7 Exposure and impact assessment

In the present work, the Impact Pathway Approach which originally focused on impacts following inhalation of toxic air pollutants by humans is extended to also cover impacts due to hazardous substances present in the media soil and water. As was argued in Chapter 2, human health impacts constitute the main component when estimating external costs. The exposure and impact assessment described in the following is, hence, exclusively addressing impacts on human beings. In contrast to the environmental fate model, the exposure as well as the impact assessment follow a spatial differentiation based on administrative units mostly according to the Nomenclature of Territorial Units for Statistics (Nomenclature des Unités Territoriales Statistiques, NUTS) used by the Statistical Office of the European Communities (EUROSTAT). Thus, the information that is available in a spatially-resolved way is attributed to the different administrative levels distinguished such as countries or municipalities (cf. section B.6).

Principally there are three main *routes of exposure*, i.e., the routes by which a chemical enters the body (United States - Environmental Protection Agency, 1992; World Health Organisation, 2000a; European Commission, 2003c):

1. inhalation: absorption through the lungs,
2. ingestion: absorption from the digestive tract, and
3. dermal absorption: penetration through the skin.

For exposure *pathways* through soil and water, the most important exposure *route* is ingestion noting that dermal exposure due to bathing and soil contact might principally also play a role (Mileson et al., 1999). In agreement with the European Union's Technical Guidance Documents (European Commission, 2003a), the dermal exposure route as well as ingestion of soil particles by humans are considered to be of importance only in the case of highly polluted soils. Only recently, however, the Directorates of Environment, Health and Research of the European Commission have jointly launched an initiative, termed 'Science, Children, Awareness, EU Legislation and Continuous Evaluation' (SCALE), in order to develop a European Environment and Health Strategy (European Commission,

2003f). As can be seen from the initiative's name, a focus is laid on children and their protection. Although mouthing behaviour is a rather normal phase of childhood development, deliberate soil ingestion also termed *pica* is considered relatively uncommon (United States - Environmental Protection Agency, 1997c). Additionally, penetration of substances through the skin is of much more concern when assessing occupational exposure (World Health Organisation, 2000a) and exposure via cosmetic products. The contribution of the dermal exposure route and soil ingestion to the overall exposure in situations of diffuse emissions is deemed negligible and, therefore, these are not further considered here.

Ingestion or the oral exposure route involves two main substrates: food and drinking water. Most (acute) heavy metal problems related to drinking water stem from the distribution system (pipes) and not from the source of the drinking water (World Health Organisation, 1992b; Becker et al., 1997; Wilhelm and Ewers, 1999; Bernigau et al., 2000). The case may be different for organic pollutants for which water treatment is not very efficient (Versteegh et al., 2001; European Commission, 2003a) and which may also lead to indoor inhalation exposure after volatilisation from tap water (e.g., McKone, 1993a; Georgopoulos et al., 1997). Nevertheless, the additional exposure due to additional human activities might still be substantial, at least in the long run. However, modelling drinking water exposure for all European residents is a task that nobody has addressed until now following a detailed site-dependent bottom-up approach that aims at giving best estimates rather than those based on conservative (reasonable) worst-case scenarios such as in European Commission (2003b) for the local scale. This is because ground water constitutes a major part of the drinking water resources (Scheidleder et al., 1999). Even at smaller scales one fails to model mass transfers in ground water aquifers due to lack of information (e.g., Eggleston and Rojstaczer, 2000). It also appears that ground water contamination due to heavy metals, for instance, is a very localised problem and is confined to areas with former or present mining activities in the case of heavy metals (Stanners and Bourdeau, 1995). Due to the lack of contamination as well as aquifer information, a modelling effort would at present result in rather unreliable concentration estimates. Thus, whereas the assessment of food ingestion is more readily feasible, the exposure via drinking water is for the moment not included in the modelling framework.

The present Chapter is divided into three parts. These describe:

1. the assessment of a substance's concentration in agricultural produce and freshwater fish,
2. the food intake, and
3. the impact assessment.

7.1 Concentration in food

In search of an existing exposure assessment scheme to be adopted for the estimation of external costs, mostly rather conservative exposure assessment frameworks have been encountered (e.g., European Commission, 2003c; International Atomic Energy Agency, 2001; cf. sections 3.1.3 and 3.1.4) which for example employ safety factors or assume protective values leading to overestimates rather than underestimates. This is desirable from a regulatory perspective. This is unacceptable, however, from a cost-benefit point of view where representative estimates are needed.

A step towards a less conservative and, thus, more representative exposure assessment is seen in the Human Health Risk Assessment Protocol (HHRAP, United States - Environmental Protection Agency, 1998). The HHRAP aims at consolidating information presented in other risk assessment guidance and methodology documents previously prepared for example by the US-EPA. Due to the fact that it constitutes a site-specific risk assessment approach, the degree of conservatism is reduced towards screening level risk assessments. Evaluating reasonable rather than theoretical worst-case maximum potential risks is recommended (United States - Environmental Protection Agency, 1998); conservative assumptions shall only be employed in order to prevent unacceptable potential damages. However, especially with respect to the exposure assessment a certain degree of conservatism is introduced: the exposure scenarios "are intended to allow standardized and reproducible evaluation of risks across most sites and land use areas, with conservatism incorporated to ensure protectiveness of potential receptors not directly evaluated, such as special sub-populations and regionally specific land uses" (p. 4-2). Thus, it is the intention of risk assessments to estimate so-called Reasonable Maximum Exposures (RME). The way how conservative elements are dealt with is described below.

United States - Environmental Protection Agency (1998) provides guidance for the assessment of ingestion exposure of

- belowground, aboveground protected and aboveground exposed produce,
- beef and dairy products, pork, chicken and eggs,
- drinking water, and
- (freshwater) fish.

Presently different types of produce (e.g., potatoes, cereals, spinach), pork, poultry, eggs, beef and dairy products, as well as freshwater fish are considered in the analysis. It has been argued above that drinking water is excluded basically due to data availability constraints. As regards fish, only freshwater fish is included albeit most of the fish eaten in Europe stems from sea catches (European Centre for Ecotoxicology and Toxicology of Chemicals, 1994). The disregard of

exposure via marine fish is due to the fact that sea fish is caught at very different places which would bring about the necessity to (a) assess the environmental fate of especially long-lived chemicals at the global scale (i.e., modelling the entire oceanic system on Earth) due to marine currents and migrating animals and (b) to include rather detailed trade patterns. Disregarding sea fish consumption leads to a substantial underestimation of impacts caused by those substances whose (effective) human exposure to a rather high degree is influenced by sea fish consumption such as methyl-mercury or dioxins (e.g., French et al., 1998; Buckley-Golder et al., 1999; Anonymous, 2000). On the other hand, tentatively assuming that all fish consumed stems from freshwater bodies may overestimate the potential impacts by 1.5 orders of magnitude (Huijbregts et al., 2000b). For the presently addressed trace elements, no attempt is, therefore, made to consider exposure due to consumption of marine fish.

In contrast to the TGD (European Commission, 2003c), the assessment scheme by United States - Environmental Protection Agency (1998) includes human exposure to pork as well as poultry meat and eggs. In particular exposure to pork is relevant since this is the dominating meat type consumed in Europe (European Centre for Ecotoxicology and Toxicology of Chemicals, 1994). One has to note, however, that the availability of substance-dependent data for the transfer from feed and/or soil into pigs and poultry is rather limited.

7.1.1 Considerations with respect to animal feed and ingested soil

The proper consideration of animal feed is fairly difficult. According to United States - Environmental Protection Agency (1998), cattle are fed forage, silage and grains, swine receive silage and grains, and poultry as well as laying hens only receive grains. However, there is hardly any production data available on forage and silage whereas 'grains' that are mostly bought on the market may vary substantially with respect to its constituents. With the exception of Corn Crop Mix (CCM) that is utilized in pig keeping only in regions where corn is grown, silage and forage are only fed to cattle. It is assumed here that forage and silage are grown and utilized on or at least near the farm to such an extent as to sustain cattle keeping while pigs similar to poultry and laying hens only get fed grains.

Grains are usually administered as mixed fodder consisting for instance of cereals (e.g., wheat and barley), legumes, and oil seeds and groats (such as soy beans). Apart from forage and silage, cereals constitute the largest quantity of the animal feed. For instance, of the 67.8 million tonnes of animal feed used in 2001/2002 in Germany, there were 30 million tonnes of forage and roughage and 25 million tonnes of cereals the remainder being concentrate (Anonym, 2002). In the average mixed fodder, the share of cereals is somewhat lower (42 %) according to Deutscher Verband Tiernahrung (2003).

The most important single components in mixed fodder are wheat and soy beans (Deutscher Verband Tiernahrung, 2003). The amounts of soy beans produced in Europe compared to those imported are small (Food and Agriculture Organization of the United Nations - Statistics Division, 2003) and are, therefore, not further considered. In contrast to the oil seeds and groats, cereals for feeding purposes are exclusively grown in Europe and not imported (104 % self-supply within the EU, Anonym, 2002).[15] The average share of wheat in mixed fodder produced in Germany in 2002/2003 for instance was 19.9 % (Deutscher Verband Tiernahrung, 2003). Total grain consumption of all animals included in the assessment is scaled by this figure to yield the exposure due to uptake of wheat taken as a proxy for the grain exposure.

Accidental swallowing of soil particles by farm animals is another exposure pathway which may contribute to human exposure towards hazardous substances. This exposure pathway depends on the degree to which the animals are kept outdoors (e.g., dioxins taken up by free-foraging hens, Anonymous, 2000). In fact, the consideration of the free-range share of the total amount produced is also necessary for some of the vegetal produces such as vegetables grown in greenhouses (e.g., tomatoes).

For cattle, it is assumed that they are kept in the free-range to a very large extent such that the exposure assessment towards soil particles as suggested by United States - Environmental Protection Agency (1998) is adopted for all cattle kept in Europe, i.e., the share of beef and cow milk produced in the free-range is set to 100 %. In fact, it appears as if the soil particle intake rate is smaller for grazing milk cows than for those that are fed grass silage (Berende (1990) quoted in McLachlan (1997)). Swine and poultry, in contrast, are kept indoors to considerable amounts in Europe. This differs by region and farm animal. For instance, the share of free-range eggs in the early 2000s amounted to 6.7 % (Anonym, 2003), 20 % (Anonymous, 2004) and 25 % (Geßl, 2004) in Germany, South East United Kingdom and Austria, respectively. The amount of pigs kept outdoors in the United Kingdom is estimated to lie between 30 % for suckling pigs, 11 % for weaner pigs and 0.3 % for finishing pigs yielding a weighted average of about 10 % (Edwards, 2004).[16] This share is only 1 % in Germany (Schulz, 2004). Free-range poultry kept on organic farms in Germany which can be taken as a lower bound estimate of the overall poultry kept outdoors was 0.6 % of the total in 2000 (Anonym, 2004). This share is about 20 % in France.[17]

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[15] Imported cereals are exclusively used for baking goods with high quality needs.

[16] The overall share of pigs kept outdoors in the UK was estimated to lie between 18 and 20 % in 1996 (Anonymous, 1996); this figure, however, most likely includes sows which constitute the largest contribution to pigs kept in the free-range (cf. the Danish situation, Temm, 2004)

The country-specific values as given above are adopted rounding the share for free-range poultry in Germany up to 1.0 % and taking 10 % for the pigs kept outdoors in the UK. For the other countries for which data are missing the default values assumed are 6.7, 1.0 and 1.0 % for free-range eggs, poultry and pigs, respectively. Although the share of free-range pigs is significant in Denmark (Geßl, 2004), the amount of fattened pigs for pork production is small while that of sows may be larger (Temm, 2004). The Danish share of free-range pigs is, therefore, also set to the default value.

7.1.2 Computation of human exposure

The concentration in food is computed according to the equations given in Table 7-2 (refer to section A.7 for more details). For the purposes of this work, the analysis is limited to the exposure pathways given there. This should not be interpreted as implying that transfers from other environmental media through alternate pathways (e.g., dermal absorption or ingestion of other food items) are unimportant. Inhalation exposure is estimated with the help of the EcoSense model (European Commission, 1999a) according to the procedure described in section A.7.2.

Generally, the values recommended by United States - Environmental Protection Agency (1998) are adopted. In case these were stated to be rather conservative, different values are assumed if provided (Table 7-1). Note that also with respect to the environmental fate assessment there are considerable deviations between the present approach and the one by the HHRAP. In particular the soil erosion and leaching to the subsurface soil layer are assumed to be zero according to the HHRAP. All these assumptions will overestimate a substance's concentration in soils. In WATSON, transport to the subsurface of soluble and (if chosen by the user) of particulate-associated substances as well as soil erosion are included for soil compartments (see sections A.3.3, A.3.7, A.3.6 and A.6.4). Due to vertical movement of substances in soils and root uptake also from the deeper parts of the soil, these soil compartments in turn are assumed to have a larger depth than one centimetre (cf. section 5.1) as assumed for untilled soil according to the HHRAP.

When determining the exposure frequency, one may need to take into account people's daily (e.g., between home and work, day care, school ...) or episodic (e.g., going on vacation, weekend trips) movement from locations with higher to lower exposure and vice versa. The difference in exposure levels in turn may depend on the emission scenario to be evaluated. Such differences will be more severe for point or (confined) line sources than for diffuse (multi-source) emissions. The differences will, furthermore, be more pronounced for inhalation

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[17] Found at http://www.free-rangepoultry.com/ as of May 2005.

Table 7-1: Parameter values adopted in the exposure assessment deviating from those recommended by the United States - Environmental Protection Agency (1998) for ingestion

Parameter	Unit	Value		
		US-EPA		Adopted
Soil ingestion by beef cattle	[kg DW per day]	0.5	(p. 5-48)	0.3
Soil ingestion by dairy cattle	[kg DW per day]	0.4	(p. 5-52)	0.2
Exposure frequency	[days]	350	(p. 6-12)	365
Exposure duration: children	[yr]	6	(p. 6-14)	n/a
Exposure duration: adults	[yr]	30-40	(p. 6-14)	70

and ingestion of locally grown and eaten food than for consumption of traded food items. United States - Environmental Protection Agency (1998) conservatively assumes that exposed people are only two weeks absent from the geographical area for which the exposure is assessed. This is defendable since the HHRAP constitutes a risk assessment framework for point sources and two weeks is the least amount of vacation that an employee gets in the US. Unless there is a net movement of people out of the area with a higher exposure, however, setting the exposure frequency to values lower than 365 days may actually underestimate the overall exposure at the (entire) population level. Many of the pollutants investigated here may be transported over longer distances when only released high enough into the atmosphere. Thus, when evaluating inhalation and ingestion of self grown/caught food due to a single point or confined line source emitting rather close to the ground shortening the exposure frequency at the population level may be valid, especially if the source is located in an unattractive area from a tourist's and/or business traveller's point of view. However, in all other cases this does not seem to be justified.

According to United States - Environmental Protection Agency (1998), exposure duration is defined as "the length of time that a receptor is exposed via a specific exposure pathway" (p. 6-13). The recommended values are shorter than a 70-year lifetime because US Americans on average do not remain in the same area over their entire life and, thus, not (necessarily) in the vicinity of a hazardous waste combustion facility. The necessity to take an exposure duration shorter than a lifetime into account may be due to the assumption that effects show thresholds.

Table 7-2: Exposure pathway formulations for ingestion exposures as used in the exposure assessment

Name	Formulation[a]	Refer to section ... for more details
Food concentrations derived from concentrations assessed for the different compartments by the soil and water fate model		
arable land - aboveground produce	$C\_w/fw = BCF\_dw/dw_{\text{plant/soil}}(p, r, e) \cdot fr\_w_{\text{solid phase/bulk}}(r, e) \cdot C\_w/dw_{\text{ag,solid}}$	A.7.6 (p. 443)
arable land - belowground produce	$C\_w/fw = emp_{\text{BCF, root crops}}(p, r, e) \cdot BCF\_dw/dw_{\text{root/soil}}(p, r, e) \cdot fr\_w_{\text{solid phase/bulk}}(r, e) \cdot C\_w/dw_{\text{ag, solid}}$	A.7.8 (p. 444)
pasture/arable land - silage/ forage - beef/milk cattle	$C\_w/fw = \{ING_{\text{feed}}(r_{\text{animal}}, e) \cdot BTF\_t/w_{\text{milk or beef/feed}}(p, r_{\text{animal}}, e)\} \cdot BCF\_dw/dw_{\text{plant/soil}}(p, r_{\text{plant}}, e) \cdot C\_w/dw_{\text{p\|ag,solid}}$	A.7.9 (p. 445) / A.7.10 (p. 447)
pasture/arable land - grains - beef/milk cattle	$C\_w/fw = fr\_w_{\text{wheat/total grain}}(r_{\text{animal}}, e) \cdot \{ING_{\text{feed}}(r_{\text{animal}}, e) \cdot BTF\_t/w_{\text{milk or beef/feed}}(p, r_{\text{animal}}, e)\} \cdot BCF\_dw/dw_{\text{plant/soil}}(p, r_{\text{plant}}, e) \cdot C\_w/dw_{\text{p\|ag,solid}}$	A.7.9 (p. 445) / A.7.10 (p. 447)
pasture (soil particles) - animal products	$C\_w/fw = \{BTF\_t/w_{\text{animal product/feed}}(p, r, e) \cdot ING_{\text{soil}}(r, e)\} \cdot fr\_w_{\text{free-range/total}}(r, n, e) \cdot C\_w/dw_{\text{p, solid}}$	A.7.11 (p. 447)
freshwater - fish	$C\_w/fw = BCF\_V/fw_{\text{fish/water}}(p, r, e) \cdot C\_w/v_{w,\text{aqueous}}$	A.7.12 (p. 448)

Table 7-2: Exposure pathway formulations for ingestion exposures as used in the exposure assessment

Name	Formulation[a]	Refer to section ... for more details
Food concentrations derived from exogenous inputs (i.e., atmospheric depositions)		
atmospheric deposition - aboveground exposed produce	$C\_w/fw = \frac{emp_{\text{plant surface loss}}(r,e) \cdot fr\_w_{\text{intercept/deposition}}(r,e)}{Y\_fw(r,n,e)} \cdot [ATMDEP_{\text{dry}}(s,p,z) + ATMDEP_{\text{wet}}(s,p,z) \cdot fr\_w_{\text{adhere/wet deposition}}(p,r,e)]$	A.7.4 (p. 441)

a.*ATMDEP*: atmospheric deposition [kg/m²/s]; *BCF_V/fw*: bioconcentration factor [m³/kg]; *BCF_dw/dw*: bioconcentration factor [-]; *BTF_t/w*: [s*capita/kg FW]; C_w/fw: estimated concentration in food [kg/kg FW]; C_w/dw: concentration in pasture soils 'p' or arable land 'ag' as predicted by the environmental fate model [kg/kg DW] (unit conversion according to the description in section A.7.1 performed); C_w/v: concentration in freshwater compartments '*w*' [kg/m³] (unit conversion according to the description in section A.7.1 performed); *emp*: empirical factor [-] or [s]; *fr_w*: mass fraction of a substance [-]; *ING*: ingestion rate of feed taken in by an animal [kg DW/capita/s]; *Y_fw*: yield of produce [kg FW/m²]; symbols in parentheses denote a parameter's dependency on the exposure assessment framework ('*e*'), administrative unit ('*n*'), pollutant ('*p*'), receptor (or crop, '*r*'), emission scenario ('*s*') and/or the zone ('*z*')

As a consequence, true individual exposures need to be known. However, when the effects are assumed not to show a threshold (cf. discussion in section 7.3) and the targeted quantity is the overall effect occurring at the population level, an exposure duration shorter than a lifetime is misleading provided that population mobility does not lead to a factual change in population density. The value of 70 years as used by Crettaz et al. (2002) is adopted here.

As will be presented below (section 7.2), the ingestion rates are formulated as consumption of an average individual of the population, i.e., without distinguishing between for example different age groups. Although consumption habits and amounts as well as body weight will be different between adults and children, there is no effect model available taking into account that effects occurring due to oral exposure to the substances investigated are prevalent for one population subgroup or the other (cf. section 7.3). The choice not to distinguish between different population sub-groups appears to be justified given the presently available effect information.

7.2 Trade of food, consumption and the effective Intake Fraction

In the previous section, it was explained how the food concentrations are processed. This section deals with the question: 'Who eats what and in which amounts leading to human exposure?'.

In particular unlike inhalation, the exposure via food does not exclusively lead to exposure of people living or staying in the contaminated environment. Owing to the efficient development of humankind to societies that are based on division of labour, people in the industrialised world like in Europe rely to a rather large degree, if not exclusively, on the production of the primary sector, i.e., agriculture, and to a lesser degree on homegrown products. Additionally, there is a demand to eat all different kinds of food irrespective of the season although these cannot be grown domestically in all countries throughout the year for example due to cold winters. Furthermore, some of the agricultural produces that are domestically produced have higher prices than those of food items produced abroad. All this results in a situation in which the produce is traded and transported over long distances leading to exposure of people even towards a rather localised source which live far away from the immediately affected environment.

Unless one aims at protecting the most exposed individual and especially when one tries to cover the impacts by a human activity as comprehensively as possible, such rather indirect impacts also need to be considered. In order to assess the exposure via ingestion, one, therefore, not only needs to take into account the environmental concentration of a contaminant and its transfer into plants and/or animals but also the trade of the 'carrier goods' food to the human population.

7.2.1 Consideration of trade

The approach taken in order to consider trade is in contrast to risk assessment frameworks where the conservative 'subsistence farmer exposure' scenario is often used (European Commission, 2003a). This means that food is only consumed close to where it was produced. Allowing for trade is in line with Pennington et al. (2005) who employed a 'production-based' approach where a so-called Intake Fraction (Bennett et al., 2002) assesses the portion of an emission that a population will be finally exposed to. The Intake Fraction is, thus, a good measure to base exposure-response functions on in order to get representative impact estimates (see below).

Due to the geographical scope of the approach presented, the export to regions outside of Europe as well as the import of toxic substances via food products is not addressed. Only the trade within Europe is considered. As an initial attempt, trade is assumed to lead to homogeneous food concentrations across the geographical scope of WATSON according to:

$$C\_w/fw(r, Europe, p, e)_{average} = \frac{t_{year} \cdot \sum_n C\_w/fw(r, n, p, e)_{theoretical} \cdot P(r, n)}{t_{year} \cdot \sum_n P(r, n)} \qquad (7\text{-}1)$$

where

C_w/fw : $C\_w/fw_{average}$: average concentration of substance *p* in food item *r* in the geographical scope of the assessment ('Europe') as a result of considering production data [$kg_{chemical}$ per kg_{food} FW]

$C\_w/fw_{theoretical}$: concentration of substance *p* in food item *r* at the administrative unit *n* which is theoretical as this concentration may be assessed to occur in an administrative unit in which no respective food item is produced [$kg_{chemical}$ per kg_{food} FW] (according to Table 7-2)

P : production rate of crop *r* in administrative unit *n* [kg FW per s] (defined as described in section B.6.1)

t : time for which the production rate is given [s], i.e., corresponding to one year.

It is applied to all produce that is traded (e.g., wheat as food and/or feed, and all animal products considered; see introduction to section A.7). In future developments, a more detailed approach may be realized in which the amount that

is eaten nationally is distinguished from that transported across national borders. An aggregation at least at the national level is suggested as food consumption/ supply data are only provided at this level within WATSON (see below and section B.6.2). There may be produces, however, that are not produced in one country but may as well be eaten in the respective country. This is the case for spinach for example. One cannot do without considering trade in such instances one way or the other unless one takes the risk to underestimate the overall exposure. The consideration of trade is, therefore, strongly recommended albeit its initial status of consideration at present.

7.2.2 Assessing human consumption of food

Human consumption data are given as nationally-averaged per capita values. These were taken from the FAO Food Balance Sheets ('food supply', cf. section B.6.2). Due to the fact that food supply data may overestimate the actual food consumption, a correction factor is introduced assuming that 5 % of the retailed food is not eaten for example due to loss and plate waste. No distinction between children and adults is made which seems to be appropriate as long as the effect information does not distinguish between these sub-groups of a population (cf. section 7.3).

The predicted substance concentrations in food are only valid for those food items that are produced within the geographical scope of the model. Therefore, it was checked to what degree the European food production can actually satisfy the demand of the same area. In general, the amounts produced in Europe can at least sustain consumption as regards the food groups considered in the assessment (cf. section B.6.2). Self-supply figures only consider net trade effects. Import of food (and feed) produced outside the geographical area covered by the assessment, however, leads to a 'dilution' of the predicted pollutant concentrations. This is because these imported goods are virtually unexposed due to the spatial limitation of the analysis. Nevertheless, it is assumed that people only take in food items produced within the area modelled if the self-supply at least amounts to 100 %. This is the case for all food groups analysed except spinach which shows a self-supply of only 97 %. Although the case of spinach may be regarded as insignificant, a correction factor is introduced in order for the exposure assessment to be applicable for any type of produce, regardless of the degree of self-supply. This correction factor is equal to those given in Table B-19 ('degree of self-supply') setting values larger than 100 % to unity ('value adopted').

Starting from the food concentrations as computed according to the equations given in Table 7-2, the effective personal intake rate is, thus, computed as:

$$IR\_p(r, n, p, e) = fr\_w_{\text{effective total}}(p, r, e) \cdot ING_{\text{human supply}}(r, n) \cdot (1 - fr\_w_{\text{not consumed food supply}}(r, e)) \cdot fr\_w_{\text{self-supply}}(r, e) \cdot C\_w/fw(r, n, p, e) \quad (7\text{-}2)$$

where

C_w/fw : concentration of substance p in food item r at the administrative unit n [$kg_{chemical}$ per kg_{food} FW] (according to Table 7-2, may consider trade of food)

fr_w : $fr\_w_{effective/total}$: mass fraction of substance p contained in food r leading to an effect [kg per kg] (defined in Table C-2)

$fr\_w_{self\text{-}supply}$: mass fraction of produce r produced in the geographical scope of the assessment [kg per kg] (defined in section B.6.2)

$fr\_w_{not\ consumed/food\ supply}$: mass fraction of (fresh) food that is produced and traded but not consumed [kg per kg] (defined in section B.6.2)

ING : ingestion of food item r by humans according to food supply information for the administrative unit n [kg FW/capita/s]

IR_p : effective personal intake rate of substance p contained in food item r by humans at the administrative unit n [kg/capita/s].

Note that the meaning of 'effective' is introduced in the following.

7.2.3 The effective Intake Fraction

As mentioned above, the overall exposure of a population is assessed by means of the population-based source-to-intake measure *Intake Fraction* (Bennett et al., 2002), sometimes also referred to as exposure efficiency (Evans et al., 2002). It is the fraction of a substance's mass released into the environment that is ultimately taken in by the human population as a result of food consumption, inhalation and dermal exposure. In case of ingestion, this implies that it aggregates the exposure towards different produces which may become contaminated due to different causes (e.g., ingestion of soil particles, forage, silage and grains by milk cattle; cf. section A.7). Each such cause-exposure chain starting at the result of the environmental fate model is termed *exposure pathway* here.

For the purpose of the present analysis, the Intake Fraction due to ingestion exposures is calculated as:

$$IF_{\text{ingestion}} = \frac{\sum_n \sum_r \sum_i IR\_p_{n,r,i} \cdot Population_n}{S} \qquad (7\text{-}3)$$

where:

IF : (effective) Intake Fraction of a substance for ingestion [$kg_{\text{effective exposure}}$ per kg_{released}]

IR_p : (effective) personal Intake Rate of the respective exposure pathway *i* related to produce/food *r* at administrative unit *n* [kg/capita/s]; see section A.7.14 for its computation

Population : population in administrative unit *n* [capita]

S : source strength of a substance [kg per s].

Note that in a spatially differentiated environmental fate and exposure model the concentration as well as the source strength need to be aggregated for the geographical scope of interest. The Intake Fraction for inhalation is computed similarly (cf. section A.7.2).

The term 'ultimately' in the definition of the Intake Fraction given above implies that it is usually defined for a steady-state situation. If dynamic calculations are performed in which the mass taken in as well as the amount emitted may accumulate over time *t* (in full years) the mathematical definition of the Intake Fraction needs to be adapted accordingly:

$$IF_{\text{ingestion},\,t} = \frac{\sum_n \sum_r \sum_i \sum_t IR\_p_{n,r,i,t} \cdot Population_n}{\sum_t S_t}. \qquad (7\text{-}4)$$

When performing the (human) impact assessment, it is of importance whether the chemical forms in the edible portions of the food items are available to humans (Markert, 1998; Welch and Norvell, 1999) and that these available forms have the potential to cause an adverse effect on a receptor. Examples are inorganic versus organic arsenic (Agency for Toxic Substances and Disease Registry, 2000b), mercury compounds (Boening, 2000) and chromium VI versus chromium III (Agency for Toxic Substances and Disease Registry, 2000a). It may even occur that the route of exposure by which most of a substance reaches a human being is not the most important one when it comes to assessing the actual impact of this substance. This could be demonstrated for marine fish and shellfish whose consumption leads to the highest exposure but not to the highest risk in the

case of arsenic (Seiwert et al., 1999; Baxter and Lewis, 2002). Gebel (1999) states that arsenic taken in via ingestion of marine fish and shellfish is generally excreted indicating that this exposure pathway towards arsenic may even be irrelevant in terms of adverse effects.

The concept of the Intake Fraction is, therefore, extended here to only cover that portion of a substance to which exposure occurs which may lead to an adverse effect, termed the *effective Intake Fraction*.

In order to arrive at the effective Intake Fraction, one needs to assess the portion of the overall mass of a substance taken in that may become effective. This would at best be done by distinguishing the different chemical species in the environmental fate model. However, due to the geographical coverage and the spatial resolution aimed for (cf. section 2.3), this procedure is not feasible especially for data availability reasons. Instead, a static value is introduced in the exposure assessment. Note that the 'Personal Intake Rate' in Eq. (7-3) already is given for the effective chemical form of the substance.

According to a report issued by the British Food Standards Agency (Baxter and Lewis, 2002), inorganic arsenic usually constitutes at most 3 % of the overall occurring arsenic in the food groups investigated which is consistent with previous results on sea fish (Muñoz et al., 1999). Due to detection limit constraints, a distinction into food groups with respect to the effective fraction is deemed inappropriate. As an upper bound estimate of the potential damages caused by inorganic arsenic the fraction of 3 % is retained for all types of food products. The data situation is even worse for the other candidate for which a distinction between total and effective food contents needs to be made, chromium (Anonymous, 2002). Hexavalent chromium tends to be a strong oxidizing agent (Gauglhofer and Bianchi, 1991) which is also used in environmental chemistry in the form of potassium dichromate in order to determine the Chemical Oxygen Demand (COD) of sewage effluents (Bliefert, 1997). Because of its strongly oxidizing nature, it, thus, is readily reduced in the environment to its trivalent form with the exception of sea water (Gauglhofer and Bianchi, 1991; Agency for Toxic Substances and Disease Registry, 2000a). Furthermore, hexavalent chromium ions are assumed to be mostly reduced in human bodily fluids (Irwin et al., 1998; Anonymous, 2002) additionally decreasing the effective portion of the amount taken in. In a recent report by the Environment Agency of the United Kingdom (Anonymous, 2002), it was suggested to assume a (conservative) value of 10 % for the hexavalent state of the total chromium content in food products. This value is adopted here, again as an upper bound estimate.

Unlike the primary proposal, the (effective) Intake Fraction may also be given for sub-populations of the geographical scope of the model (like populations of different countries) and/or integrated only over a certain time period rath-

er than over infinity. Heijungs (1995) has shown that the steady-state solution of a multimedia model can under certain conditions help to assess the time-integrated exposure to pulse emissions which does not require dynamic computations. The prerequisites are: the model must be linear, the matrix must be non-singular and the system should converge to a steady-state when integrating over infinity. Although all these prerequisites are fulfilled by the modelling approach adopted (cf. section A.1) and the processes considered, the convergence criterion is critical because the integrating time for non-degradable substances might be rather long, i.e., in the order of centuries and longer. The effective Intake Fraction of trace elements is, therefore, also computed for the dynamic case in order to get an idea of the time scales involved for the development to steady-state (see also footnote f of Table 2-3).

7.3 Impact assessment

In order to assess the impacts from exposures to substances, it is generally preferable to use dose- or exposure-response relationships in order to estimate effects that can be derived based on observations on human populations. Combining these effects with an appropriate measure of severity then yields impacts (see below). As experiments at least with human beings are not ethically defendable, the best information available is provided by epidemiological investigations. Epidemiologically derived exposure-response functions are widely used in the context of human health assessments due to inhalation for policy decision purposes (e.g., European Commission, 1999a; Friedrich and Bickel, 2001a).

In contrast to inhalation, however, the available information for exposure-response functions due to food ingestion is scarce (Searl, 2002; Agency for Toxic Substances and Disease Registry, 2003; United States - Environmental Protection Agency, 2005). Most effect information is given as thresholds like No Observed Adverse Effect Levels (NOAEL) or Lowest Observed Adverse Effect Levels (LOAEL). Such measures bring about two main problems in the context of pan-European external cost assessments:

1. during marginal external cost assessments, a threshold-based effect measure 'punishes' the human activity that emits the final amount of a substance causing the threshold to be exceeded by holding it responsible for all effects to occur. However, there were usually other human activities as well that used up the 'assimilative capacity of the environment' (Pearce and Turner, 1990) or from an exposure perspective the human population's ability to accommodate emissions, i.e., the 'erosion of the available Margin Of Exposure' (MOE, Crettaz et al., 2002). Comparative analyses are, thus, hampered.

2. in order to decide whether a threshold exceedance is likely to occur, true environmental concentrations need to be estimated. Due to limited resources and imperfect information for instance on all emissions and processes influencing the environmental fate of a substance, a modelling exercise at the regional scale needs to fail to predict true environmental concentrations if it does not succeed by accident.

It is consequently necessary to look for alternatives.

7.3.1 Approach by Crettaz and co-workers

Recently, an approach has been proposed to also convert threshold effect information into linear so-called β_{ED10} *slope factors* for cancer (Crettaz et al., 2002) and non-cancer effects (Pennington et al., 2002) based on Crettaz (2000). Drawing on the benchmark dose BMD_{10} concept (Crump, 1995) that is discussed within the US-EPA (e.g., United States - Environmental Protection Agency, 1995), the effective dose ED_{10h} is the maximum likelihood (rather than the 95 % lower confidence limit for the BMD_{10}) estimate of the dose corresponding to 10 % response of humans over background.[18] It is derived by fitting a steady model through a discrete set of measured dose-response data employing a so-called linear multistage model. The ED_{10h} is taken as the point of departure in order to extrapolate to lower doses. It is assumed that the dose-response curve is linear and crosses at the origin for substances not showing thresholds in their effects. The slope factor β_{ED10} is, thus, computed as (Crettaz et al., 2002):

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[18] Note that Crettaz et al. (2002) use a variable with the acronym q1* when directly deriving the β_{ED10} *slope factor* from linear exposure-response information. When applying Eq. (7-12), Crettaz (2000) makes use of different kinds of linear exposure-response models, i.e., *slope factors* or *unit (lifetime) risks* in US-EPA (United States - Environmental Protection Agency, 1996b) or WHO terminology (World Health Organisation, 2000b), respectively. Both measures relate a risk or the probability of an individual to develop a disease (especially cancer) due to a lifetime exposure to a substance. The 'unit' in the WHO's name hints at the risk that a continuous exposure to one microgram per litre in water or one microgram per cubic metre in air poses. These units indicate that they need to be adjusted to match the US-EPA's slope factor (cf. Table 7-4). - The q_1* is estimated as follows: In order to fit a dose-response curve through a set of bioassay data, the US-EPA suggests the use of the linearised multistage model (LMS) which allows for non-linearities at high doses but forces a linear component at low doses. The respective linear low-dose slope is termed *slope factor* (United States - Environmental Protection Agency, 1996b) and denoted by q_1, the corresponding 95 % upper bound confidence limit by q_1*. The latter, thus, introduces an element of conservatism which the β_{ED10} *slope factor* approach intends to overcome. For more information, refer to Crettaz et al. (2002) and the literature cited therein.

$$\beta_{ED10}(p) = \frac{0.1}{ED_{10h}(p)} \tag{7-5}$$

where

β_{ED10} : slope factor for substance p based on effective dose affecting 10 % of a population over background [individual lifetime risk per mg/kg Body Weight/day]

ED_{10h} : maximum likelihood estimate of the effect dose of substance p inducing an added response of 10 % over background incidence for humans [mg/kg Body Weight/day]

0.1 : human response level corresponding to the dose ED_{10h} [-].

The slope factor, thus, represents a measure for the population-averaged excess individual risk of an effect per unit daily dose for a lifetime exposure (70 years for humans). The linearisation is based on a non-threshold assumption. In contrast to non-carcinogenic effects (Pennington et al., 2002), it is generally well accepted that there is no threshold for genotoxically acting carcinogens even at the individual level (International Programme on Chemical Safety, 1999; World Health Organisation, 2000b; Tennant, 2001). On the other hand, the linearisation is considered justified irrespective of the type of effect due to the growing recognition that 'no evidence' does not necessarily mean 'no effect' and that bioassays cannot give real insights on linearity or non-linearity at low doses, which only depend on the extrapolation model adopted. While toxicologists argue that mechanistic threshold concentrations or doses may exist for human health effects for many (non-genotoxically carcinogenic) substances, usually it has not been possible to establish the existence of mechanistic thresholds in epidemiological studies (e.g., European Commission, 1999a). Populations consist of individuals that show different susceptibilities or sensitivities to develop the investigated diseases even at low ambient levels (Hurley and Miller, 2001). Additionally, it may be argued not to assume thresholds from a precautionary principle perspective which is adopted by the European Council (European Commission, 2000a). However, care must be taken not to bias the assessment through rather conservative approaches.

The ED_{10h} from which the β_{ED10} *slope factor* is derived according to Eq. (7-5) can be estimated from different threshold effect measures (Crettaz, 2000; Crettaz et al., 2002; Pennington et al., 2002). The estimation schemes employed in this study are given in Table 7-3. It must be emphasized that the derivation particularly of the LOAEL and NOAEL strongly depends on the experimental design. As a result, also the ED_{10} and, thus, the β_{ED10} are more uncertain when these are derived based on threshold effect data. It is, therefore,

strongly suggested to derive the β_{ED10} from *slope factors* or *unit (lifetime) risks* in US-EPA (United States - Environmental Protection Agency, 1996b) or WHO terminology (World Health Organisation, 2000b), respectively, if sufficient data from laboratory bioassays are available. The respective equations are given in Table 7-4 which distinguishes between inhalation and ingestion exposures.

Before addressing the issue how health effects shall be quantified, a note on the consideration of mixtures in the present study shall be made. Effect assessments of mixtures still constitute a rather open field of research (e.g., Steinberg et al. 6/1995; Escher and Hermens, 2002). Therefore, the potential for more than additive or antagonistic interactions of different substances (Mücke 6/1995; Kroes, 1996) is at present not taken into account during the effect assessment of this study. As a result, the different contaminants are assumed to exert their effects in a non-interactive and additive way for example by simple similar or simple dissimilar action (Kroes, 1996).

Because 'effects' in general may lead to consequences with different severities like acute death or some short-lived skin irritation, it is necessary from a valuation perspective to distinguish between such diverse effects. In case of fatal diseases, the concept of Years of Life Lost (YOLL) is recommended in different contexts (Murray, 1994; European Commission, 1999a; Krewitt et al., 2002). The YOLL indicator measures the reduction in life expectancy resulting from an increased level of exposure to pollutants in the environment. In order to also account for effects related to morbidity, Crettaz et al. (2002) and Pennington et al. (2002) make use of the Disability Adjusted Life Years (DALY) concept (according to Murray and Lopez, 1996a, 1996b cited therein). It comprises the effects measured by the YOLL indicator and adds the measure Years of Life lived with a Disability (YLD). Although the approach has some disadvantages related to the derivation of the YLD and when applied to non-cancer effects (see below), it is deemed a step towards a more differentiated assessment of cancers for whose valuation only one generic monetary value for any type of cancer is used according to the latest ExternE methodology (European Commission, 2004).

The β_{ED10} *slope factor* is combined with the DALY concept in order to yield the *effect factor* according to the terminology of Life Cycle Impact Assessment (LCIA). As the slope factor gives the increase in risk for one 'standard' individual if he/she is continuously (i.e., daily) exposed to a given dose, one needs to divide this cumulative dose by the individual's lifetime in order to allow for analyses of an exposure situation that lasts potentially shorter than a lifetime (Hurley and Miller, 2001). This way, it is implicitly assumed that the increase in risk depends linearly on the mass of a substance taken in regardless of when and for how long the exposure takes place. In order to express the effect factor on a personal level, one needs to divide additionally by the 'standard' body weight.

Table 7-3: Estimation of ED_{10} from threshold effect measures

Threshold effect measure	Remarks	Equation[a]	
BMD_{10}	Equation 4 in Pennington et al. (2002) solved for ED_{10h}	$ED_{10h} = \frac{BMD_{10}}{0.54}$	(7-6)
BMC_{10}	Equation 4 in Pennington et al. (2002) solved for ED_{10h} and adapted to inhalation exposures	$ED_{10h} = \frac{BMD_{10}}{0.54} = \frac{INH}{BW} \cdot \frac{BMC_{10}}{0.54}$	(7-7)
LOAEL	Equation 8 in Pennington et al. (2002)	$ED_{10h} = \frac{0.3 \cdot LOAEL_{\text{animal, subchronic}}}{emp_{\text{animal} \rightarrow \text{human}} \cdot emp_{\text{subchronic} \rightarrow \text{chronic}}}$	(7-8)
NOAEL	Equation 7 in Pennington et al. (2002)	$ED_{10h} = \frac{1.6 \cdot NOAEL_{\text{animal, subchronic}}}{emp_{\text{animal} \rightarrow \text{human}} \cdot emp_{\text{subchronic} \rightarrow \text{chronic}}}$	(7-9)
TD_{50} from rats	Equation 8 in Crettaz et al. (2002) updated by Keller (2005) for application to rat bioassays	$ED_{10h} = \frac{TD_{50,\text{ rats}}}{18}$	(7-10)
TD_{50} from mice	Equation 8 in Crettaz et al. (2002) updated by Keller (2005) for application to mouse bioassays	$ED_{10h} = \frac{TD_{50,\text{ mice}}}{39}$	(7-11)

a. BMC_{10}: benchmark (air) concentration [mg/m^3]; BMD_{10}: benchmark dose [mg/kg Body Weight/day]; BW: body weight [kg per person], here: 70; ED_{10h}: maximum likelihood estimate of the effect dose of a substance inducing an added response of 10 % over background incidence for humans [mg/kg Body Weight/day]; *emp*: extrapolation coefficients from animal to human (if applicable: 1.6 for dogs, 6 for rats, 13 for mice, otherwise 1) and from subchronic to chronic exposures (if applicable: 3.3, otherwise 1) [-] (cf. Pennington et al. (2002)); INH: inhalation rate [m^3 per capita and s], here: 20 m^3 per capita and day (cf. section A.7.2); $LOAEL$: Lowest Observed Adverse Effect Level [mg/kg Body Weight/day]; $NOAEL$: No Observed Adverse Effect Level [mg/kg Body Weight/day]; TD_{50}: median Tumor Dose [mg/kg Body Weight/day]; *0.3*, *1.6*, *18*, *39*: linear regression coefficients [-]

Table 7-4: Estimation of the β_{ED10} slope factor based on linear exposure-response information (cf. footnote 18)

Route of exposure	Remarks	Equation[a]	
Ingestion, food	Equation 5 in Crettaz et al. (2002), $r^2 = 0.95$	$\beta_{ED10} = 0.5 \cdot ERF\_dose$	(7-12)
Ingestion, water	Conversion of water concentrations into dose based on average drinking rate and body weight; note that Eq. (7-15) partly reverses the conversion from *ERF_conc* into *ERF_dose*	$\beta_{ED10} = 0.5 \cdot ERF\_dose = 0.5 \cdot \frac{BW}{ING_{water}} \cdot ERF\_conc_{water}$	(7-13)
Inhalation	Conversion of air concentrations into dose based on average inhalation rate and body weight; note that Eq. (7-15) partly reverses the conversion from *ERF_conc* into *ERF_dose*	$\beta_{ED10} = 0.5 \cdot ERF\_dose = 0.5 \cdot \frac{BW}{INH} \cdot ERF\_conc_{air}$	(7-14)

a. β_{ED10}: slope factor for a substance based on effective dose affecting 10 % of a population over background [individual lifetime risk per mg/kg Body Weight/day]; *BW*: body weight [kg per person], here: 70; *ERF_conc*: linear exposure-response information for inhalation [individual lifetime risk per mg/m^3] or ingestion of water [individual lifetime risk per mg/l], usually given as *unit (lifetime) risk*; *ERF_dose*: linear exposure-response information particularly for ingestion of food [individual lifetime risk per mg/kg BW/day]; ING_{water}: drinking rate [l/day], here: 2 as for adults (United States - Environmental Protection Agency, 1997c); *INH*: inhalation rate [m^3 per capita and s], here: 20 m^3 per capita and day (cf. section A.7.2); *0.5*: linear regression coefficient [-]

Since this effect factor is usually given for the steady-state situation, the β_{ED10} slope factor needs to be adapted to allow for an exposure situation which involves an average person over his/her entire life. The effect factor is calculated as (Crettaz et al., 2002; Pennington et al., 2002):

$$EF(p) = \frac{\beta_{ED10}(p)}{BW \cdot t_{lifetime} \cdot 365} \cdot DALY_{personal} \tag{7-15}$$

where

EF : effect factor of substance p [yr lost per mg intake]

β_{ED10} : slope factor for substance p based on effective dose affecting 10 % of a population over background [individual lifetime risk per mg/kg BW/day]

BW : body weight [kg per person]; here: 70

t : human lifetime (or 'exposure duration', cf. Table 7-1) [yr]; here: 70

365 : conversion factor [days per yr]

DALY : Disability Adjusted Life Years per affected person [yr lost per incidence].

In the ExternE methodology, morbidity and mortality impacts are regularly treated separately rather than being combined in a single measure like DALYs. This is basically due to the fact that the different health states are valued differently in monetary terms. When adopting the concept of the β_{ED10} *slope factor* as a linear dose-response function and differentiating the aggregated DALY value into a mortality component (YOLL) and a morbidity component (YLD), Eq. (7-15) serves to assess human health impacts according to the following equations:

$$YOLL_{population}(s,p) = S_{total}(s,p) \cdot t_{emission\ duration} \cdot IF(s,p) \cdot \frac{\beta_{ED10}(p)}{BW \cdot t_{lifetime} \cdot 365} \cdot 1000^2 \cdot YOLL_{personal} \tag{7-16}$$

$$YLD_{population}(s,p) = S_{total}(s,p) \cdot t_{emission\ duration} \cdot IF(s,p) \cdot \frac{\beta_{ED10}(p)}{BW \cdot t_{lifetime} \cdot 365} \cdot 1000^2 \cdot YLD_{personal} \tag{7-17}$$

where

1000^2 : conversion factor [mg per kg]

365 : conversion factor [days per yr]

β_{ED10} : slope factor for substance p based on effective dose affecting 10 % of a population over background [individual lifetime risk of incidence per mg/kg BW/day]; defined in Tables 7-6 and 7-7

BW : body weight [kg per person]; here: 70

IF : effective Intake Fraction of substance p of emission scenario s [$kg_{effective\ exposure}$ per $kg_{released}$]; computed according to Eq. (7-3)

S : source strength of substance p for emission scenario s [kg per yr]

t : $t_{emission\ duration}$: emission duration [yr]

$t_{lifetime}$: human lifetime (or 'exposure duration', cf. Table 7-1) [yr]; here: 70

YLD : $YLD_{population}$: overall Years of Life lived with a Disability for emission scenario s of substance p [yr lost]

$YLD_{personal}$: personal Years of Life lived with a Disability due to a disease related to the slope factor β_{ED10} [yr lost per person and incidence]; defined in Tables 7-6 and 7-7

YOLL : $YOLL_{population}$: overall Years of Life Lost for emission scenario s of substance p [yr lost]

$YOLL_{personal}$: personal Years of Life Lost due to a disease related to the slope factor β_{ED10} [yr lost per person and incidence]; defined in Tables 7-6 and 7-7.

Note that the impact per kilogram of substance released is computed by neglecting the source strength and the emission duration.

7.3.2 Dynamically computing the impact

It has been mentioned above that the Intake Fraction may not only be calculated for steady-state situations but also dynamically. When analysing an emission scenario dynamically, the amount of a substance released into the environment and also the amount taken in by the human population may vary over time. As a result,

Eqs. (7-16) and (7-17) need to be adjusted in order to allow for dynamic analyses of pulse and no-pulse emission scenarios.

The adaptation depends on the time step t_{step} chosen for the analysis and also the investigated integration time. Due to the temporal resolution especially of the employed environmental data which are given as long-term averages (section 2.3.2), only time steps may be investigated that are given in full years. Note that the time step should match the investigated substances' dynamics in terms of exposure, i.e., at least not longer than a substance's residence time in the exposure media. Further note that this may also imply that different time steps be used for ingestion and inhalation-based impact assessments. This will especially be the case for pulse emission scenarios of substances with a residence time in air of one year maximally.

Thus, when analysing the temporal development of (human) exposure towards a continuous or pulse emission of $t_{emission\ duration}$ time until the end of the investigated time horizon (n iterations times t_{step}), Eqs. (7-16) and (7-17) are reformulated according to Eqs. (7-18) and (7-19) using Eq. (7-4) in order to compute the effective Intake Fraction.

$$YOLL_{\text{population},\, i \cdot t_{\text{step}}} = \sum_{i=1}^{n} [S_{\text{total}} \cdot \min\{t_{\text{emission duration}}, i \cdot t_{\text{step}}\} \cdot (IF_{i \cdot t_{\text{step}}} - IF_{(i-1) \cdot t_{\text{step}}})] \cdot \frac{\beta_{\text{ED10}}(p)}{BW \cdot t_{\text{lifetime}} \cdot 365} \cdot 1000^2 \cdot YOLL_{\text{personal}} \tag{7-18}$$

$$YLD_{\text{population},\, i \cdot t_{\text{step}}} = \sum_{i=1}^{n} [S_{\text{total}} \cdot \min\{t_{\text{emission duration}}, i \cdot t_{\text{step}}\} \cdot (IF_{i \cdot t_{\text{step}}} - IF_{(i-1) \cdot t_{\text{step}}})] \cdot \frac{\beta_{\text{ED10}}(p)}{BW \cdot t_{\text{lifetime}} \cdot 365} \cdot 1000^2 \cdot YLD_{\text{personal}} \tag{7-19}$$

7.3.3 Distinction of severity for cancer effects

The magnitude of the personal YOLLs and YLDs depends on the severity of the disease or damage related to the slope factor. For cancers, Crettaz and co-workers (Crettaz, 2000; Crettaz et al., 2002) provide statistics on the values for the YLD and YOLL indicators per specific cancer types. The list does not contain all types of cancer which is why a $DALY_{personal}$ is also given for an average cancer case

by weighting each $DALY_{personal}$ according to the prevalence of the associated cancer. The average cancer $DALY_{personal}$ is 6.7 years per person/incidence (Crettaz et al., 2002). Although the authors state that they "do not apply specific weightings to the importance of one year of life lost based on the age at which death occurs and do not discount future damages compared to the present ones" (p. 942), Keller (2005) recently found out that the personal YOLLs had in fact been provided considering these value judgements. While the personal YLDs are maintained, the personal YOLLs increase by about a factor of two towards the ones provided by Crettaz et al. (2002). The $DALY_{personal}$ value consequently increases to 12.8 years per person/incidence. When exploring the contribution of the YLD to the $DALY_{personal}$ using the data given by Keller (2005) for carcinogens, it is found that the YOLL indicator's contribution to the DALY is larger than 85 % for all types of cancer and equal or more than 95 % for more than two thirds of the cancer types. This shows that the Years of Life lived with a Disability (YLDs) are almost negligible for cancers.[19] The YOLLs and YLDs will, however, not be treated separately in the present study, i.e., the same monetary value will be used (cf. section 8.2). This is because the YLD is a measure that is supposed to be commensurate to a life year lost due to morbidity effects (Murray, 1994).

7.3.4 Distinction of severity for non-cancer effects

In order to distinguish the severity of the non-cancer effects, use will be made of a proposal by an expert panel at the International Life Science Institute (ILSI) to subdivide toxicological impacts into several subcategories (Burke et al., 1996 quoted in Owens, 2001 and Pennington et al., 2002). Three categories have been distinguished taking into account reversibility and life-shortening potentials of the respective impacts (Table 7-5). Other than for inhalation-related effects (Hofstetter, 1998; Hurley and Miller, 2001), quantitative measures such as DALYs are currently not readily available for non-cancer effects. In line with Pennington et al. (2002), the simplified classification in Table 7-5 is modified to be compatible with the DALY approach by assuming as a preliminary basis a $DALY_{personal}$ of

[19] In order to distinguish morbidity from mortality effects for unspecified, average cancers, one may choose that 97.3 % of the $DALY_{personal}$ corresponding to the median value provided by Keller (2005) are attributed to YOLLs leaving 2.7 % to YLDs, i.e., 12.5 and 0.3 years per person/incidence, respectively. The average weight for the unspecified average cancer YLD is stated to be 0.809 (Crettaz et al., 2002). This means that when assuming the generic YLD of 0.3 years per person/incidence the time duration during which the corresponding impaired health status prevails is 0.3 / 0.809 = 0.42 years. However, the value of 0.809 appears rather large when compared to the otherwise explicitly stated disability weights in Crettaz et al. (2002).

12.8 years per person/incidence for category 1. This initial value is based on the average for cancer effects (see above) given that these effects are included in this category. The ILSI panel subjectively scaled the differences between the three categories by factors of 10 (reflected in the weights in Table 7-5). Consequently, the non-cancer effects of category 2 and 3 are attributed $DALY_{personal}$ values of 1.28 and 0.128 years per person/incidence, respectively. Given the rather undefined quality of the non-cancer health endpoints, no distinction into YOLLs and YLDs is made despite the same apportionment as for the general cancer case would be straightforward.

7.3.5 β_{ED10} slope factors and physical impacts used in this study

Tables 7-6 and 7-7 summarize the slope factors either taken from Crettaz (2000) or derived according to the equations reproduced in Tables 7-3 and 7-4 for the selected trace elements as well as the $YOLL_{personal}$ and $YLD_{personal}$ values for cancer and the $DALY_{personal}$ values for non-cancer effects (Keller, 2005) employed in this study. Note that both the slope factors and the health quality measures are given per incidence.[20]

7.3.6 Value choices and DALYs

Furthermore, it needs to be noted that the DALY concept in general builds on some inherent value choices made. According to the Impact Pathway Approach, such value choices should be kept out of the determination of the physical impact to the extent possible and should instead only be applied during the valuation step. When deriving DALYs one of whose purposes it is to inform resource allocation decisions (Nord, 2002), value choices made are (Murray, 1994):

- the way how morbidity effects are converted into YOLL-equivalents,
- the assumed life expectancy which complies to the highest occurring on earth, i.e., that in Japan; the duration of time lost due to a death at each age is determined according to this life expectancy of 82.5 and 80 years for females and males, respectively,
- valuing the time lived at different ages differently according to the societal/social perception which leads to the introduction of an age-weighting function, and
- the employed discount rate of 3 %.

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[20] Incidences should not be confounded with prevalences. These are different measures of a disease's occurrence. The *prevalence* of a condition means the number of people who currently have the condition, whereas *incidence* refers to the annual number of people who have a new case of the condition.

Table 7-5: International Life Sciences Institute classification scheme for human health impact categories (Burke et al., 1996 taken from Owens, 2001 and Pennington et al., 2002)

Criteria	Category 1 Irreversible / Life-shortening effects	Category 2 Probably irreversible / Life-shortening effects	Category 3 Reversible / Non life-shortening effects
Examples	• Cancer • Reproductive effects • Teratogenic effects (birth defects) • Acute fatal or acute severe and irreversible effects (e.g., fatal poisoning) • Mutagenicity	• Immunotoxicity • Neurotoxicity[a] • Nephrotoxicity (kidney damage) • Hepatotoxicity (liver damage) • Pulmonary toxicity (lung damage) • Cardiotoxicity (heart damage)	• Irritation (eye, skin, mucosal; that is transient) • Sensitisation (allergy) • Reversible acute organ or system effects (gastrointestinal inflammation)
Weight	1	0.1	0.01
$DALY_{personal}$	12.8	$12.8 \cdot 0.1 = 1.28$	$12.8 \cdot 0.01 = 0.128$
$YOLL_{personal}$	12.5	1.25	0.125
$YLD_{personal}$	0.3	0.03	0.003

a.Neurotoxicity may also be ranked in category 1.

First, in order to convert the time lived with a disability into years of life lost, principally different approaches can be followed (e.g., Murray, 1994, see also below). Presently, the weighting factors are yielded by employing the so-called *person trade-off* (PTO) method (Murray and Lopez, 1996a cited in Müller-Wenk and Hofstetter, 2003 and Essink-Bot, 1998). Two variants had been used in order to promote explicit deliberation within and among the subjects by framing the same question from two different viewpoints. Essink-Bot (1998) explains it in the following way: "In the first, PTO1, a respondent is asked to decide for how many N (N > 1000 persons) in health state X he would be willing to trade one year of life extension of 1000 healthy individuals for the extension of life by one year for the group in the health state X. In the second variant (PTO2), the respondent

Table 7-6: Cancer effect-related β_{ED10} slope factors and physical impacts for mortality (YOLL) and morbidity (YLD) due to inhalation and ingestion exposure of selected trace elements

Trace element	Exposure route	β_{ED10} [risk of incidence per (mg/kg BW and day)]	$YOLL_{personal}$ [years lost-equivalents per person and incidence]	$YLD_{personal}$[a] [years lost-equivalents per person and incidence]		Remarks
Arsenic, inorganic	ingestion	0.75	6.09	0.045 · 4.2 =	0.19	β_{ED10}: oral slope factor for skin cancer of 1.5 [risk per mg/kg-day] (United States - Environmental Protection Agency, 2005), converted according to Eq. (7-12); impact: melanoma (Keller, 2005)
	inhalation	7.5	15.95	0.146 · 1.8 =	0.26	β_{ED10}: unit risk for lung cancer of 4.3 [risk per mg/m^3] (United States - Environmental Protection Agency, 2005), converted according to Eq. (7-14); impact: lung cancer (Keller, 2005)
Cadmium [b]	inhalation	3.2	15.95	0.146 · 1.8 =	0.26	β_{ED10}: unit risk for lung cancer of 1.8 [risk per mg/m^3] (United States - Environmental Protection Agency, 2005), converted according to Eq. (7-14); impact: lung cancer (Keller, 2005)

Table 7-6: Cancer effect-related β_{ED10} slope factors and physical impacts for mortality (YOLL) and morbidity (YLD) due to inhalation and ingestion exposure of selected trace elements

Trace element	Exposure route	β_{ED10} [risk of incidence per (mg/kg BW and day)]	$YOLL_{personal}$ [years lost-equivalents per person and incidence]	$YLD_{personal}$[a] [years lost-equivalents per person and incidence]		Remarks
Chromium, hexavalent [b]	inhalation	21	15.95	$0.146 \cdot 1.8 =$	0.26	β_{ED10}: unit risk for lung cancer of 12 [risk per mg/m^3] (United States - Environmental Protection Agency, 2005), converted according to Eq. (7-14); impact: lung cancer (Keller, 2005)
Lead [c]	ingestion	0.039	12.5 [d]		0.3 [d]	β_{ED10}: oral median tumor dose of 46.6 [mg/kg Body Weight/day] for kidney cancer derived by administration of lead acetate to rats (Gold and Zeiger, 1997 cited in Crettaz, 2000, p. 61), converted according to Eqs. (7-5) and (7-10); impact: average cancer (Keller, 2005)

a.The YLD is yielded by multiplying a disability weight by the duration of the respective disability.
b.No cancer effect information via ingestion available.
c.No cancer effect information via inhalation available.
d.See footnote 19 for the derivation.

Table 7-7: Non-cancer effect-related β_{ED10} slope factors and aggregated physical impacts for mortality and morbidity in terms of DALYs due to inhalation and ingestion exposure of selected trace elements

Trace element	Exposure route	β_{ED10} [risk of incidence per (mg/kg BW and day)]	$DALY_{personal}$[a] [years lost-equivalents per person and incidence]		Remarks
Arsenic, inorganic	ingestion	78	12.8 · 0.1 =	1.28	β_{ED10}: oral chronic NOAEL of 0.0008 [mg/kg BW/day] for a human population (United States - Environmental Protection Agency, 2005), converted according to Eqs. (7-9) and (7-10), extrapolation coefficients set to 1; impact: skin lesions (category 2 effect)
Cadmium	ingestion	41.5	12.8 · 0.1 =	1.28	β_{ED10}: oral chronic BMD_{10} of 0.0013 [mg/kg BW/day] for a human population (Crump, 1998 cited in Crettaz, 2000, p. 96), converted according to Eqs. (7-5) and (7-6); impact: kidney damage (category 2 effect)
Chromium, hexavalent	ingestion	0.15	12.8 · 0.1 =	1.28	β_{ED10}: oral chronic NOAEL of 2.5 [mg/kg Body Weight/ day] derived by administration of dipotassium chromate to rats (United States - Environmental Protection Agency, 2005), converted according to Eqs. (7-9) and (7-10); impact: reduction in water consumption by rats (category 2 effect)

Table 7-7: Non-cancer effect-related β_{ED10} slope factors and aggregated physical impacts for mortality and morbidity in terms of DALYs due to inhalation and ingestion exposure of selected trace elements

Trace element	Exposure route	β_{ED10} [risk of incidence per (mg/kg BW and day)]	$DALY_{personal}$[a] [years lost-equivalents per person and incidence]	Remarks
Chromium, hexavalent (continued)	inhalation	39.0	$12.8 \cdot 0.01 = 0.128$	β_{ED10}: subchronic BMC_{10} of 0.016 [mg/m³] (United States - Environmental Protection Agency, 2005), converted according to Eqs. (7-5) and (7-7) and employing a subchronic to chronic extrapolation factor of 3.3 (cf. Pennington et al., 2002); impact: enzyme (lactate dehydrogenase) affected in rats (category 3 effect)
Lead	ingestion	143	$12.8 \cdot 0.1 = 1.28$	β_{ED10}: most sensitive oral chronic LOAEL of 0.014 [mg/kg Body Weight/day] derived by administration of lead acetate to rats (Agency for Toxic Substances and Disease Registry, 1999), converted according to Eqs. (7-5) and (7-8), as United States - Environmental Protection Agency (2005) do not provide any non-cancer effect measures in spite of evidence that lead causes hypertension, the slope factor needs to be used with caution; impact: high blood pressure in rats (category 2 effect)

a. The calculations demonstrate the derivation of the final DALY value from the generic DALY value weighted by the category weight as given in Table 7-5.

is asked to estimate for how many individuals in health state X he would be prepared to surrender one year of extended life for 1000 individuals in perfect health in exchange for the complete recovery followed by one year of perfect health for the group in the given health state." (point 18). This way of determining weights does not allow for subjective valuation by (potentially) affected people like in contingent valuation studies and might, therefore, affect the stated weight. However, Hofstetter and Hammitt (2001) conclude that the difference between individual and altruistic preferences is small.

Assuming the highest life expectancy (at birth) observed on earth to be applicable to Europe's population is deemed not to introduce an unacceptable bias. Many of the countries included in the assessment can be considered as highly developed with an on average high standard of living, i.e., about or higher than 75 and 80 years for males and females, respectively (Lopez et al., 2001a; Lopez et al., 2001b). However, it is unclear how diseases or premature deaths are taken into account by the DALY concept for those people that have survived this period life expectancy at birth. The general assumption of the highest life expectancy at birth might compensate for the assumed disregard of the health effects for these age groups.

According to the age-weighting employed, the DALY concept assigns values below to life years lived before the age of 9 and after the age of 55; the ages in between receive weights larger than unity (Murray, 1994). The rationale behind this is that individuals within a society assume different roles and have changing levels of dependency with age, thus, having different social values. This, however, is in contrast to the methodological individualism which constitutes one of the bases for the theory of welfare economics (Rennings, 1994) which provides the context for the external cost assessment.

A discount rate of 3 % is selected in order to avoid "the difficulty of the time paradox and of overvaluing eradication programmes when no discount rate is used" (Murray, 1994, p. 440). By 'time paradox' it is meant in the DALY context that one would postpone investments into health projects to the future if health benefits would be discounted at a smaller rate than the monetary costs. In contrast, if it was possible to launch a project now that will eradicate a disease for good and zero-discounting was assumed one might conclude to spend a fortune to achieve this goal as this would pay-off due to efficiently avoided DALYs caused by the respective disease during the future existence of humankind. As a result, "(o)nly when costs and benefits are discounted at the same rate do we become indifferent to the time when a project is implemented" (ibid., p. 440).

It needs to be emphasized that two of these four value choices have been addressed by Crettaz and co-workers (Crettaz, 2000; Crettaz et al., 2002) and Keller (2005), namely age-weighting and discounting. These value choices are not

taken into consideration in the DALYs used and published by these authors. The DALYs given in Tables 7-6 and 7-7 can, therefore, be used as traditionally done with the YOLL values within the ExternE project series when it comes to monetary valuation (cf. section 8.2).

7.3.7 Discussion on the magnitude of the assessed DALYs

For consistency and comparability reasons between the assessed trace elements, the values provided by Keller (2005) are adopted. Only one $YOLL_{personal}$ value could be found in publications of the ExternE project series. It is given for lung cancers and amounts to 16 (Table 12.8 on p. 252 in European Commission, 1999a) which compares well with the 15.95 $YOLL_{personal}$ as suggested by Keller (2005).

The disability weights given to the different years lived with a particular cancer as reproduced by Crettaz et al. (2002) appear rather small. These have been maintained by Keller (2005). At present, it is unclear whether these disability weights also take potential depressions, pain and/or suffering appropriately into account which constitute the lost utility component related to an illness (European Commission, 1999a, see section 8.2). In principle, they should do so since "scenarios to be valued were presented consistently in the form of a disease label, a brief clinical description of the disease stage, and a generic health state profile" in the case of the European Disability Weight project (Schwarzinger et al., 2003) which has been carried out similarly as in the Global Burden of Disease study. The assignment of disability weights in the Global Burden of Disease study was according to six disability classes ranging from perfect health to death. Each class represents a greater loss of welfare or increased severity than the class before (cf. Table 7-8). As regards comparability of diseases assigned to the same class, Murray (1994) states that these "may restrict different abilities or functional capacities but the impact on the individual is considered to be similar" (p. 438). This all allows the conclusion that the overall welfare of an individual that comprises physical as well as mental aspects should have been addressed by the respondents when assigning weights to different diseases. However, there are doubts on the generalisability of the disability weights computed from the person trade-off method used in the Global Burden of Disease study when compared to those according to the European Disability Weights project using a similar method (Schwarzinger et al., 2003). The results of the visual analogue and the time trade-off method which were used additionally to the person trade-off method for the derivation of disability weights employed in the latter study, furthermore, deviate rather substantially from those reproduced by Crettaz et al. (2002). For instance, the disability weight for 'Breast cancer (disease-free stage without sequelae)',

Table 7-8: Definitions of disability weighting in the Global Burden of Disease Study according to Murray (1994)

Class	Description[a]	Weight
1	Limited ability to perform at least one activity in one of the following areas: recreation, education, procreation or occupation	0.096
2	Limited ability to perform most activities in one of the following areas: recreation, education, procreation or occupation	0.220
3	Limited ability to perform in two or more of the following areas: recreation, education, procreation or occupation	0.400
4	Limited ability to perform most activities in all of the following areas: recreation, education, procreation or occupation	0.600
5	Needs assistance with instrumental activities of daily living such as meal preparation, shopping or housework	0.810
6	Needs assistance with activities of daily living such as eating, personal hygiene or toilet use	0.920

a.Limited ability has been arbitrarily defined as a 50 % or more decrease in ability.

i.e., the stage after successful treatment of breast cancer, may be as large as 0.4 (Schwarzinger et al., 2003) whereas the disability weight for breast cancer-related morbidity amounts only to 0.069 (Crettaz et al., 2002) although also comprising more severe health state stages. These disease stages are found to be most influential on the magnitude of the disability weights at least in the case of the visual analogue scale method (Essink-Bot, 1998). Thus, the disability weights as given in Tables 7-6 and 7-7 are considered to underestimate the weight of years lived with a disability to some extent. As a change of single disability weights may have an impact on the DALYs associated with an average cancer, no attempt will be made here to change the disability weights. This may need to be addressed in the future.

7.3.8 Temporal delays

There are two main time delays between the emission of a substance into the environment and its effect on human health (cf. Fig. 2-2). First, the environmental fate of the substance from the source to the medium to which a person is exposed may vary substantially depending on the medium (e.g., air vs. food) and on the persistence of the substance. This may well be in the order of millennia for per-

sistent substances such as metals (Hellweg, 2000; van den Bergh et al., 2000; Huijbregts et al., 2001). Second, the time gap between exposure and the health effect, i.e., latency time, leads to another postponement of the effect to occur (Mücke 6/1995; Mersch-Sundermann, 1996; United States - Environmental Protection Agency, 1998; Hurley and Miller, 2001). In case of premature death in the long run (so-called chronic mortality), one may distinguish between a period with health impairments (morbidity, e.g., expressed in Years of Life lived with Disabilities) and years of not realized life expectancy (e.g., Years Of Life Lost, European Commission, 1999a; Hurley and Miller, 2001) in addition to these (apparent) latency times. One has to note, however, that the YLD indicator as such does not tell over which time period the health impairment occurs which may in principle be relevant when valuing the impact with a non-zero discount rate. These time spans for apparent morbidity and the respective weights are also provided in Table 7-6.

In general, there is hardly any information about time delays between exposure and impact (i.e., latency times) available with respect to the trace elements investigated (Searl, 2004). This may have an effect especially in the valuation of the impacts (discounting, cf. section 8.1 and Hammitt, 2000). When performing non-zero discounting, the distribution of when the assessed DALYs occur within a given population is rather important. By default, no (minimum) latency time is, therefore, assumed noting that delays between exposure and effect may occur due to different susceptibilities of the individuals in the population when distributing the DALYs over time (see section 8.2).