs. Further, intraspecies differences in susceptibility in humans may be due to differences in cytochrome P-450 enzymes.

Studies of Weyand et al. (1995) and Culp et al. (1996) give support to the guideline for benzo(a)pyrene, estimated as 0.7 µg/l. Therefore, this value is still recommened.

Table K4: Relative cancer potencies of PAHs.

PAH	CAL-EPA, 2002; Collins et al., 1998	Chu & Chen, 1984	Clement Associates, Inc., 1988; Krewski et al., 1989	Kalberlah et al., 1995	Larsen & Larsen, 1998	Malcolm & Dobson, 1994	McClure & Schoeny, 1995	Meek et al., 1994; ECHC, 1994	Muller, 1997; 2002; Muller et al., 1995a,b, 1996; MOE, 1997	Nisbet & LaGoy, 1992	Sloof et al., 1989	US-EPA, 1993 $	WHO, 1998b: Summary TEFs !	Range
Acenaphthene	ND	ND	ND	0.001	ND	0.001	ND	ND	ND	0.001	ND	ND-0	ND	ND-0.001
Acenaphthylene	ND	ND	ND	0.01	ND	0.001	ND	ND	ND	0.001	ND	ND-0.01	ND	ND-0.01
Anthracene	ND	ND	0.32 *	0.01	0.0005	0.01	ND	ND	ND	0.01	0	ND-0.01	ND	ND-0.32*
Benzo(a)anthracene	0.1	0.013	0.145	0.1	0.005	0.1	0.1	ND	0.014	0.1	0-0.04	0.1-0.145	0.1	ND-0.145
Benzo(a)pyrene	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Benzo(b)fluoranthene	0.1	0.08	0.14(1) @	0.1	0.1	0.1	0.1	0.06	0.11	0.1	ND	0.1-0.167	0.1	ND-0.167
Benzo(g,h,i)perylene	ND	ND	0.022	0.01	0.02	0.01	ND	ND	0.012	0.01	0.01-0.03	ND-0.01	§	ND-0.03
Benzo(k)fluoranthene	0.1	0.004 ***	0.066	0.1	0.05	0.1	0.1	0.04	0.037	0.1	0.03-0.09	0.01-0.02	0.1	0.004-0.1
Chrysene	0.01	0.001	0.0044	0.01	0.03	0.01	0.1	ND	0.026	0.01	0.05-0.89	0.001-0.01	ND	ND-0.89
Dibenzo(a,h)anthracene	1.09 #	0.69	1.1(1) **	1.0	1.1	1.0	1.0	ND	0.89	5.0 £	ND	1.0-1.11	1.0	ND-5.0 £
Fluoranthene	ND	ND	ND	0.01	0.05	0.001	ND	ND	ND	0.001	0-0.06	ND-0.01	§	ND-0.06
Fluorene	ND	ND	ND	0	ND	0.001	ND	ND	ND	0.001	ND	ND-0	ND	ND-0.001
Indeno(1,2,3-cd)pyrene	0.1	0.017	0.232	0.1	0.1	0.1	0.1	0.12	0.067	0.1	0-0.08	0.055-0.1	0.1	0-0.232
Naphthalene	ND	ND	ND	ND	ND	0.001	ND	ND	ND	0.001	0	ND	ND	ND-0.001
Phenanthrene	ND	ND	ND	0	0.0005	0.001	ND	ND	0.00064	0.001	0.01	ND-0	ND	ND-0.01
Pyrene	ND	ND	0.081	0.001	0.001	0.001	ND	ND	0	0.001	ND	ND-0	§	ND-0.081

ND: no data.

*: Nisbet & LaGoy (1992) refer to a study of Clement (1986) in which a TEF value of 0.32 for anthracene is given; Boström et al. (2002) also refer to the study of Clement (1986) -as cited by Nisbet & LaGoy (1992)- but do not give a TEF value for anthracene; instead, a TEF of 0.32 is given for anthantrene.

**: Nisbet & LaGoy (1992) refer to a study of Clement (1986) in which a TEF value of 1.1 for dibenzo(a,h)anthracene is given; Boström et al. (2002) also refer to a study of Clement (1986) -as cited by Nisbet & Lagoy (1992)- but give a TEF value of 1.11. WHO (1998a) refer to a study of Krewski et al. (1989) in which a TEF value of 1.11 for dibenzo(a,h)anthracene is given.

***: Nisbet & LaGoy (1992) refer to a study of Chu & Chen (1984) in which a TEF value of 0.004 for benzo(k)fluoranthene is given; Boström et al. (2002) also refer to a study of Chu & Chen (1984) -as cited by Nisbet & Lagoy (1992)- but give a TEF value of 0.04.

@: A TEF of 0.14 is given by Boström et al. (2002) who refer to Clement Associates, Inc. (1988) and Krewski et al. (1989); WHO (1998a) also refer to Krewski et al. (1989) but gives a TEF value of 0.141.

£: A TEF of 1.0 appears to be appropriate for high doses of dibenz(a,h)antracene but the TEF value of 5.0 is considered more likely to be applicable to environmental exposures (Nisbet & LaGoy, 1992).

#: A TEF of 0.4 was determined in 1994 by dividing the inhalation unit risk factor for dibenzo(a,h)anthracene by the inhalation unit risk factor of benzo(a)pyrene, 3.9×10^{-4} and 1.1×10^{-3} $(\mu g/m^3)^{-1}$ respectively (CARB & OEHHA, 1994; Collins & Alexeeff, 1994). Using the unit risks of 1.2×10^{-3} and 1.1×10^{-3} $(\mu g/m^3)^{-1}$ for dibenzo(a,h)anthracene and benzo(a)pyrene respectively, as rapported in CAL-EPA (2002), a TEF value of 1.09 can be calculated.

$: The original US-EPA document could not be retrieved. Different authors who cite US-EPA (1993) report different TEF values. In the table, for each PAH compound, a range is given on the basis of TEF values reported by Brown & Mittelman (1993), WHO (1998a) and Schneider et al. (2002).

!: Summary on the basis of Kalberlah et al. (1995), Krewski et al. (1989), Malcolm & Dobson (1994), McClure & Schoeny (1995), Muller et al. (1995a,b, 1996), Nisbet & LaGoy (1992), and US-EPA (1993).

§: Benzo(g,h,i)perylene, fluoranthene, and pyrene are not included owing to their negative or uncertain rating as carcinogens.

The WHO also calculated a health-based value for fluoranthene, derived from a 13-week oral gavae study in mice with a NOAEL of 125 mg/kg body weight per day, based on increased SGPT levels, kidney and liver pathology, and clinical and haematological changes. An uncertainty factor of 10,000 (100 for inter- and intraspecies, 10 for the use of a subchronic study and inadequate database, and 10 because of clear evidence of co-carcinogenicity with benzo(a)pyrene in mouse skin painting studies) gives a *TDI* of 0.0125 mg/kg body weight per day. Assuming a 60-kg adult drinking 2 liters of water per day with an allocation of 1% of the *TDI* to water, because there is significant exposure from food, a health-based value of 4 µg/l (rounded figure) can be calculated. However, since the presence of this compound in drinking-water does not represent a hazard to human health, the establishment of a numerical guideline value is not deemed necessary. On the other hand, they recommend the monitoring of fluoranthene as an indicator PAH in drinking water.

PAH compounds are currently not under revision by the WHO (WHO, 2005).

5.1.2 Inhalation exposure

PAH compounds vary in volatility, the less volatile compounds being associated with particulate matter. The proportion of different PAHs detected in different emissions and workplaces sometimes differ widely from each other and from PAH profiles in ambient air. However, the profiles of PAHs in ambient air do not seem to differ much from one area to another, although large variations may be seen under special conditions. Furthermore, the carcinogenicity of PAH mixtures may be influenced by synergistic and antagonistic effects of other compounds emitted together with the PAHs during incomplete combustion. It should also be recognized that in ambient air the carcinogenic 4 to 7 ring PAHs (representing the majority of PAHs) are preferentially attached to particles, and only a minor fraction, depending on the temperature, exists as volatiles. A few studies indicate that the toxicokinetic properties of inhaled benzo(a)pyrene attached to particles are different from those of pure benzo(a)pyrene alone. Virtually nothing is known about other PAHs in this respect (WHO, 2000).

Based upon epidemiological data from studies in coke-oven workers, a unit risk for benzo(a)pyrene as an indicator in air constituent is estimated to be 8.7×10^{-5} per ng/m³ which is the same as that established by WHO in 1987. The corresponding concentrations of benzo(a)pyrene producing excess lifetime cancer risks of 1/10,000, 1/100,000 and 1/1,000,000 are 1.2, 0.12 and 0.012 ng/m³ respectively. The WHO notes that similar risks have been derived from studies of individuals exposed to other mixtures containing PAHs and took into consideration some recent animal data from which a unit risk of the same order of magnitude can be derived.

5.2 US-EPA

In Table K5, toxicological reference values for non-carcinogenic and carcinogenic effects, both for the oral and inhalation exposure routes, are compiled as derived and reported by the US-EPA/IRIS (available on the IRIS-website).

Table K5: Toxicological values for PAHs (BCL, 1980; NTP, 1992; US-EPA, 1984, 1986, 1987, 1988, 1989a,b,c,d, 1990, 1991a,b, 1998, 2004b, 2005).

PAH	Non-carcinogenic effects			Carcinogenic effects (1/10^5)		
	Oral [mg/kg/d]	Inhalation [mg/kg/d]	Drinking water limit [mg/l]	Oral [mg/kg/d]	Inhalation [mg/kg/d]	Drinking water limit [mg/l]
Acenaphthene	$6x10^{-2}$	NA	2	NA	NA	NA
Acenaphthylene	NA	NA	NA	NA	NA	NA
Anthracene	$3x10^{-1}$	NA	10	NA	NA	NA
Benzo(a)anthracene	NA	NA	NA	NA	NA	NA
Benzo(a)pyrene	NA	NA	NA	$1,4x10^{-6}$ *	NA	$5x10^{-5}$ ***
Benzo(b)fluoranthene	NA	NA	NA	NA	NA	NA
Benzo(g,h,i)perylene	NA	NA	NA	NA	NA	NA
Benzo(k)fluoranthene	NA	NA	NA	NA	NA	NA
Chrysene	NA	NA	NA	NA	NA	NA
Dibenzo(a,h)anthracene	NA	NA	NA	NA	NA	NA
Fluoranthene	$4x10^{-2}$	NA	1	NA	NA	NA
Fluorene	$4x10^{-2}$	NA	NA	NA	NA	NA
Indeno(1,2,3-cd)pyrene	NA	NA	NA	NA	NA	NA
Naphthalene	$2x10^{-2}$	$8.6x10^{-4}$ **	0.1 / 0.7 $	NA	NA	NA
Phenanthrene	NA	NA	NA	NA	NA	NA
Pyrene	$3x10^{-2}$	NA	NA	NA	NA	NA

NA: Not assessed.

*: Calculated using an oral slope factor of 7.3 per mg/kg/d (geometric mean; range: 4.5-11.7 per mg/kg/d).

**: Calculated using a *RfC* of $3x10^{-3}$ mg/m³, an inhalation volume of 20 m³/d and a body weight of 70 kg.

***: The Maximum Contaminant Level (MCL) is $2x10^{-4}$ mg/l.

$: The Drinking Water Equivalent Level (DWEL), a lifetime exposure concentration protective of adverse, non-cancer health effects, that assumes all of the exposure to a contaminant is from drinking water is 0.7 mg/l. The concentration in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, based on exposure of a 70 kg adult consuming 2 l/d, and adjusted for possible carcinogenicity is 0.1 mg/l.

5.3 EC

As reported by the WHO (2000), there are no studies which show the effects on health of vapour phase PAH compounds. However, the current body of opinion is that the bulk of the key carcinogenic PAH compounds found in ambient air would be associated with particles and thus it is likely that a similar pattern of exposure will occur. Particles bearing PAH compounds will be inhaled and deposited in the airways in accordance with well understood physical principles. In their evaluation, the EC assumed that a significant proportion of the particles in both the industrial mixtures and the ambient aerosol are likely to reach the intra-thoracic part of the respiratory system, i.e., are likely to be of less than about 10 μm aerodynamic diameter. They also assumed that the bio-availability of PAH compounds associated with the ambient aerosol will not be significantly less than that of PAH compounds associated with particles in industrial mixtures.

A number of studies of the effects of benzo(a)pyrene and other PAH compounds involving inhalation and implantation have been undertaken in animals. These studies have been used to generate models linking exposure to PAH compounds and risk of lung cancer. The Working Group on Polycyclic Aromatic Hydrocarbons (EC, 2001b) has not used these studies in developing a limit value for PAHs. Their rationale for this was that (i) all quantitative extrapolations from animals to man involve assumptions about comparative, i.e. inter-species, sensitivity, and (ii) adequate human epidemiological studies are available.

However, they turned back to the animal data in developing their case for recommending that benzo(a)pyrene should be used as an indicator of the ambient PAH mixture (EC, 2001b).

By adopting benzo(a)pyrene as an indicator compound, determining the concentration of benzo(a)pyrene in ambient air and estimating the increased risk likely to be associated with the life-time exposure from a unit risk estimate derived from the studies of occupational exposures, a risk estimate for exposure to PAH compounds in ambient air is derived. Use of benzo(a)pyrene as an indicator does not at all require that the mixture of PAH compounds met with in ambient air should be identical with, or even similar to, that met with in the occupational setting, but only that benzo(a)pyrene should make a similar contribution to the total carcinogenicity of both (EC, 2001b).

Boström et al. (2002) argued that fluoranthene could be used as an additional indicator for the ambient mixture of PAH compounds, because it is an experimental mutagen and carcinogen in certain test systems and it occurs at relatively high concentrations in the environment. Also, in contrast to benzo(a)pyrene (which is found predominantly in the particulate phase), fluoranthene is considered to be a representative of more volatile PAHs and it is expected that the relative contribution of high-molecular PAHs, such as benzo(a)pyrene will probably decrease in the future when better diesel technology and qualities have become more common (Boström et al., 2002). However, the EC have not pursued this but included a recommendation that fluoranthene concentration in air should be monitored (EC, 2001b).

The unit risk (lifetime exposure to a mixture represented by 1 ng/m³ benzo(a)pyrene) based on a number of occupational studies, is in the range $80\text{-}100 \times 10^{-6}$. As a result of developing knowledge there is increasing uncertainty about the reliability of the unit risk estimate. The Working Group on Polycyclic Aromatic Hydrocarbons stated that 'We acknowledge that we know of no means of identifying which of the epidemiological studies listed is the most suitable for use as a basis for developing a Limit Value for PAH compounds. We recommend, nevertheless, that the Unit Risk estimate adopted by WHO (WHO, 1987, 2001) from the US coke oven workers study, i.e. 8.7×10^{-5}, be taken as a starting point for developing a Limit Value. This study has been considered in detail by a number of authors and the Unit Risk estimate produced is towards the centre of the Unit Risk estimates produced by the range of epidemiological studies listed above. To us this seems a reasonable choice.' Therefore, the Working Group determined 0.1 ng/m³ benzo(a)pyrene corresponding to an increased cancer risk (life-time exposure) of 1×10^{-5}.

The Working Group recommended that the EU should adopt an air quality limit of between 0.5-1.0 ng benzo(a)pyrene/m³, annual mean, measured in the PM_{10} fraction and expressed at ambient conditions. PM_{10} is the most appropriate measurement fraction because lung cancer associated with inhaled PAH compounds occurs both in the large airways and in the deep lung. Provisions for 'alert tresholds' to protect against short term exposures were considered inappropriate since there is no evidence for acute effects at likely ambient concentrations. They also concluded that the limit should be reviewed in the light of improved knowledge after 5 years; a suitable margin of tolerance could be 50%, and the limit should be attained by 2010.

Recently, the European Directive 2004/107/EC (European Parliament and the Council, 2005) has set a target value for benzo(a)pyrene in ambient air. This compound should be used as marker for the carcinogenic risk of PAH in ambient air. As already mentioned, this target value is not to be considered as an environmental quality standard but means a concentration in the ambient air fixed with the aim of avoiding, preventing or reducing harmful effects on human health and the environment as a whole, to be attained where possible over a given period. The Member States of the EU therefore need to take all necessary measures not entailing disproportionate costs to ensure that, as from 31/12/2012, the concentration of Cd in ambient air does not exceed 5 ng/m³ (for the total content in the PM_{10} fraction averaged over a calendar year).

5.4 TPH Criteria Working Group

The TPH (Total Petroleum Hydrocarbons) Criteria Working Group has established toxicity criteria for fate and transport fractions of TPH, appropriate for quantifying human health risk. By relying on the fate and transport fractions, human health risk can be evaluated using toxicity criteria that approximate the mixtures as they occur in the environment. To assign toxicity criteria to the fate and transport fractions, the Working Group compiled and reviewed available toxicity data for individual TPH constituents within each fraction for well defined mixtures that are components of several fractions and for whole products, such as gasoline and fuel oils (TPH Criteria Working Group, 1996, 1997). From these data, the Working Group derived toxicity criteria (for non cancer endpoints) for each fate and transport fraction. For aromatic TPH, the results are summarized in Table K6.

Table C6: Working Group toxicity criteria for aromatic TPH fractions (TPH Criteria Working Group, 1996a,a, 1997).

EC *	Oral *RfD* [mg/kg/d]	Inhalation *RfC* [mg/m³]	Critical effect
C_5-C_7 >C_7-C_8	0.2 (toluene, styrene)	0.4 (toluene)	Hepatotoxicity Nephrotoxicity
>C_8-C_{10} >C_{10}-C_{12} >C_{12}-C_{16}	0.04 (isopropylbenzene, naphthalene, fluorene, fluoranthene)	0.2 (C9 aromatics)	Decreased body weight
>C_{16}-C_{21} >C_{21}-C_{35}	0.03 (pyrene as conservative surrogate for this fraction)	Not available (fraction is not volatile)	Nephrotoxicity

*: EC: equivalent carbon number range as defined in TPHC Critical Working Group (1996a).

In this respect, acenaphthylene (C_{12}), naphthalene (C_{10}), and phenanthtrene (C_{14}) can be allocated an oral *RfD* of 4×10^{-2} mg/kg/d and a *RfC* of 2×10^{-1} mg/m³. An oral *RfD* of 3×10^{-2} mg/kg/d can be assigned to benzo(g,h,i)perylene (C_{20}).

5.5 MDEP

The Massachusetts Department of Environmental Protection (MDEP) developed in 1994 the first fractional approach to evaluate human health risks from oral exposures to mixtures of petroleum hydrocarbon compounds and developed oral *RfD*s for various fractions. However, fraction-specific toxixity values for inhalation exposures were not derived. Subsequent to this effort, the ad hoc workgroup TPH Criteria Working Group introduced a modified version of the fractional approach and derived fraction-specific oral *RfD*s and inhalation *RfC*s (cfr. above).

MDEP has used data available after its 1994 work to update its oral toxicity values and identify inhalation *RfC*s for the volatile petroleum hydrocarbon fractions specified in 1994. For aromatic fractions, MDEP recommends the toxicity values as given in Table K7.

Table K7: MDEP toxicity criteria for aromatic TPH fractions (MDEP, 2003).

Carbon range *	Oral *RfD* [mg/kg/d]	Critical effect	Inhalation *RfC* [mg/m³]	Critical effect
C_6-C_8	Evaluate each chemical in the series separately.	-	Use individual *RfC*s for compounds in this range.	-
C_9-C_{18}	0.03 (pyrene)	Nephrotoxicity	0.05 (as a surrogate toxicity number for the C_9-C_{18} aromatic TPH fraction which is based on mixture studies)	Body weight reduction, hepatic, renal, and developmental effects
C_{19}-C_{32}	0.03 (pyrene)	Nephrotoxicity	Not applicable (fraction is not volatile)	-

In this respect, acenaphthylene (C_{12}), naphthalene (C_{10}), phenanthtrene (C_{14}) benzo(g,h,i)perylene (C_{20}) can be allocated an oral *RfD* of 3×10^{-2} mg/kg/d. A *RfC* of 5×10^{-2} mg/m³ can be assigned to acenaphthtene (C_{12}) and phenanthtrene (C_{14}).

6. Methodology S-RISK

For the development of generic guideline values for the PAH compounds under consideration, two questions must be addressed: (i) are the respective PAHs threshold compounds, and (ii) is application of a TEF scheme appropriate. In the proposed methodology, these two issues are handled together on the basis of consensus of opinion.

6.1 Treshold versus no-treshold

Genotoxic carcinogenic PAHs are considered to have no threshold for (these) effects. IARC (1983, 1987, 2005), US-EPA (1993) and WHO (1998, 2005) provide toxicological evaluations on the carcinogenic and genotoxic properties of the PAHs considered. These evaluations are not always conclusive. Also, there were not always enough data available to assess the genotoxicity and carcinogenicity. In this case, the compound can be inputted to S-RISK as a compound with either threshold or no-treshold for effects.

6.2 TEF scheme

Although use of TEF schemes is debated, application of TEF values provides a relatively simple risk assessment approach. Considering the different methods and applied studies employed for the derivation of the different TEF schemes as summarized in Table K4, TEFs should best be expressed as order of magnitude. The proposed TEFs are derived taken into account the consistency of the different TEF schemes for each PAH compound. An overview of the TEFs is given in Table K8.

Although the use of the benzo(a)pyrene surrogate method could give a better (i.e. with less chance of underestimating) estimate of carcinogenic potency of a PAH mixture, the guideline method is directed towards individual PAHs (probably those for which it is expected that they contribute most to the carcenogenic potency of a PAH mixture). Changing the individual compounds approach towards a benzo(a)pyrene surrogate approach would have significant consequences for the guideline system and its application. As it was not the aim of the project, nor is it feasible within this context, to explore the impact of such a decision, it is found reasonable and scientifically defensible to use the TEF approach (including additivity assumptions) as a best estimate within the actual framework.

6.3 Overall evaluation and proposed toxicological input values

On the basis of classification of the considered PAH compounds as to their genotoxic and carcinogenic properties, and the proposed TEF scheme, the final suggested toxicological input scenarios and reference values for PAHs are presented in Table K8 and Table K9 respectively. In these tables, the TEFs and toxicological values used for the derivation of soil guideline values in Flanders and the Netherlands are also given.

Table K8: Summarizing classification of PAHs according to their genotoxic and carcinogenic properties and overview TEF values.

PAH	S-RISK		Vlier-Humaan – Flanders (Nouwen et al., 2001)		CSOIL – the Netherlands (Baars et al., 2001)	
	Proposed TEF	Proposed input scenario	TEF	Input scenario	TEF	Input scenario
Acenaphthene	0.001	NT	0.001	NT	0.001	NT
Acenaphthylene	0.01	NT	0.01	NT	0.01	NT
Anthracene	0	T	0	T	0	T
Benzo(a)anthracene	0.1	NT	0.1	NT	0.1	NT
Benzo(a)pyrene	1.0	NT	1.0	NT	1.0	NT
Benzo(b)fluoranthene	0.1	NT	0.1	NT	0.1	NT
Benzo(g,h,i)perylene	0	T	0	T	0	T
Benzo(k)fluoranthene	0.1	NT	0.1	NT	0.1	NT
Chrysene	0.01	NT	0.01	NT	0.01	NT
Dibenzo(a,h)anthracene	1.0	NT	1.0	NT	1.0	NT
Fluoranthene	0.01	NT	0.01	NT	0.01	NT
Fluorene	0	T	0	T	0	T
Indeno(1,2,3-cd)pyrene	0.1	NT	0.1	NT	0.1	NT
Naphthalene	0	T	0	T	0	T
Phenanthrene	0.001	NT	0.001	NT	0 *	T
Pyrene	0.001	NT	0.001	NT	0.001	NT

NT: no threshold for effects (genotoxic and carcinogenic compound);

T: threshold for effects;

*: phenanthrene is considered to be carcinogenic but its carcinogenic potency is extremely low (<0.001) and therefore a *TDI*-approach is applied (Baars et al., 2001).

6.3.1 Oral exposure

▪ *Carcinogenic effects*

The toxicological values corresponding with an excess cancer risk of 10^{-5} for (only) benzo(a)pyrene, derived by WHO and US-EPA are 2.2×10^{-5} mg/kg.d and 1.4×10^{-6} mg/kg.d respectively. The WHO value is preferred. When using the TEF scheme as proposed above, toxicological reference values can be calculated for each carcinogenic or supposed carcinogenic PAH compound.

▪ *Non-carcinogenic effects*

For non-carcinogenic effects, *RfD*s of US-EPA can be used (c.q. anthracene, fluorene and naphthalene). For those compounds for which no toxicological reference value is given, the use of *RfD*s, derived by the TPH Criteria Working Group is recommended. This is the case for benzo(g,h,i)perylene.

▪ *Drinking water*

A reference drinking water concentration can be calculated, using a (calculated) toxicological reference concentration for oral exposure. According to the WHO methodology, a 2 liter drinking water consumption for a person weighing 60 kg is assumed. For non-carcinogenic effects, 10% of the toxicological reference dose is the basis for the drinking water limit. For carcinogenic effects, the excess lifetime risk of $1/10^5$ is completely assigned to drinking water.

In case a calculated drinking water limit exceeds the water solubility, an additional adjustment is made. In case the drinking water limit corresponding to carcinogenic effects exceeds the drinking water limit for non-carcinogenic effects, the latter is used in the calculations.

6.3.2 Inhalation exposure

- *Carcinogenic effects*

The basis of the toxicological reference values is the unit risk of 8.7×10^{-5} per ng/m³ derived by WHO (1987, 2000) and accepted by the EC Working Group on Polycyclic Aromatic Hydrocarbons. The corresponding concentration of benzo(a)pyrene producing an excess lifetime cancer risk of $1/10^5$ is 0.12 ng/m³, the inhalation dose is 3.4×10^{-8} mg/kg.d. When using the TEF scheme as proposed above, toxicological reference values can be calculated for each PAH compound.

- *Non-carcinogenic effects*

For non-carcinogenic effects, *RfC*s of US-EPA can be used (c.q. naphthalene). If no *RfC* is available, a toxicological reference dose for inhalation exposure and a *RfC* can be calculated on the basis of the (oral) *RfD* (c.q. anthracene, fluorene and naphthalene). For those compounds for which no toxicological reference value is given, the use of *RfC*s, derived by the TPH Criteria Working Group is recommended. This is the case for benzo(g,h,i)perylene.

6.4 Comparison with Vlier-Humaan (Flanders) and CSOIL (the Netherlands)

6.4.1 Vlier-Humaan (Flanders)

For the derivation of the soil guideline values for PAHs in Flanders, the Flemish Institute for Technological Research (VITO) compiled toxicological data primary from WHO and US-EPA/IRIS (Nouwen et al., 2001). The following PAHs were considered to have carcinogenic potencies: acenaphthene, acenaphthylene, benzo(a)antracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenzo(a,h)anthracene, fluoranthene, indeno(1,2,3-c,d)pyrene, phenanthrene and pyrene.

The TEF-scheme reported in WHO (1998a) was taken as a starting point for the derivation of TEFs for the carcinogenic PAHs. Data of Nisbeth & Lagoy (1992) were omitted since their TEFs were believed to overestimate the carcinogenic risk. Considering the large uncertainties, only the order of magnitude of the remaining TEFs were appraised. Then, for each carcinogenic PAH, the largest TEF was taken.

Table K9: Toxicological values used in S-RISK and comparison with Vlier-Humaan (Flanders) and CSOIL (the Netherlands).

PAH	S-RISK						Vlier-Humaan – Flanders (Nouwen et al., 2001)						CSOIL – the Netherlands (Baars et al., 2001)	
	Non-carcinogenic effects			Carcinogenic effects ($1/10^5$)			Non-carcinogenic effects			Carcinogenic effects ($1/10^5$)			Non-carcinogenic effects	Carcinogenic effects ($1/10^5$) #
	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Inhalation [mg/kg.d]	Drinking water limit [mg/l] *	Oral [mg/kg.d]	Oral [mg/kg.d]
Acenaphthene	-	-	-	2.2×10^{-2}	3.4×10^{-5} (1.2×10^{-4} mg/m³)	1.8×10^{-1} £	-	-	-	2.2×10^{-2}	3.3×10^{-5} (1.2×10^{-4} mg/m³)	1.8×10^{-1} £	-	5×10^{-2}
Acenaphthylene	-	-	-	2.2×10^{-3}	3.4×10^{-6} (1.2×10^{-5} mg/m³)	7×10^{-2}	-	-	-	2.2×10^{-3}	3.3×10^{-6} (1.2×10^{-5} mg/m³)	7×10^{-2}	-	5×10^{-3}
Anthracene	3×10^{-1}	3×10^{-1} $ (1.1 mg/m³)	**6.81x10⁻²**	-	-	-	3×10^{-1}	3×10^{-1} $ (1.1 mg/m³)	**7.5x10⁻²**	-	-	-	4×10^{-2}	-
Benzo(a)anthracene	-	-	-	2.2×10^{-4}	3.4×10^{-7} (1.2×10^{-6} mg/m³)	7×10^{-3}	-	-	-	2.2×10^{-4}	$3.3.10^{-7}$ (1.2×10^{-6} mg/m³)	7×10^{-3}	-	5×10^{-4}
Benzo(a)pyrene	-	-	-	2.2×10^{-5}	3.4×10^{-8} (1.2×10^{-7} mg/m³)	7×10^{-4}	-	-	-	2.2×10^{-5}	3.3×10^{-8} (1.2×10^{-7} mg/m³)	7×10^{-4}	-	5×10^{-5}
Benzo(b)fluoranthene	-	-	-	2.2×10^{-4}	3.4×10^{-7} (1.2×10^{-6} mg/m³)	**2.48x10⁻³**	-	-	-	2.2×10^{-4}	3.3×10^{-7} (1.2×10^{-6} mg/m³)	**1.2x10⁻³**	-	5×10^{-4}
Benzo(g,h,i)perylene	3×10^{-2}	3×10^{-2} $ (1.1×10^{-1} mg/m³)	**4x10⁻⁴**	-	-	-	3×10^{-2}	3×10^{-2} $ (1.1×10^{-1} mg/m³)	**2.6x10⁻⁴**	-	-	-	3×10^{-2}	-
Benzo(k)fluoranthene	-	-	-	2.2×10^{-4}	3.4×10^{-7} (1.2×10^{-6} mg/m³)	**1.22x10⁻³**	-	-	-	2.2×10^{-4}	3.3×10^{-7} (1.2×10^{-6} mg/m³)	**7.6x10⁻⁴**	-	5×10^{-4}
Chrysene	-	-	-	2.2×10^{-3}	3.4×10^{-6} (1.2×10^{-5} mg/m³)	**2.78x10⁻³**	-	-	-	2.2×10^{-3}	3.3×10^{-6} (1.2×10^{-5} mg/m³)	**1.5x10⁻³**	-	5×10^{-3}
Dibenzo(a,h)anthracene	-	-	-	2.2×10^{-5}	3.4×10^{-8} (1.2×10^{-7} mg/m³)	7×10^{-4}	-	-	-	2.2×10^{-5}	3.3×10^{-8} (1.2×10^{-7} mg/m³)	**5x10⁻⁴**	-	5×10^{-5}
Fluoranthene	-	-	-	2.2×10^{-3}	3.4×10^{-6} (1.2×10^{-5} mg/m³)	4×10^{-3} £	-	-	-	2.2×10^{-3}	3.3×10^{-6} (1.2×10^{-5} mg/m³)	4×10^{-3} £	-	5×10^{-3}
Fluorene	4×10^{-2}	4×10^{-2} $ (1.4×10^{-1} mg/m³)	1.2×10^{-1}	-	-	-	4×10^{-2}	4×10^{-2} $ (1.4×10^{-1} mg/m³)	1.2×10^{-1}	-	-	-	4×10^{-2}	-
Indeno(1,2,3-cd)pyrene	-	-	-	2.2×10^{-4}	3.4×10^{-7} (1.2×10^{-6} mg/m³)	7×10^{-3}	-	-	-	2.2×10^{-4}	3.3×10^{-7} (1.2×10^{-6} mg/m³)	**1.10⁻⁴**	-	5×10^{-4}
Naphthalene	2×10^{-2}	8.6×10^{-4} (3×10^{-3} mg/m³)	6×10^{-2}	-	-	-	2×10^{-2}	8.6×10^{-4} (3×10^{-3} mg/m³)	6×10^{-2}	-	-	-	4×10^{-2}	-
Phenanthrene	-	-	-	2.2×10^{-2}	3.4×10^{-5} (1.2×10^{-4} mg/m³)	1.2×10^{-1} £	-	-	-	2.2×10^{-2}	3.3×10^{-5} (1.2×10^{-4} mg/m³)	1.2×10^{-1} £	4×10^{-2} &	-
Pyrene	-	-	-	2.2×10^{-2}	3.4×10^{-5} (1.2×10^{-4} mg/m³)	9×10^{-2} £	-	-	-	2.2×10^{-2}	3.3×10^{-5} (1.2×10^{-4} mg/m³)	9×10^{-2} £	-	5×10^{-2}

$: The same as the toxicological reference value for oral exposure.

*: Calculated on the basis of the toxicological reference value for oral exposure, assuming a 2 liter drinking water consumption per day for a person weighing 60 kg; for non-carcinogenic PAHs, the drinking water limits corresponds to 10% of the *TDI*.

£: Calculated on the basis of 10% of the non-carcinogenic toxicological reference value for oral exposure, assuming a 2 liter drinking water consumption per day for a person weighing 60 kg.

( ): The maximal concentration in air and the inhalation dose are linked by taking into account a body weight of 70 kg and a daily consumption of 20 m³ air/day.

6.81x10⁻²: Drinking water limits in bold and italic format are adjusted to the water solubility (since the calculated drinking water limit exceeds the water solubility).

#: In the Netherlands, the cancer risk estimate is expressed as an excess lifetime cancer risk of $1/10^4$. The toxicological values for the carcinogenic compounds were recalculated to a lifetime excess cancer risk of $1/10^5$.

&: Phenanthrene is considered to be carcinogenic but its carcinogenic potency is extremely low (<0.001) and therefore a *TDI*-approach is applied (Baars et al., 2001).

Toxicological values to express the excess lifetime cancer risks $(1/10^5)$ associated with oral and inhalation exposure for benzo(a)pyrene were derived from WHO (oral exposure: 2.2×10^{-5} mg/kg.d; inhalation: 1.2×10^{-7} mg/m³, 3.3×10^{-8} mg/kg.d).

For the non-carcinogenic PAHs, *TDI*s were used from US-EPA (anthracene, fluorene and naphthalene) or the Total Petroleum Hydrocarbons Criteria Working Group (benzo(g,h,i)perylene).

6.4.1 CSOIL (the Netherlands)

The National Institute of Public Health and the Environment (RIVM, the Netherlands) first evaluated 11 PAHs (anthracene, benzo(a)anthracene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, fluoranthene, indeno(1,2,3-c,d)pyrene, naphthalene, phenanthrene, and pyrene) in 1991 (Vermeire et al.) and 1993 (Vermeire). In 2001, RIVM re-evaluated the toxicological data of these 11 PAHs (Baars et al., 2001). Also acenaphthene, acenaphthylene, benzo(b)fluoranthene, benzo(j)fluoranthene, dibenzo(a,h)anthracene, and fluorene were evaluated at this time. For the carcinogenic risk estimation, the studies of US-EPA (1993) and Kalberlah et al. (1995) were considered and the largest of the two potency factors reported were used as TEFs. For phenanthrene, a *TDI*-approach is applied since although this compound is considered to be carcinogenic, its carcinogenic potency is extremely low (<0.001).

On the basis of the study of Kroese et al. (1999), a chronic oral gavage rat study with benzo(a)pyrene, a lifetime excess cancer risk $(1/10^4)$ of 5×10^{-4} benzo(a)pyrene per kg bw per day is used in the risk estimation of the carcinogenic PAHs.

Regarding the non-carcinogenic PAHs and phenanthrene, the *TDI*s were derived from the Total Petroleum Hydrocarbons Criteria Working Group. For anthracene, fluorene, naphthalene and phenanthrene a *TDI* of 4×10^{-2} mg/kg.d is used. A *TDI* of 3×10^{-2} mg/kg.d is used for benzo(g,h,i)perylene (Baars et al., 2001).

APPENDIX H: INPUT PARAMETER VALUES IN S-RISK

In Table L1 , the basic default input parameters in S-RISK and S-EPA are compared.

Table L1: Comparison between the S-RISK and S-EPA default input parameters.

Parameter	Description	Dimension	Value S-EPA	Value S-RISK
C_{ad}	Annual average dust concentration in inhaled air	mg/m³	41×10^{-3}	5×10^{-3}
DF_{ia}	Dilution factor indoor air to soil air	-	1/20,000	1/700 (no basement, medium to fine sand) 1/16,000 (no basement, silty sand) 1/52,000 (no basement, clayey loam) 1/1,100 (basement, medium to fine sand) 1/43,000 (basement, silty sand) 1/110,000 (basement, clayey loam)
DF_{gw}	Dilution factor groundwater to porewater	-	1/15 (KM) 1/30 (MKM)	1/15 (KM) 1/30 (MKM)
DF_{sw}	Dilution factor surface water to groundwater	-	1/4,000	1/4,000
f_{exp}	Fraction of time spent on the site	-	1 (KM) 0.33 (MKM)	1 (KM) 0.33 (MKM)
f_h	Fraction of vegetables grown on the site	-	0.3 (KM)	0.3 (KM)
f_{leaf}	Fractional consumption of root vegetables	-	0.5	0.24
f_{OC}	Organic carbon content	%	2	1
f_{root}	Fractional consumption of leaf and stem vegetables	-	0.5	0.76
R_{du}	Long-term dermal soil exposure per unit body weight	mg/kg.d	20 (child, KM) 7 (MKM)	20 (child, KM) 7 (MKM)
	Integrated lifetime dermal soil exposure	mg/kg.d	3 (KM) 1 (MKM)	3 (KM) 1 (MKM)
R_{id}	Long-term dust inhalation exposure per unit body weight	(mg/kg.d)/(g/m³)	-	0.016 (child, KM) 0.005 (child, MKM)
	Integrated lifetime dust inhalation exposure	(mg/kg.d)/(g/m³)	-	0.01 (KM) 0.003 (MKM)
R_{ig}	Long-term consumption of vegetables per unit body weight	kg/kg.d	0.01 (child, KM)	0.0079 (child, KM)
	Integrated lifetime consumption of vegetables	kg/kg.d	0.005 (KM)	0.0041 (KM)
R_{is}	Long-term soil intake per unit body weight	mg/kg.d	10 (child, KM) 0.3 (MKM)	7 (child, KM) 0.3 (MKM)
	Integrated lifetime soil intake	mg/kg.d	1.5 (KM) 0.1 (MKM)	1.3 (KM) 0.1 (MKM)

Table L1 (cont.): Comparison between the S-RISK and S-EPA default input parameters.

Parameter	Description	Dimension	Value S-EPA	Value S-RISK
R_{iv}	Long-term vapour inhalation exposure per unit body weight	(mg/kg.d)/(g/m³)	-	500 (child, KM) 170 (child, MKM)
	Integrated lifetime vapour inhalation exposure	(mg/kg.d)/(g/m³)	-	300 (KM) 100 (MKM)
R_{iw}	Long-term drinking water consumption per unit body weight	l/kg.d	0.067 (child, KM, and MKM GV)	0.067 (child, KM, and MKM GV)
	Integrated lifetime drinking water consumption	l/kg.d	0.03 (KM and MKM GV)	0.03 (KM and MKM GV)
ρ_b	Dry soil bulk density	kg soil/dm³ soil	1.5	1.69 (medium till fine sand) 1.56 (silty sand) 1.42 (clay loam)
θ_a	Soil air content	dm³ air/dm³ soil	0.2	0.3 (medium till fine sand) 0.276 (silty sand) 0.263 (clay loam)
θ_t	Soil total porosity	dm³ /dm³ soil	0.5	0.358 (medium till fine sand) 0.387 (silty sand) 0.444 (clay loam)
θ_w	Soil water content	dm³ water/dm³ soil	0.3	0.058 (medium till fine sand) 0.111 (silty sand) 0.181 (clay loam)

In Table L2, default input parameter values for the soil-atmosphere vegetation transfer pathway for PAHs in S-RISK are given.

Table L2:Default input parameter values soil-atmosphere vegetation transfer pathway for PAHs (S-RISK).

Parameter	Description	Dimension	Value S-EPA	Value S-RISK
A	Plant surface area	m²	-	5
a_{growth}	Sink term, loss by growth	d^{-1}	-	0.035
$a_{metabolism}$	Sink term, loss by metabolism	d^{-1}	-	0
$a_{photodegradation}$	Sink term, loss by photodegradation	d^{-1}	-	0
b_{cf}	Octanol-lipid correction factor	-	-	0.95
dw	Fresh to dry weight conversion factor (above-ground vegetation)	-	0.117	0.12
g	Conductance	m/d	-	86.4
k_w	Plant weathering constant	1/d	-	0.049
m	Regression constant, K_{VG}	-	-	$10^{-2.53}$
n	Regression constant, K_{VG}	-	-	1.09
Q_{transp}	Transpiration rate	m³/d	-	0.001
R_n	Annual rainfall	m/d	-	1.48×10^{-3} (Stockholm) 2.05×10^{-3} (Göteborg) 1.64×10^{-3} (Malmö)
R_w	Fraction retained after rainfall	-	-	0.3
ρ	Wet plant density	kg fw/m³	-	700
ρ_r	Wet root density	kg fw/m³	-	700
t	Time (growing period)	d	-	100
$\theta_{w,v}$	Volumetric plant water content	m³ water/m³ plant	-	0.65
$\theta_{l,v}$	Volumetric plant lipid content	m³ lipid/m³ plant	-	0.01
V	Plant volume	m³	-	0.002
V_d	Dry deposition velocity	m/d	-	43.2
W_p	Volumetric washout factor	-	-	105
Y_v	Plant yield	kg dw/m²	-	0.38

In Tables L3-L6, the compound specific parameter values in S-RISK are given (S-RISK database).

Table L3: S-RISK database (compound specific physico-chemical parameter values).

Chemical	MW [g/mol]	S [mg/l]	P [Pa]	H [-]	K_{OW} [l/kg]	K_{OC} [l/kg]	K_d * [l/kg]	K_{OA} [l/kg]
Cadmium	112.4	-	-	-	-	-	1.020×10^2	-
Acenaphthene	154.21	3.59	4.21×10^{-1}	7.49×10^{-3}	$10^{4.05}$	$10^{3.55}$	3.55×10^1	1.50×10^6
Acenaphthylene	152.20	6.71	9.45×10^{-1}	8.84×10^{-3}	$10^{3.94}$	$10^{3.23}$	1.70×10^1	9.85×10^5
Anthracene	178.23	6.81×10^{-2}	2.32×10^{-3}	5.67×10^{-3}	$10^{4.44}$	$10^{4.34}$	2.19×10^2	4.86×10^6
Benzo(a)anthracene	228.22	1.59×10^{-2}	1.68×10^{-5}	1.83×10^{-4}	$10^{5.83}$	$10^{5.24}$	1.74×10^3	3.69×10^9
Benzo(a)pyrene	252.56	3.23×10^{-3}	1.09×10^{-6}	2.60×10^{-4}	$10^{6.27}$	$10^{5.88}$	7.59×10^3	7.16×10^9
Benzo(b)fluoranthene	252.24	2.48×10^{-3}	8.91×10^{-6}	9.66×10^{-4}	$10^{6.32}$	$10^{5.93}$	8.51×10^3	2.16×10^9
Benzo(g,h,i)perylene	276.34	4.00×10^{-4}	2.99×10^{-8}	2.17×10^{-5}	$10^{6.91}$	$10^{6.52}$	3.31×10^4	3.75×10^{11}
Benzo(k)fluoranthene	252.24	1.22×10^{-3}	3.24×10^{-7}	2.30×10^{-3}	$10^{6.55}$	$10^{5.82}$	6.61×10^3	1.54×10^9
Chrysene	228.28	2.78×10^{-3}	1.96×10^{-6}	8.82×10^{-4}	$10^{5.78}$	$10^{5.12}$	1.32×10^3	6.83×10^8
Dibenzo(a,h)anthracene	278.36	7.73×10^{-4}	1.27×10^{-9}	8.72×10^{-6}	$10^{6.54}$	$10^{5.95}$	8.91×10^3	3.98×10^{11}
Fluoranthene	202.20	1.95×10^{-1}	4.48×10^{-3}	9.30×10^{-4}	$10^{5.19}$	$10^{4.97}$	9.33×10^2	1.67×10^8
Fluorene	166.22	2.03 g	2.29×10^{-1}	4.65×10^{-3}	$10^{4.19}$	$10^{4.15}$	1.41×10^2	3.33×10^6
Indeno(1,2,3-cd)pyrene	276.33	6.20×10^{-2}	1.35×10^{-8}	8.54×10^{-6}	$10^{6.28}$	$10^{7.09}$	1.23×10^5	2.23×10^{11}
Naphthalene	128.18	3.10×10^1	1.27×10^1	2.12×10^{-2}	$10^{3.38}$	$10^{3.17}$	1.48×10^1	1.20×10^5
Phenantrene	178.23	9.03×10^{-1}	3.99×10^{-2}	1.66×10^{-3}	$10^{4.50}$	$10^{4.15}$	1.41×10^2	1.90×10^7
Pyrene	202.27	1.52×10^{-1}	1.11×10^{-3}	9.51×10^{-4}	$10^{5.05}$	$10^{4.78}$	6.03×10^2	1.18×10^8

*: for PAHs, K_d is calculated as: $K_d = K_{OC} \times f_{OC}$, with $f_{OC} = 0.01$.

Table L4: S-RISK database (compound specific biological parameter values).

Chemical	BCF_{root} [(mg/kg dw)/(mg/kg dw)]	BCF_{stem} [(mg/kg dw)/(mg/kg dw)]	K_{pl} [(mg/kg fw)/(mg/kg dw)]
Cadmium*	0.158	0.483	0.031
Acenaphthene	2.32	2.32	0.421
Acenaphthylene	2.32	2.32	0.421
Anthracene	0.022	0.002	0.0009
Benzo(a)anthracene	0.007	0.015	0.0025
Benzo(a)pyrene	0.002	0.012	0.002
Benzo(b)fluoranthene	0.014	0.005	0.0012
Benzo(g,h,i)perylene	0.004	0.011	0.002
Benzo(k)fluoranthene	0.003	0.015	0.002
Chrysene	0.008	0.013	0.002
Dibenzo(a,h)anthracene	0.0003	0.0005	0.0001
Fluoranthene	0.029	0.023	0.004
Fluorene	0.005	0.009	0.002
Indeno(1,2,3-cd)pyrene	0.0001	0.0002	0.00003
Naphthalene	2.92	2.92	0.53
Phenantrene	0.041	0.031	0.006
Pyrene	0.011	0.021	0.004

*: on fresh weight basis: BCF_{root}: 0.024 (mg/kg fw)/(mg/kg dw) and BCF_{stem}: 0.052 (mg/kg fw)/(mg/kg dw).

Table L5: S-RISK database (compound specific toxicological parameter values).

Chemical	TDI_{oral} [mg/kg/d]	Cancer Risk (oral) [mg/kg/d]	Threshold	Background exposure in % of TDI [%]	RfC [mg/m³]	Cancer Risk (inh.) [mg/m³]	Threshold	f_{du} [-]	Drinking water limit [mg/l]	Intake by drinking water as fraction of TDI_{oral} [-]	Cancer class US-EPA	Cancer class IARC	Adjustment for acute toxicity
Cadmium	1.00×10^{-3}	NA	Y	2.50×101	5.00×10^{-6}	NA	Y	4.00×10^{-2}	3.00×10^{-3}	1.00×10^{-1}	B2	1	not adjusted
Acenaphthene	NA	2.20×10^{-2}	N	NA	NA	1.20×10^{-4}	N	1.30×10^{-1}	1.80×10^{-1}	NA	D	NA	not adjusted
Acenaphthylene	NA	2.20×10^{-3}	N	NA	NA	1.20×10^{-5}	N	1.30×10^{-1}	7.00×10^{-2}	NA	D	NA	not adjusted
Anthracene	3.00×10^{-1}	NA	Y	NA	1.10	NA	Y	1.30×10^{-1}	6.81×10^{-2}	NA	D	3	not adjusted
Benzo(a)anthracene	NA	2.20×10^{-4}	N	NA	NA	1.20×10^{-6}	N	1.30×10^{-1}	7.00×10^{-3}	NA	B2	2A	not adjusted
Benzo(a)pyrene	NA	2.20×10^{-5}	N	NA	NA	1.20×10^{-7}	N	1.30×10^{-1}	7.00×10^{-4}	1.00	B2	2A	not adjusted
Benzo(b)fluoranthene	NA	2.20×10^{-4}	N	NA	NA	1.20×10^{-6}	N	1.30×10^{-1}	2.48×10^{-3}	NA	B2	2B	not adjusted
Benzo(g,h,i)perylene	3.00×10^{-2}	NA	Y	NA	1.10×10^{-1}	NA	Y	1.30×10^{-1}	4.00×10^{-4}	NA	D	3	not adjusted
Benzo(k)fluoranthene	NA	2.20×10^{-4}	N	NA	NA	1.20×10^{-6}	N	1.30×10^{-1}	1.22×10^{-3}	NA	B2	2B	not adjusted
Chrysene	NA	2.20×10^{-3}	N	NA	NA	1.20×10^{-5}	N	1.30×10^{-1}	2.78×10^{-3}	NA	B2	3	not adjusted
Dibenzo(a,h)anthracene	NA	2.20×10^{-5}	N	NA	NA	1.20×10^{-7}	N	1.30×10^{-1}	7.00×10^{-4}	NA	B2	2A	not adjusted
Fluoranthene	NA	2.20×10^{-3}	N	NA	NA	1.20×10^{-5}	N	1.30×10^{-1}	4.00×10^{-3}	NA	D	3	not adjusted
Fluorene	4.00×10^{-2}	NA	Y	NA	1.40×10^{-1}	NA	Y	1.30×10^{-1}	1.20×10^{-1}	NA	D	3	not adjusted
Indeno(1,2,3-cd)pyrene	NA	2.20×10^{-4}	N	NA	NA	1.20×10^{-6}	N	1.30×10^{-1}	7.00×10^{-3}	NA	C	2B	not adjusted
Naphthalene	2.00×10^{-2}	NA	Y	NA	3.00×10^{-3}	NA	Y	1.30×10^{-1}	6.00×10^{-2}	NA	C	NA	not adjusted
Phenantrene	NA	2.20×10^{-2}	N	NA	NA	1.20×10^{-4}	N	1.30×10^{-1}	1.20×10^{-1}	NA	D	3	not adjusted
Pyrene	NA	2.20×10^{-2}	N	NA	NA	1.20×10^{-4}	N	1.30×10^{-1}	9.00×10^{-2}	NA	D	3	not adjusted

NA: not available or not applicable.

Table L5: S-RISK database (compound specific ecotoxicological parameter values).

Chemical	AWQ Fish [mg/l]	Dutch C-value ecotox [mg/kg]	CCME aq life clean up [µg/l]
Cadmium	NA	1.20×10^1	1.00×10^{-2}
Acenaphthene	NA	NA	NA
Acenaphthylene	3.10×10^{-5}	NA	NA
Anthracene	NA	NA	NA
Benzo(a)anthracene	3.10×10^{-5}	4.00×10^1	NA
Benzo(a)pyrene	3.10×10^{-5}	4.00×10^1	NA
Benzo(b)fluoranthene	3.10×10^{-5}	NA	NA
Benzo(g,h,i)perylene	3.10×10^{-5}	4.00×10^1	NA
Benzo(k)fluoranthene	3.10×10^{-5}	4.00×10^1	NA
Chrysene	3.10×10^{-5}	4.00×10^1	NA
Dibenzo(a,h)anthracene	3.10×10^{-5}	NA	NA
Fluoranthene	5.40×10^{-2}	4.00×10^1	NA
Fluorene	3.10×10^{-5}	NA	NA
Indeno(1,2,3-cd)pyrene	3.10×10^{-5}	4.00×10^1	NA
Naphthalene	NA	4.00×10^1	NA
Phenantrene	3.10×10^{-5}	4.00×10^1	NA
Pyrene	3.10×10^{-5}	NA	NA

NA: not available or not applicable.