

# Risk Assessment of Bioaccumulative Substances Part I: A Literature Review

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Holistic Integrated Tiered Risk Assessment

# HITRA

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## Summary

This report describes a review of literature that has been used as input for a proposal for risk assessment of bioaccumulative substances, either from produced water discharges or present as background contamination. It is presented such that each chapter deals with the four major aspects in the environmental risk assessment of bioaccumulative substances: Exposure (which substances are relevant to consider and what are their concentrations in the environment?), Bioaccumulation (which internal concentrations are the result of the exposure to external concentrations and through indirect exposure via the food?), Effects (which internal concentrations will adversely affect species in the ecosystem?), Environmental Risk (how can the potential effects be translated into a environmental risk indicator?).

In the final chapter an outline of a proposed modelling concept is presented. This concept is presented in more detail a separate report [1], which also includes a research proposal for the implementation of the modelling concept.

# 1 Introduction

## 1.1 Background

The interest in exploring oil and gas reserves in the Arctic region is increasing. However, operating in these areas asks for special requirements. Operating in icy conditions and coldness includes technical challenges and special HSE (Health Safety and Environment) issues. Arctic environments are considered to be sensitive to physical-chemical stress. Before these areas can be entered, environmental risks of oil and gas exploration and production activities need to be assessed. Tools for environmental assessment presently available derive risk from ambient environmental concentrations (water, sediment). The application of these existing assessment tools in the arctic is not straightforward. For as yet it is not known how sensitive arctic species are compared to temperate species. Furthermore, the persistence in the environment of some pollutants allows them to accumulate in animals, and transport through the food-web, becoming more concentrated in top predators. There are numerous accumulating substances that are not related to the petro-industry that are already present in Arctic species. Those background contaminants already pose a risk to Arctic species. The risk of bioaccumulating pollutants does not only directly descend from concentrations in water and sediment, exposure through the food-web is also relevant. Persistent bioaccumulative substances are not included in most current risk assessment tools and will therefore need to be addressed in a separate tool. StatoilHydro recognised these issues and initiated a programme, called “Tuning existing environmental risk assessment tools to the arctic environment”, in order to properly evaluate the potential for impacts of StatoilHydro’s future activities.

Further, StatoilHydro has already initiated a project focussing on the risk assessment of cold water corals (CORAMM). Within that project, the Dutch institute of Ecology Research (NIOO) is developing a bioaccumulation model including principles from the OMEGA model (Nijmegen University) to model bioaccumulation in the (arctic) food-web. This model could potentially be used within the proposed risk assessment tool of bioaccumulating substances in the Arctic environment. However, it needs to be determined whether this model is actually a good basis for environmental risk assessment. Therefore, a feasibility/concept development study was initiated with the aim to set up a modelling concept for (probabilistic) environmental risk assessment of bioaccumulating substances and to identify studies required to further develop this concept. This study consists of the following main tasks:

1. Description of the aim and requirements of the intended risk model
2. Identification and analyses of available methods from literature
3. Refining the conceptual model
4. Gap analysis and development of research proposal

This report describes the results of tasks 2 and 3. The aim and requirements of the intended model (task 1) are briefly described the section below. A research proposal (task 4) is reported separately [1].

## 1.2 Aim and Scope

As mentioned previously, the risk assessment should be designed such that it is compatible with current risk assessment techniques of non-bioaccumulating toxicants, so that it can be used in StatoilHydro’s integrated toolbox, e.g. the EIF (Environmental Impact Factor<sup>1</sup>). The preliminary study should result in a research proposal for the actual development of the model and bridging eventual gaps in knowledge.

This report describes available and potential methods for risk assessment of bioaccumulating pollutants. In general, risk assessment comprises of exposure- and effect assessment. These methods are evaluated for their application potential in risk assessment of bioaccumulating pollutants in the Arctic environment within the EIF concept. Based on these findings, a conceptual risk assessment method is derived, together with an overview of knowledge gaps.

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<sup>1</sup> The EIF produced water is an indicator based on the volume of water around a discharge point were adverse effects to biota might occur as a result of the discharge

### 1.3 Approach

For the concept development the generic approach to risk assessment is used, which states that risk is characterised by both the exposure and effect levels. For bioaccumulating substances that magnify in the food-chain the risk characterisation needs to be done based on internal concentrations in species (Figure 1).

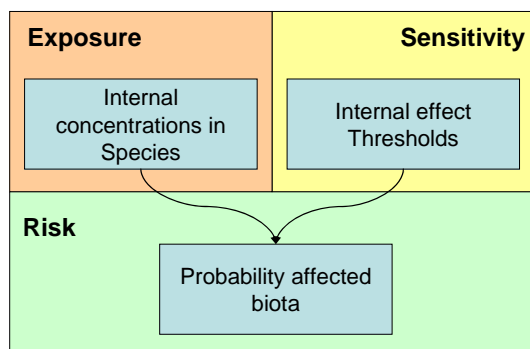


Figure 1 *Risk characterisation of bioaccumulating substances needs to be done on the basis of internal concentrations*

When the generic approach to risk assessment is further specified for bioaccumulating substances four phases can be distinguished (Figure 2):

- Exposure
- Bioaccumulation
- Effect
- Risk

In this report, each phase will be discussed in separate chapters. In each chapter the intended model requirements for the specific phase are further crystallised. Based on these requirements and the review of literature gaps in knowledge and data are identified. Specific recommendations will be made in order to obtain the missing data. These recommendation will then be used in the research proposal (separately from this report [1]).

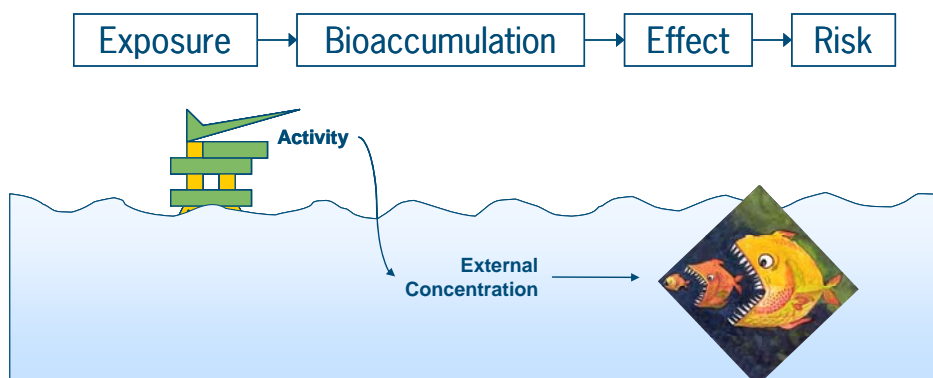


Figure 2 *Activities may lead to external concentrations (exposure), which can then accumulate in the food-chain (bioaccumulation), which in turn can lead to effects, which finally can be expressed as a certain risk.*

## 2 Exposure

### 2.1 Introduction

Effects of bioaccumulating substances can best be assessed based on their internal concentrations in species. However, if the risk of these substances is to be related to certain activities (e.g. discharging produced water), we need to model the external concentrations (that is, in the water compartment) of the contaminants and then determine the accumulation in the food chain (Figure 2). The contaminants that emanate from petro-industry operation activities contribute to the risk of substances that have already accumulated in the Arctic environment from other sources. The intended modelling tool intends to quantify the contribution of operation activities to the already present risk.

### 2.2 Background Contaminants

The intended model needs to be able to distinguish between background contaminants (bioaccumulating substances that are already present in the Arctic) and additional levels of petro-industry related substances. A link is required between the concentrations inside an organism and the activities that need to be assessed, in the outside world. As many bioaccumulating substances do not reach detectable concentrations in water but can be measured in species, the concentrations of these substances in water should be derived from the internal concentrations, using the bioaccumulation module of the intended model. This, however, is only necessary for substances that emanate from operation activities and are also considered background contaminants. In that case, external concentrations from both sources (petro-industry and background) need to be added to determine the resulting internal concentrations. Background contaminants that are not related to petro-industry activities can directly be assessed on internal concentrations measured in biota.

The relative contribution of petro-industry related activities to the environmental risk can be determined by first calculating the risk for the background contaminants in absence of the substances released by those activities. This should then be compared to the risk, calculated for the situation with the petro-industry related substances in addition to the background contaminants.

### 2.3 Substances to Consider

There are many substances that should be considered when calculating the background risk. Today, the main Persistent Organic Pollutants (POPs) of concern within the Arctic ecosystems are still chlorinated pesticides (e.g. p,p'-o,p'-DDT, hexachlorocyclohexanes (HCH), toxaphene (CTT) and cyclodiene compounds including chlordanes, industrial chemicals and by-products (e.g. PCB, hexachlorobenzene (HCB)), combustion products (e.g. chlorinated dibenzodioxines/dibenzofuranes (PCDD/F) and polycyclic aromatic hydrocarbons (PAHs)). Lately increased focus has been directed towards metabolites of the parent POPs as well as "new" environmental toxins such as perfluorinated alkylated substances (PFAS), chlorinated naphthalenes (PCN), polychlorinated paraffins (CPs), polybrominated diphenylethers (PBDE), polybrominated biphenyls (PBB), Polyfluorinated dibenzodioxins and furans (PFDD/F), synthetic musk, phosphorous containing flame retardants, etc. Many of these substances have been found to biomagnify [2-5]. The list of background contaminants can be extended by screening substances on chemical-physical properties [6]. A choice will need to be made on the relevance and the data-availability of the substances that will be included in the intended model.

There are several operational petro-industry activities (and calamities such as spills) that introduce contaminants into the environment. In this report we will focus on substances in produced water which have been identified in previous studies. Other substances may be related to other activities (e.g. drilling). The produced water chemicals serve as an example in this report and although the other substances are not presented here, the same principles will apply. Substances, or groups of substances that are currently implemented in the produced water EIF are listed in Table 1 [7].



Data is required for each substance (group) that is going to be included in the intended model. For instance, effect data is needed (Chapter 4), in addition to compound related model parameters for bioaccumulation modelling (Chapter 3). Availability of required data is important for the success of the intended model. Many studies have been performed on the bioaccumulation of PAHs [8, 9], aromatic hydrocarbons [10] and (other) organic contaminants [11-13], resulting in available internal effect concentrations for these substances. Kinetic parameters (required for bioaccumulation modelling) are available to some extent [12, 14, 15]. Data is also available for metals [16-18]. In addition to the literature search, the ERED (Environmental Residue-Effects Database) has been accessed to check the availability of internal effect concentrations for produced water substances (Table 2). Data is available for most of the representative substances. However, these data are not standardised, i.e. concentrations are measured in different tissues with different corresponding effects (sizes) under various conditions. Specific consideration should thus be taken when using these concentrations for the derivation of effect endpoints within the risk module. Data for bioaccumulative substances that are known to be present in the Arctic, but not related to the oil and gas industry, are readily available. For example, the ERED contains 956 results for PCBs based on 122 studies and 87 different species. Polybrominated diphenyl ethers result in 34 hits in the ERED from 5 studies of 5 species. "New" POPs are less available in the database, PFOS for instance could not be found with the initial screening of the database.

Table 1 *Composition of groups of substances in produced water as defined for the produced water EIF*

No.	Main group	Substances	Representative
1	BTEX	Benzene, toluene, ethylbenzene, xylene	Benzene
2	Naphthalenes	Naphtalene + C1-C3 Alkylhomologues	Naphthalene
3	PAH 2-3 ring	Substances on the EPA 16 PAH list with 2-3 rings	Phenanthrene
4	PAH 4-ring+	Substances on the EPA 16 PAH list with 4 ring or more	Benz(a)pyrene
5	Alkylphenols C0-C3	Phenol + C1-C3 alkylphenols, incl. Alkyl-homologues	Phenol
6	Alkylphenols C4-C5	C4-C5 alkylphenols, incl. Alkyl homologues	Pentylphenol
7	Alkylphenols C6+	C6-phenol and higher, incl. Alkyl homologues	Nonylphenol
8	Aliphatic hydrocarbons	Various	-
9	Metals	Zn, Cu, Ni, Pb, Cd and Hg	Field specific
10	Organic acids	Total organic acids (<C6)	Field specific
11-1n	Exploration and production chemicals	mixture	-

Table 2 *Internal effect data availability of produced water substances in ERED (Source: ERED Website <http://el.erd.c.usace.army.mil/ered/index.cfm>, accessed on September 18, 2008)*

Group No.	Representative substance	Number of results	Studies	Species	Effects	Endpoints
1	Benzene	6	5	3 (2 fish, 1 algae)	4	3
2	Naphthalene	20	10	9 (fish, crustacean, bird, bivalve)	7	9
3	Phenanthrene	63	13	8 (fish, crustacean, polychaete, bivalve)	9	20
4	Benz(a)pyrene	189	27	18 (fish, crustacean, bivalves)	9	62
5	Phenol	41	8	3 (fish, echinodermata)	5	19
6	Pentylphenol	n.a.				
7	Nonylphenol	41	4	4 (fish, crustacean)	4	4
8	Octane	2	2	2 (fish, bivalve)	2	2
9	Cu / Mg	1167 / 393	107 / 59	81 / 55	11 / 11	188 / 44
10	Organic acids	n.a.				
11-1n	Exploration and production chemicals	n.a.				

n.a. = not available (i.e. not present in database or no representative substance available)

### Exploration/production chemicals

The exploration and production chemicals will need to be further specified, if they are to be included in the intended model. However, these substances need to comply to the Harmonised Offshore Chemical Notifications Format (HOCNF), which means that these substances have been screened for PBT (Persistence, Bioaccumulation and (eco)Toxicological) properties. As potentially persistent and bioaccumulating substances are generally not allowed, it would suffice to deal only with direct exposure from the water compartment.

### Metals

(Heavy) metals are also known to bioaccumulate in the Arctic environment [19]. As heavy metals are found in the Arctic and are also part of produced water, they are relevant in risk assessment. The general consensus for metals is that they usually don't biomagnify, but can bioconcentrate [16, 20], as most metals are regulated and excreted. The nuance between bioconcentration and biomagnification will be discussed in chapter 3. However, some organometals (e.g., methyl mercury) can biomagnify.

Bioavailability is an important aspect in the assessment of accumulating substances (see also section 3.1) but a complex issue for metals in the marine environment. So called Biotic Ligand Models (BLMs) that deal with the bioavailability of metals are plentiful for the freshwater environment. BLMs for the marine environment, however, have only emerged recently. For copper a marine BLM has been developed to predict toxicity in *Mytilus sp.* for specific marine water types [21]. Each species and metal requires a different BLM which are currently not available for all metals and species of interest. By assuming 100% bioavailability for metals, we can ensure that the risk assessment is conservative.

### PAHs

Bioavailability of organic substances should also be studied. Black carbon content is for instance an important parameter for PAH-binding, thereby reducing its bioavailability. Furthermore, benthos can be exposed to elevated levels when particulate matter, to which PAHs (or other bioaccumulating substances) are bound, sediment. PAHs, especially the more soluble low molecular weight compounds, can bioconcentrate in fish from sediments. However, PAHs are less prone to bioaccumulation or biomagnification than for instance the organochlorines, partly because of metabolic degradation of PAHs in top predators and their prey [22, 23].

## 2.4 Conclusions and Recommendations

From the text above the following is concluded:

- Due to, a.o. metabolism, not all petro-industry related substances accumulate to the top of the food chain;
- Bioavailability is an important aspect to consider when modelling bioaccumulation
- The list of known background contaminants is extensive

Based on the conclusions and the text above, we suggest the following:

- Screen all petro-industry related substances for accumulating potential along the food-chain (using techniques described in Chapter 3). We anticipate that most of those substances do not accumulate in trophic levels higher than fish. This has consequences for the level of complexity of the bioaccumulation model (see Chapter 3)
- Modelling of total water concentrations can be done with existing models, without major adjustments. The accumulation and risk module will be designed such that there is no feedback with the external concentration module. The particle tracking model developed by Sintef for current produced water ELF calculations can be used for this purpose, although alternatives are available. However, they need to be checked whether they are suitable for modelling bioaccumulating substances. An important check would be whether the model has implemented binding of the substances to particulate matter. This is important for the bioavailability of the substance and elevated exposure of the benthos due to sedimentation of the particulate matter.
- Calculated water concentrations will serve as input for the bioaccumulation module of the model. In this module species will be exposed to the external concentrations, but also through their food (food chain). Bioaccumulating substances will enter the food chain through the primary producers and in some case primary consumers (e.g., filter feeders). For this purpose a partitioning constant, describing the partitioning of the substance between water and primary producers (algae), needs to be determined from literature or by calibration of the intended model
- Internal concentrations of the background contaminants in Arctic species need to be collected from literature. For species for which internal concentrations are not available, the internal concentration needs to be modelled with a bioaccumulation model (Chapter 3). These values are not only necessary to determine effect levels, but also the potential relevance of background contaminants may be reflected by internal concentrations or data availability. The information can therefore be used to narrow down the extensive list of background contaminants.

## 3 Bioaccumulation

### 3.1 Introduction

The term bioaccumulation can be used in a general sense to describe situations where organisms acquire higher concentrations of certain contaminants in their body than are present in their food and/or the ambient medium in which they live. Bioaccumulation is the result of both bioconcentration and biomagnification, where bioconcentration is the internal concentration as a result of waterborne exposures, while the latter is the result of exposure to the contaminant through the food. Each of the two processes (bioconcentration and biomagnification) will be discussed in more detail in the sections 3.2 and 3.3 respectively.

Organisms can take up contaminants from water or from contaminated food and can eliminate contaminants by excretion or metabolism. If the uptake is higher than the elimination, elevated levels of contaminants occur within the organism. Uptake and elimination processes vary between species and substances. Most apolar organic substances (e.g. naphthalene and other PAHs (Polycyclic Aromatic Hydrocarbons)) are hydrophobic (poorly soluble in water) and (therefore) lipophilic. While the concentrations of these contaminants dissolved in water are very low, the concentrations in aquatic organisms can be very high. This is because accumulation of these compounds in the lipid rich tissues of organisms takes place as a result of equilibrium partitioning between water

and lipids. Contaminants can not only accumulate in biota but also tend to adsorb to (sediment) organic carbon or other environmental matrices (Figure 3).

### **total or nominal concentration**

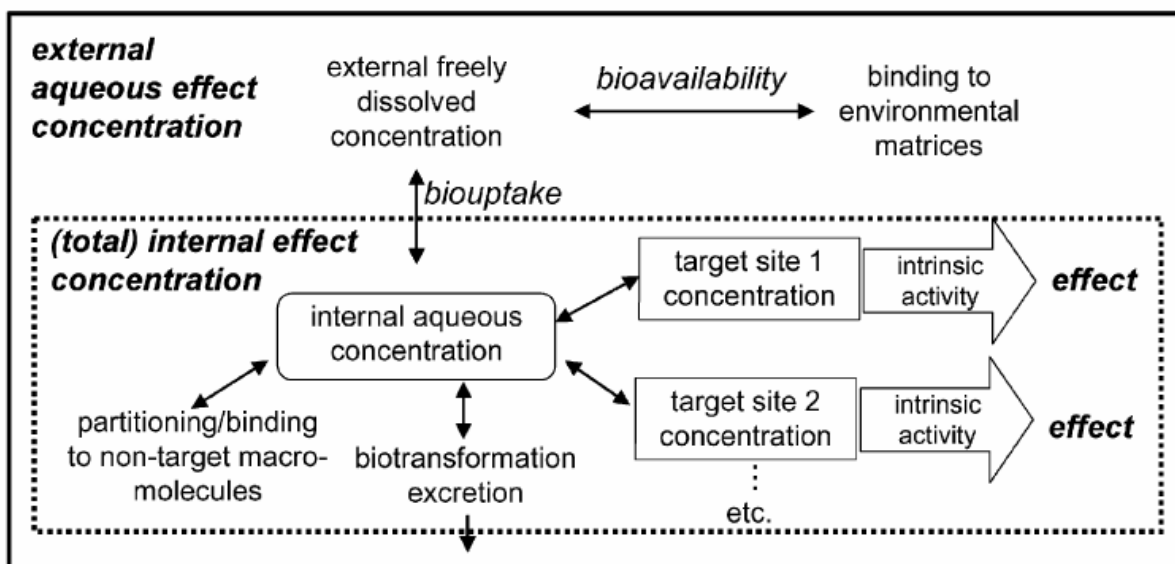


Figure 3 Relationship between total, external, and internal effect concentrations and distribution to different target sites [24].

Considering the fate and pathways of bioaccumulative substances, it is important how to express the concentration of these substances (e.g. based on total-, dissolved or target site concentrations; based on dry weight, wet weight, or lipids). For example, when expressing the concentration of a bioaccumulative substance in fish as ppb in dry weight of the whole organism, the concentration will be higher than expressed in ppb wet weight of the whole organism but lower than ppb in lipids. Concentrations in whole fish will also be lower than concentrations in the liver, for example. The study of Smítková et al. [25] shows the importance of the lipid content of fish for accumulation of chemicals in fish. In fat fish (20% lipid content), concentration factors are higher than in fish with 5% lipid content. The authors conclude that incorporation of lipid content improves exposure assessment for human and ecological risks substantially. No relationship was observed between trophic position and lipid content of organisms [26].

Bioaccumulation of a substance into an organism is not an 'effect' or hazard in itself. Bioconcentration and biomagnification together describe bioaccumulation, which will result in a body burden which may or may not lead to toxic effects. Therefore, when appropriate, the potential of a substance to bioaccumulate in the aquatic environment should be included as an exposure related parameter in the risk assessment [3]. This chapter describes approaches to assess the bioaccumulating properties substances.

## **3.2 Bioconcentration**

Bioconcentration is a process that results in an organism having a higher concentration of a substance than is in its surrounding environmental media as a result from direct exposure to that media. Fish for instance can take up substances from the water through the gills. Some of those substances can get 'stuck' in fatty tissues and accumulate there. In a situation where the concentration in the water is in equilibrium with the internal concentration in the biota, the static BioConcentration Factor (BCF) can be determined, which is the ratio between the two (Figure 4). The BCF can be determined experimentally to quantify the bioaccumulating potential of a substance. It can also be used to calculate the internal concentration for any external concentration. When comparing concentrations in different mediums it is important to consider the unit of expression, i.e. whether to use fresh weight, dry weight or lipids as a basis. When uptake and depuration kinetics are measured, the dynamic BCF can be calculated from the quotient of the uptake and depuration rate constants [27].

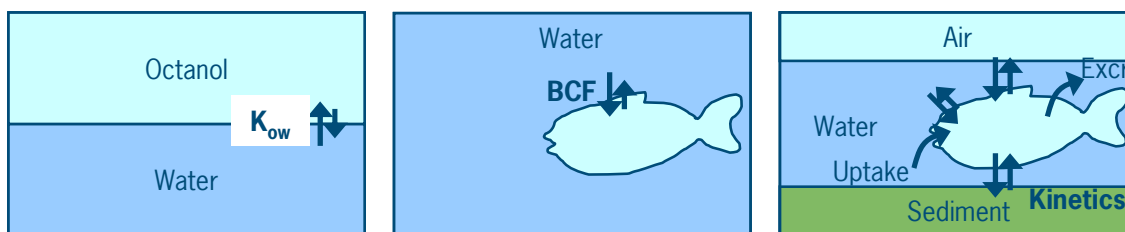


Figure 4 *Different increasingly complex models for determining internal concentrations as a result of bioconcentration. Left: partitioning between octanol and water; middle: partitioning between water and biota; right: describing the assimilation and elimination routes with rate constants (kinetic model). When the kinetic model uses feeding as an exposure route, the process described is no longer just bioconcentration, but includes biomagnification as well.*

If measured BCF values are not available, the BCF for fish can be predicted from the relationship between  $K_{ow}$  and BCF. The relation between the  $K_{ow}$  and the BCF is usually derived on a lipid weight basis [28], but can also be based on the total (wet or dry) weight. In the most simplistic case, the lipids in aquatic organisms are resembled by octanol and therefore it is assumed that  $BCF = K_{ow}$ . There are a number of factors that are not taken into consideration when BCF is estimated only on the basis of  $\log K_{ow}$  values. These are [27]:

- phenomena of active transport;
- metabolism in organisms and the accumulation potential of any metabolites;
- affinity due to specific interactions with tissue components;
- special structural properties (e.g. amphiphilic substances or dissociating substances that may lead to multiple equilibrium processes);
- uptake and depuration kinetics (leading for instance to a remaining concentration plateau in the organism after depuration).

Quantitative structure activity relationships (QSARs) are commonly used in bioaccumulation assessment. This relationship may provide good predictions of fish BCF for lipophilic, nonionic substances undergoing minimal metabolism or biotransformation within the organism. Bioaccumulation and food transfer models based on this relationship will overestimate the potential of substances to bioconcentrate and to transfer up the food-web [3]. Thus, the incorporation of processes such as volume exclusion, metabolism, and others into these models should result in more realistic results.

The distribution of a chemical in the aquatic environment is not only between the water phase and the organism. Chemicals also tend to adsorb to environmental matrices (e.g. sediment, particulate matter). The limited availability of substances due to strong sorption to carbonaceous materials (such as black carbon, kerogen and coal in sediment), has been identified as an important factor for the predictive power of bioaccumulation models [29].

When the ratio between water and internal concentration includes exposure through the food, the ratio is called the BioAccumulation factor (BAF). In the BioMagnification Factor (BMF), the ratio is based on the concentration in predator and prey as will be discussed in section 3.5.

### 3.2.1 Kinetic Models

An advantage of kinetic (or mechanistic) models is that they quantify different processes of varying importance of bioaccumulation, such as respiration and feeding rates, growth dilution and biotransformation. As a result, mechanistic models have higher demand of parameterisation. Required parameters for mechanistic models are, to some extent, available from literature or can be calculated. For example, a model was developed to determine rate constants and equilibrium ratios for accumulation of organic substances [12] and inorganic substances [30]. Some kinetic model parameters are: chemical uptake from diet; food ingestion rate; gut absorption efficiency; chemical loss by metabolism; volume of organism, growth dilution; gill ventilation; and lipid fraction of organism and diet. The increased need for environmental information and extrapolation between systems and species might lead to increased uncertainty in terms of model predictability [31].

The key factor controlling bioaccumulation in marine mammals is metabolism, and not equilibrium partitioning [32]. Therefore, the equilibrium approach can only provide a good estimate for organisms that are not able to metabolise the substance of concern. Most kinetic models have implemented metabolism, but only as a form of elimination, i.e. chemical loss by metabolism. Degradation products are usually not included in the assessment.

Kinetic models can include exposure through food and can thereby also describe biomagnification. The importance of metabolism in biomagnification can be illustrated by a comparison of two classes of lipophilic xenobiotics, polycyclic aromatic hydrocarbons (PAH) and polychlorinated biphenyls (PCB). PAHs are metabolised by most taxa of marine organisms, e.g. polychaetes, fish, birds and mammals [28]. It has been observed that PAH were metabolised along the food-web, unlike PCB which exhibited higher bioaccumulation in fish. Although invertebrates accumulated PAH, the levels of these contaminants were generally low in fish that were fed contaminated invertebrates. It has been demonstrated that PCB residues increased from roach → eel → pike obtained from Dutch lakes, but levels of PAH were similar in all three species [3]. Schenker et al. [33] shows that the hazard of some substances is underestimated if the degradation products of these substances are not included in the assessment. Metabolic transformation has been identified as an issue that may limit the predictive power of bioaccumulation models when they are not included [29].

Landrum et al. [34] compared equilibrium and kinetic models (see Table 3). They found that the bioenergetic-based (BE) toxicokinetic model may be the best choice when the importance of the various routes should be determined [34]. An organism's contact with the external environment is directly related to the flux of water across its gills to obtain oxygen and the flux of food/sediment through its gut to obtain nutrients. BE toxicokinetic models predict pollutant uptake as a function of these fluxes, assuming that uptake from each source is proportional to its flux [34]. The BE models integrate most of the key physiological process in the energetic terms and can directly incorporate season effects on most of the parameters. The physiological-based pharmacokinetic (PBPK) models were originally developed to describe drug metabolism kinetics in mammals. PBPK models separate an organism into anatomical compartments, each representing a particular organ or group of kinetically related tissues. PBPK models require relatively more data and resources for development and it may be necessary to modify the model structure for invertebrates.

Table 3 *Comparison of equilibrium and kinetic models [34]*

Model/attribute	Model					
	Equilibrium	RC <sup>a</sup>	Fugacity	CV <sup>b</sup>	PBPK <sup>c</sup>	BE <sup>d</sup>
Requires assumption of equilibrium	Yes	No	No	No	No	No
Models multiple compartments	No	Yes	Yes	Yes	Yes	Yes
Models multiple uptake routes	No	Yes	Yes	No	Yes	Yes
Can be used to model internal distribution of toxicants	No	Yes	Yes	Yes	Yes	No
Potential to scale to other species	Yes (by lipid content)	Some	Some	No	Yes	Yes
Data requirements	Low	Moderate	Moderate-High	Moderate	High	High

<sup>a</sup>RC = rate coefficient.

<sup>b</sup>CV = clearance volume.

<sup>c</sup>PBPK = physiological-based pharmacokinetic.

<sup>d</sup>BE = bioenergetic.

PCB concentrations in zooplankton and fish were predicted using a mechanistic model parameterised for the Arctic marine ecosystem [31]. It predicted PCB concentrations that were two orders of magnitude lower than measured. The assumption of steady state was identified as a possible limitation of the food-web model for environments with high seasonal amplitude, such as the Arctic. Sensitivity analyses identified water concentration as the most sensitive input parameter for PCBs in all biota [31].

### 3.3 Linking Bioconcentration and Biomagnification

Bioaccumulation potential is an important property of substances in environmental risk assessment. As shown in the previous section there are a wide range of factors to express this potential, which also indicates there is no standardised protocol. The Technical Guidance Document from the EC [27] on environmental risk assessment

make some recommendation on how to deal with bioaccumulation (see section 3.6.1). Most current techniques estimate internal concentration for bioaccumulating substances and compare those with expected internal effect levels (e.g. [7, 13]).

Several authors have suggested a tiered approach for addressing bioaccumulation [35, 36]. Two different aspects can be considered: bioconcentration (exposure from water) and biomagnification (exposure from food) (Figure 5). Food-web models assess the bioaccumulation of a substance via the food chain. The equilibrium approach, or partitioning model, is generally used in risk assessment (e.g. the EU-TGD [27]) and is based on thermodynamic equilibrium [34]. As steady state is driven not only by thermodynamics, but also by active metabolic processes, improved prediction of toxicant accumulation requires the application of kinetic models. Major difficulty in using such models lies in the number of parameters that must be known for a wide range of species and the difficulty in obtaining some of these parameters [34].

As schematically presented in Figure 5, possible tiers for bioaccumulation modelling are:

- I. Based on equilibrium assumption ( $K_{ow}$ );
- II. Based on kinetics (uptake/elimination);
  - a. Single compartment;
  - b. Multiple compartments;
  - c. Multiple compartments and uptake routes.

Possible tiers for food-web modelling are:

- I. Simplified food chain;
- II. Generic food chain;
- III. Realistic food chain.

These approaches are further described in the remainder of this chapter.

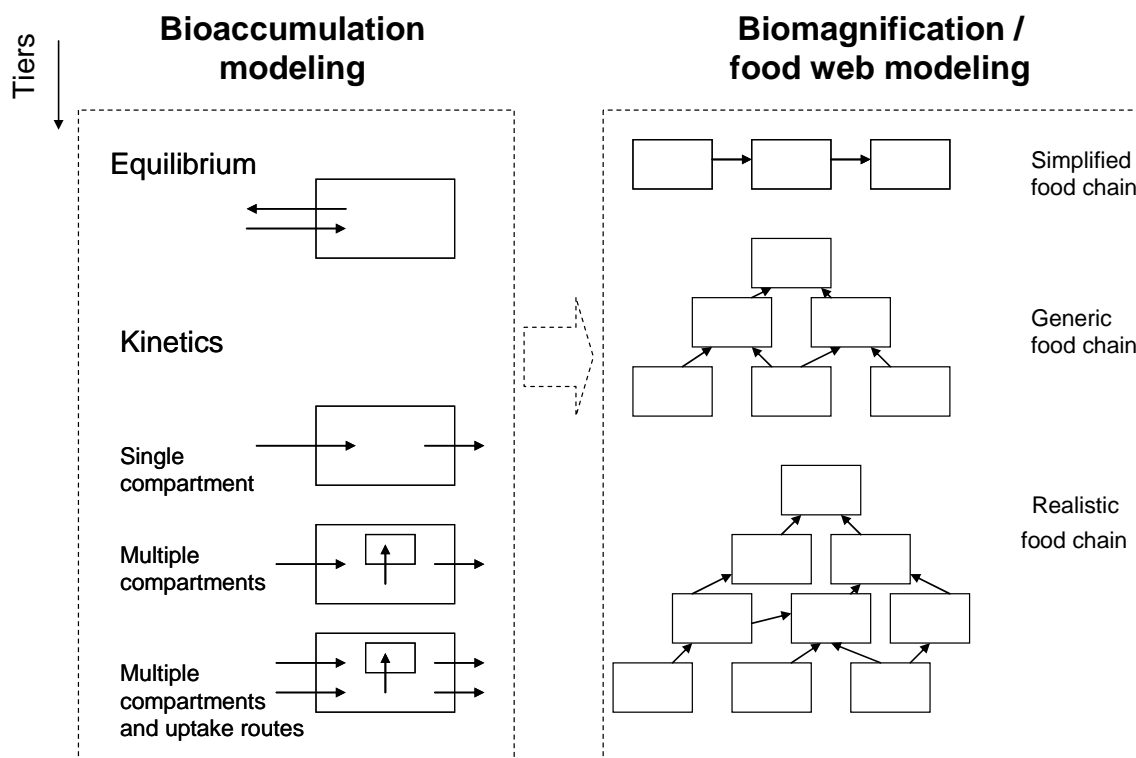


Figure 5 *Tiers in risk assessment of bioaccumulating substances with distinction in levels related to bioaccumulation and biomagnification*

### 3.4 Biomagnification

Highly bioaccumulative substances have both a very high bioconcentration potential and are also resistant to biotransformation in animals. Numerous laboratory and field studies indicate that food-web transfer in the aquatic environment is significant at higher trophic levels (secondary, tertiary consumers) only when poorly metabolised, nonpolar substances have a lipophilicity above a log Kow of 4.5 to 5.0 [3]. Although laboratory tests have revealed the correlation between the Kow and the BCF of organic chemicals, studies in real food-webs demonstrated that bioaccumulation is not solely a lipid-water partitioning process. Dietary accumulation or biomagnifications can cause additional bioaccumulation, resulting in an increase in chemical concentration with increasing trophic level in food-webs [37]. Biomagnification of such chemicals is a major risk to the top predators of food-webs, as the consumption of contaminated food is a major source of contaminants in predatory marine birds and mammals. In contrast, the direct uptake of substances from the environment (that is from water and sediment) is only of minor relevance [27].

In 1995, biomagnification was only been demonstrated for a limited number of substances, i.e. DDT (dichlorodiphenyltrichloroethane), DDE (dichlorodiphenyldichloroethylene), PCB (polychlorobiphenyl), toxaphene, methyl mercury, total mercury and arsenic were known to have the potential to biomagnify in aquatic systems [3]. Since then, an increasing number of substances have been found to biomagnify, such as PBDE (polybrominated diphenyl ethers), PCDFs (polychlorinated dibenzofurans), PCDDs (polychlorinated dibenzo-p-dioxins) and PFAS (per- and polyfluorinated alkyl substances) [2, 4, 5].

To incorporate the process of biomagnification in bioaccumulation models, food-web interactions need to be included, or at least the diet composition of the biota of interest. Models range from single-compartment, no growth, first-order depuration models to complex assemblages of proposed trophic levels with increased detail within each level. Some of these models focus predominantly on the chemical's properties, although growth dilution and other biological processes may be incorporated [3]. Most single species and food-web models are validated for temperate lakes [38]. The use of simple food chains is described in paragraph 3.6.1. However, the Arctic environment, which is the subject of this study, has very specific and unique conditions. These conditions and related consequences for food-web modelling and risk assessment of bioaccumulating substances are discussed in paragraph 3.6.2.

A more pragmatic approach to assess the risk of accumulation and transfer of chemicals via the food chain is the use of biomagnification factors which are described in section 3.5.

### 3.5 Which Substances Bioaccumulate?

Not all substances have a potential to bioaccumulate. The most important and widely accepted indication of bioaccumulation potential is a high value of the n-octanol/water partition coefficient (log Kow) or the related BCF (Bioconcentration Factor). For example, a BCF trigger value has been introduced in risk assessment (Figure 6) [3]. When the estimated BCF value is above 1,000 (related to a log Kow of 4.3) a PEC/PNEC assessment for predators is made. The scientific value of this approach has been questioned and it has been advised to consider parameters other than BCF, particularly for covering bioaccumulation from oral exposures [32]. Factors that are known to influence/determine bioaccumulation potential are:

- Log Kow
- Adsorption
- Hydrolysis
- Degradation
- Molecular mass (greater than 700 unlikely to accumulate)

Dietary uptake by aquatic organisms is considered only significant if the substance has low water solubility, high lipid solubility and is slowly metabolised or eliminated by the prey organism. Within Europe, a step-wise approach is recommended to integrate bioaccumulation in an environmental risk assessment [27]. In practice, substances which are bioaccumulative, persistent and exhibit negligible metabolism will be evaluated in this scheme. A substance is indicated as potential bioaccumulating when:

- it has a log Kow  $\geq 3$ ; or
- it is highly adsorptive; or



- it belongs to a class of substances known to have a potential to accumulate in living organisms; or
- there are indications from structural features; and
- there is no mitigating property such as hydrolysis (half-life less than 12 hours).

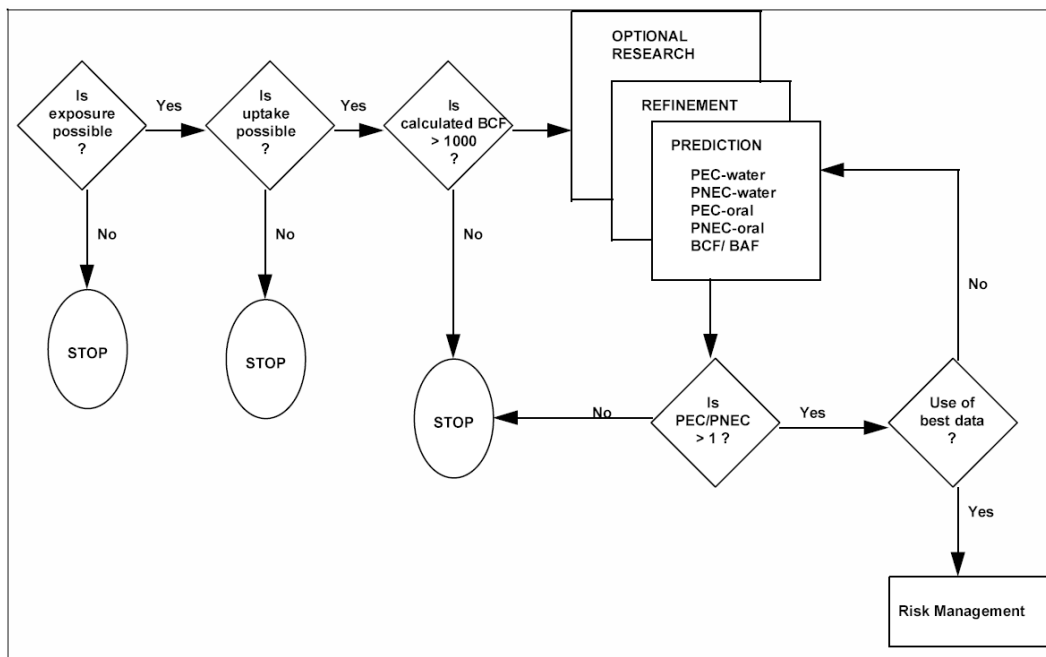


Figure 6 *Decision Flow Diagram for Integration of Bioaccumulation in a Tiered Risk Assessment Scheme [3].*

### 3.5.1 Use of Magnification Factors and Multipliers

Next to the BCF and BAF (described earlier), magnification factors can also be used to quantify bioaccumulation potential. These factors can be used to derive internal concentrations from external concentrations, but also to screen substances.

#### Biomagnification factor (BMF)

The BMF is defined as the relative concentration in a predatory animal compared to the concentration in its prey ( $BMF = C_{predator}/C_{prey}$ ) (Figure 7). The BMF should ideally be based on measured data. However, as the availability of such data is limited, the TGD provides default values (Table 4). The resulting maximum BMF from fish to top predators is 100. CSTEE [32] notes that these default values are underestimating the biomagnification for (top) predators and cite studies that found body burdens to increase 10-100 from fish to seals and 100-1000 from seals to polar bears. As mentioned previously in this report, the BMF is dependent on the basis on which the concentration is expressed (i.e. fresh weight, dry weight, lipids). The EU-TGD applies concentrations in fish on a wet weight basis and note that the concentrations used to derive and report BMF values should, where possible, be lipid normalised [27].

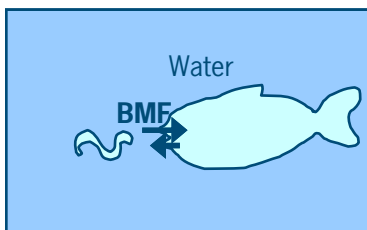


Figure 7 *The BioMagnification Factor (BMF) is described by the ratio (at equilibrium) of internal concentrations between predator and prey.*

The EU-TGD covers the biomagnification process in the marine risk assessment by multiplying the fish BCF by two BMFs (BMF<sub>1</sub> fish/predator; BMF<sub>2</sub> predator/top-predator), representing the food-biota accumulation which are estimated directly by the Kow.

Table 4 *Default BMF values used by the TGD for organic substances [27]*

log Kow	BCF (fish)	BMF <sub>1</sub>	BMF <sub>2</sub>
<4.5	< 2,000	1	1
4.5 - < 5	2,000-5,000	2	2
5 – 8	> 5,000	10	10
>8 – 9	2,000-5,000	3	3
>9	< 2,000	1	1

Besides measured data and default values, BMFs can also be calculated with the use of food-web models (Table 5). It was reported that the modelling results showed good agreement with observed concentrations [37]. The calculated BMFs, as shown above, distinguish between water-respiring and air-breathing organisms. The TGD does not consider possible differences in BMF between gill breathing and lung breathing (mammals, birds) animals. There is evidence in the literature of differences of 2-3 orders of magnitude between concentrations of chemicals with biomagnification potential in top predators with comparable behaviour belonging to the two different groups (e.g. biomagnification in dolphins much higher than in tuna) [32].

Table 5

*Calculated BMFs [37]*

**Table 1.** Calculated BMFs (25) of selected industrial chemicals in aquatic invertebrates, fish, reptiles, amphibians, birds, nonhuman mammals, and humans for organic chemicals varying in molecular weights ( $M_w$ , g·mol<sup>-1</sup>), log  $K_{OW}$ , and log  $K_{OA}$ . No metabolic transformation was assumed. Biomagnification occurs if BMF > 1. Low  $K_{OW}$ –low  $K_{OA}$  chemicals show no biomagnification in

water-respiring or air-breathing organisms because of efficient respiratory elimination to air and water. Low  $K_{OW}$  (log  $K_{OW}$  ~2 to 5)–high  $K_{OA}$  (log  $K_{OA}$  ~6 to 12) chemicals exhibit no biomagnification in water-respiring organisms but biomagnify in air-breathing organisms. High  $K_{OW}$ –high  $K_{OA}$  chemicals biomagnify in water-respiring and air-breathing organisms.

Chemical*	$M_w$ (g·mol <sup>-1</sup> )†	Log $K_{OW}$ †	Log $K_{OA}$ †	Water-respiring organisms			Air-breathing organisms						
				Zooplankton	Forage fish	Predatory fish	Reptile	Amphibian	Seabird	Marine mammal	Terrestrial herbivore	Terrestrial carnivore	Human
Low $K_{OW}$ –low $K_{OA}$													
1,4 butadiene	145	1.9	2.9	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
1,3,5 TriCBz	182	3.5	4.7	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Hexachlorobutadiene	261	4.5	3.9	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Styrene	150	2.5	4.9	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Low $K_{OW}$ –high $K_{OA}$													
Dicofol	371	3.5	8.9	<1	<1	<1	6.1	12	16	42	3.9	76	76
β-endosulfan	391	3.7	7.1	<1	<1	<1	4.9	11	10	22	2.5	28	23
β-HCH	291	3.8	8.9	<1	<1	<1	7.5	12	17	45	4.0	85	84
Musk xylene	297	4.1	8.9	<1	<1	<1	8.5	12	18	47	4.1	90	89
Trifluralin	350	4.4	7.2	<1	<1	<1	6.2	11	12	26	2.8	34	28
Tetradifon	356	4.6	11.4	<1	<1	<1	9.4	12	19	49	4.1	97	97
1,2,4,5 TeCBz	247	4.7	6.3	<1	<1	<1	2.4	7.3	3.9	7.3	1.0	7.6	5.9
High $K_{OW}$ –high $K_{OA}$													
PeCBz	247	5	6.9	<1	<1	<1	4.8	10	8.4	17	2.1	21	16
Dieldrin	381	5.4	8.8	1.7	2.4	2.3	9.6	12	19	48	4.1	94	93
HCBz	285	5.5	7.7	1.8	2.7	2.7	8.4	12	16	38	3.6	62	55
PCB 153	361	7.5	9.6	2.5	5.0	7.7	9.6	12	18	49	3.3	97	96
PCB 180	381	7.5	10.9	2.5	5.1	8.0	9.3	12	18	47	2.4	95	95
PBDE 47	308	6.0	9.8	2.2	4.0	5.1	9.7	12	19	49	4.1	98	98
PBDE 99	342	6.8	11.2	2.5	4.9	7.5	9.7	12	19	49	3.6	98	98
Mirex	546	8.1	9.6	1	1	2	8.2	10	14	16	1	82	82
PBDE 209	600	9.9	13.1	1	1	1	1	1	1	3	1	8	8

\*Chemical abbreviations not provided in the text include TriCBz (trichlorobenzene), PeCBz (pentachlorobenzene), PCB 153 (2,2',4,4',5,5' hexachlorobiphenyl), PCB 180 (2,2',3,4,4',5,5' heptachlorobiphenyl), PBDE 47 (2,2',4,4' tetrabromodiphenyl ether), PBDE 99 (2,2',4,4',5 pentabromodiphenyl ether), and PBDE 209 (2,2',3,3',4,4',5,5',6,6' decabromodiphenyl ether). †See supporting online material for physical-chemical properties.

In this Table, molecular weights were incorrectly reported for six chemicals. The corrected molecular weights (in parentheses) for the following compounds are: trifluralin (335); 1,2,4,5 TeCBz (216); PCB 180 (395); PBDE 47 (486); PBDE 99 (565); and PBDE 209 (960).

Figure 8 shows the relationship between substance properties  $K_{ow}$  and  $K_{oa}$  (octanol-air partitioning) and food-web magnification. Substances require sufficient lipophilicity to get 'stuck' in fatty tissues, but should not be too lipophilic to be able to pass through biological membranes. This is why the plot shows optimum  $K_{ow}$  for magnification. It also shows that substances with a wider range of  $K_{ow}$  values are able to magnify in the higher trophic levels (the mammals)

Although less hydrophobic compounds ( $K_{ow} < 10^5$ ) such as hexachlorocyclohexanes (HCHs) do not biomagnify in the piscivorous food-web, they have shown a high degree of biomagnification in air-breathing organisms of the marine mammalian food-web [37]. The study of Kelly et al. [37] shows that although substances with a  $K_{ow}$  below  $10^5$  cannot biomagnify in fish, they can biomagnify in birds and mammals. It seems that for substances with an octanol-air partition coefficient ( $K_{oa}$ )  $\geq 10^6$  and a  $K_{ow} > 10^2$ ,  $K_{ow}$  and the BCF in fish are not good predictors of biomagnification in air-breathing animals (Figure 8).

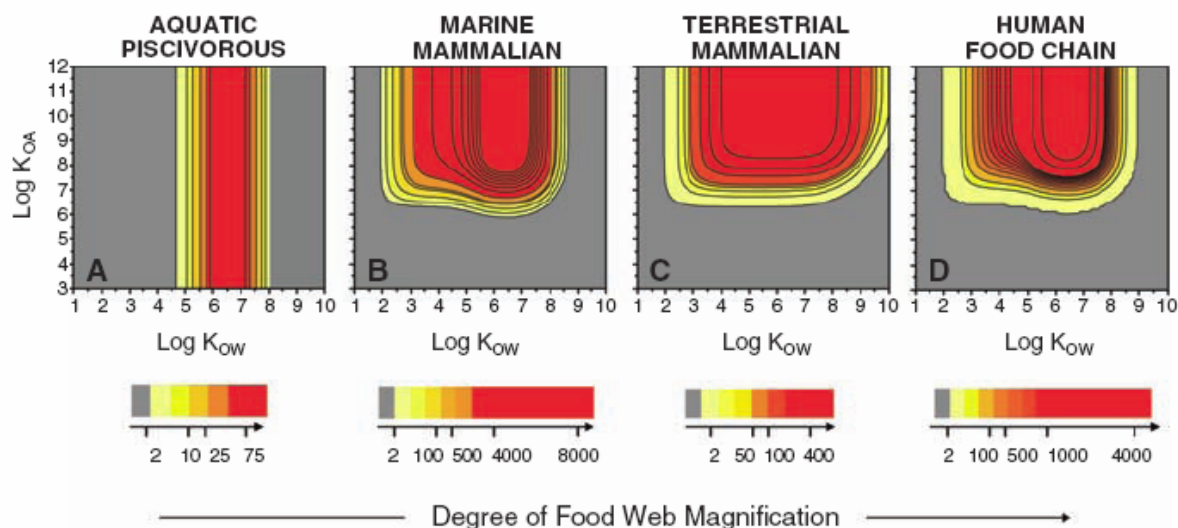


Figure 8 *Contour plots illustrating the relationship between chemical Kow (x axis), Koa (y axis), and food-web magnification (z dimension represented as contours) in the aquatic piscivorous food-web (A), marine mammalian food-web (B), terrestrial mammalian food chain (C), and Arctic indigenous human food chain (D). The data represent the combined magnification of the chemical concentrations (ng.g<sup>-1</sup> lipid equivalent) in the top predator over the concentrations at the base of the food-web [e.g., primary producers at trophic level (TL) = 1 to polar bear at TL = 5.4]. A matrix table was generated with ~30,000 Kow-Koa combinations over a log Kow range of 1 to 10 and a log Koa range of 3 to 12. These data demonstrate the combined effect of Kow and Koa on chemical bioaccumulation [37].*

In mammalian top predators, the assessment of a BMF is important to assess internal concentrations and possible transfer to milk, producing toxic effects on young specimens. Marine mammals transfer large amounts of fat soluble and persistent substances to their offspring during periods of lactation which may last from a couple of weeks (certain seal species) to 2 years (polar bear). The milk has a fat percentage ranging from 30-60%. Thus, marine mammal offspring are potentially exposed to a large amount of fat soluble substances during periods of development and maturation of vital organs [32].

BMFs are not only the result of exposure (e.g. diet) but also of elimination (e.g. biotransformation) [38]. Many organochlorines, such as PCBs, will have BMFs mainly reflecting the half-life of the compound in the specific organism. It was found for example that harbour porpoises have more difficulties in metabolising several PCB and PBDE congeners compared to harbour seals, concluding that biomagnification in the Southern North Sea mainly occurs in harbour porpoises [39].

### Food chain multipliers (FMs)

Bioaccumulation considerations are integrated into the USEPA water quality criteria equations by using food chain multipliers (FMs) in conjunction with the BCF [40]. The bioaccumulation and bioconcentration factors for a chemical are related as follows:  $BAF = FM \times BCF$ . In case measured BAF's are not available, estimated FMs can be used. In Table 6, FM values are listed according to log Kow value and trophic level of the organism. In this table, FMs keep increasing with increasing Kow values. As substances with very high Kow usually don't magnify (Figure 8), this is not realistic but conservative.

Table 6

*Estimated Food Chain Multipliers (FMs) [40]*

Log Kow	Trophic Levels		
	2	3	4
3.5-3.9	1.0	1.0	1.0
4.0	1.1	1.0	1.0
4.1-4.3	1.1	1.1	1.1
4.4	1.2	1.1	1.1
4.5	1.2	1.2	1.2
4.6	1.2	1.3	1.3
4.7	1.3	1.4	1.4
4.8	1.4	1.5	1.5
4.9	1.5	1.8	2.0
5.0	1.6	2.1	2.6
5.1	1.7	2.5	3.2
5.2	1.9	3.0	4.3
5.3	2.2	3.7	5.8
5.4	2.4	4.6	8.0
5.5	2.8	5.9	11.0
5.6	3.3	7.5	16.0
5.7	3.9	9.8	23.0
5.8	4.6	13.0	33.0
5.9	5.6	17.0	47.0
6.0	6.8	21.0	67.0
6.1	8.2	25.0	75.0
6.2	10.0	29.0	84.0
6.3-6.4	13.0	34.0	84.0
6.5	19.0	45.0	100.0
≥6.5	19.2*	45.0*	100.0*

\* These recommended FMs are conservative estimates; FMs for log Kow values greater than 6.5 may range from the values given to a low as 0.1 for contaminants with very low bioavailability.

#### **Trophic magnification factors (TMFs)**

Trophic magnification factors (TMFs) have been used to describe the increase of organochlorines from one trophic level to the other [38]. TMFs are derived from the slope of the relationship between an organism's log lipid-normalised organochlorine concentrations and trophic position. Borga et al. [38] provide TMFs for selected persistent organic pollutants. TMFs might be used to replace other parameters with unknown values in most food-webs (e.g. assimilation efficiencies and feeding rates in individual species).

#### **Food-web magnification factors (FWMFs)**

Food-web magnification factors (FWMFs) and biomagnifications factors for selected prey-predator scenarios were calculated to describe pathways of heavy metals in the Arctic [41]. Although it is recognised that metal concentrations in animals are probably not related to the trophic level in the food chain, some metals (e.g. methyl-mercury, zinc) may potentially be influenced by the numbers of trophic interactions involved [30, 42]. The study of Dehn et al. [41] indicated that magnification of the heavy metals silver, cadmium and total mercury in the Arctic food-web is not significant.

## 3.6 The Food-Web

### 3.6.1 Simple Food Chain

The EU-TGD calculates the concentration in fish to represent the intake via food for predators ( $PEC_{\text{coral}_{\text{predator}}}$ ), which is based on both the BCF and the BMF [27]. This approach has been questioned with the argument that it is ecologically incorrect to start the food chain with fish [32].

The TGD provides a relatively simple food chain which consists of the marine water phase, marine food, marine fish and two separate levels of predators (Figure 9). It has been recommended that biomagnification should be based on at least 4 levels [32].

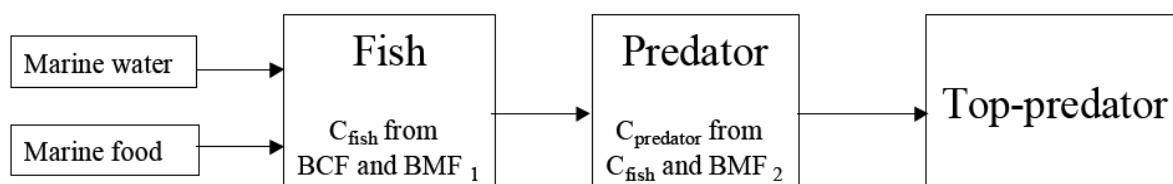


Figure 9 *Assessment of secondary poisoning according to the TGD [27]*

In the relatively simple food chain given above (Figure 9) the concentration in the fish (i.e. the food for the fish-eater) ideally should take account of all possible exposure routes, but in most instances this will not be possible because it is not clear what contribution each potential exposure route makes to the overall body burden of a contaminant in fish species. Therefore for very hydrophobic substances a simple correction factor for potential biomagnification on top of the bioconcentration through the water phase is applied [27]. Food chains of the marine environment can be very long and complex and may consist of 5 or more trophic levels. According to the TGD [27], the possible extent of bioaccumulation in marine food chains with more than the above three to four trophic levels should be evaluated case by case if necessary input data for such an evaluation is available, using the principles for the shorter food chain. Also if further data are available it may be possible to refine the assessment of secondary poisoning via marine food chains by employing more advanced modelling that takes the differences in, for instance, uptake and metabolic rates into account for the different trophic levels.

### 3.6.2 The Arctic Environment

The Arctic ecosystem has a number of unique attributes, including long food chains, reduced diversity of species, similar food-webs across the entire region, and limited influence from pollution point sources [38]. A simplified schematic diagram showing the Arctic marine food-web is presented in Figure 10. Recent studies of arctic marine food-webs have been summarised, with an emphasis on identifying important ecological factors for explaining variability of persistent organochlorine (OC) concentrations among organisms [38]. They found that: "Lipid content, body size, age, gender, reproduction, habitat use, migration, biotransformation, seasonal changes in habitat conditions, feeding ecology, and trophic position have all been demonstrated to influence OC concentrations and bioaccumulation in arctic marine biota. The relative importance of each factor varies among OCs and organisms. Diet or trophic level is the dominant factor influencing OC concentrations and dynamics in seabirds and marine mammals, although biotransformation can significantly influence nonrecalcitrant OCs, such as hexachlorocyclohexane isomers. Dietary accumulation of OCs is also an important route of exposure for arctic fish and zooplankton, and biomagnification of OCs may also occur among these organisms. To date, only limited attempts have been made to model trophic transfer of OCs in the arctic marine food-web. Although models developed to assess OC dynamics in aquatic food-webs have included some biological variables (e.g., lipid content, feeding rate, diet composition, and growth rate), selection of processes included in these models as well as their mathematical solutions and parameterisation all introduce simplification. This reduces biological validity of the models and may be particularly problematic in a highly seasonal environment, such as the Arctic Ocean." [38].

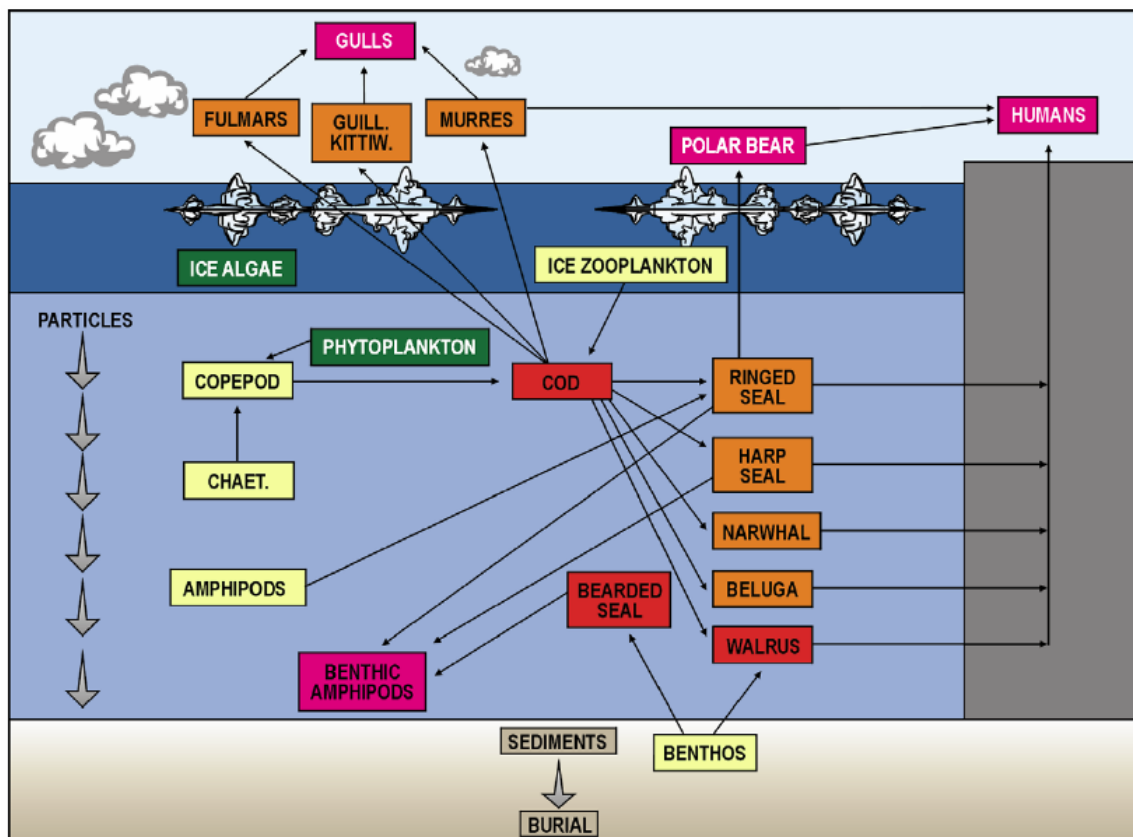


Figure 10 A simplified schematic diagram showing the marine food-web in the Arctic environment [43].

Factors that make very hydrophobic substances of particular concern to the marine environment include:

- longer food chains,
- migratory and reproductive aspects that may cause especially high exposure of progeny of marine species likely,
- long-life of many marine predators, and
- higher fat content.

The assumption of equilibrium may not be valid in a highly fluctuating situation such as the arctic ecosystem [31, 38]. During high-productive months (summer) when the ecosystem undergoes rapid changes in terms of primary production, concentrations in water and organism lipid content, equilibrium may not be reached and thus should not be used to describe the current process. The influence of seasonality on organochlorine bioaccumulation has been studied in terms of primary production and bioavailability, seasonal lipid dynamics because of reproduction and build up of reserves in invertebrates and seabird [38]. However, Borga et al. [38] found no studies over a complete-year cycle. Borga et al. [31] found a large difference between measured and predicted PCB concentrations in arctic biota, too large to be explained by temporal variation and equilibrium only. Reducing growth rate in the model and therewith internal dilution also did not account for the large difference.

Patwa et al. [44] explored the role of mysids in benthic-pelagic coupling and biomagnifications using a dynamic bioaccumulation model. The inclusion of mysids in a simple food-web model demonstrates higher concentration in upper-level organisms by two effects: introduction of another trophic level in the food-web and increased benthic-pelagic coupling.

Factors shown to be of importance for describing differences in OC concentration and pattern among species are as follows [38]:

- Organism's body size
- Habitat use (e.g., benthos are exposed to OCs bound to particulate matter that sediments)
- Reproductive status
- Seasonality
- Biotransformation
- Trophic position

Cannibalism and scavenging of dead conspecifics generally results in an increase in concentration by self-biomagnification, but the effect is small and unlikely to exceed 5% on the average [45].

Slow growth at the primary producer level is a likely contributor to higher biotic metal concentrations in shaded, oligotrophic, or cold ecosystems [46].

Even organisms such as zooplankton show highly species-specific bioaccumulation of contaminants [38].

The ECETOC study [3] points out that the use of food-web models may still suffer from the lack of knowledge in:

- feeding frequency of predators on contaminated prey;
- bioavailability of the substance in food and water;
- estimates of metabolism in predators;
- dietary uptake efficiency;
- diet and lifespan.

These factors will be taken into account when evaluating the currently available food-web models.

In recent work De Laender et al. used a dynamic food-web model (using inverse modelling techniques) in combination with a kinetic bioaccumulation model. They showed that the dynamic food-web (extending up to cod in the food chain) only explained a small fraction of the variance in bioaccumulation of PCBs [47].

### 3.7 Comparison of a Selection of Food-Chain Based Bioaccumulation Models

#### 3.7.1 Introduction

There are several models available that calculate bioaccumulation along the food-chain. Four bioaccumulation models from our search of literature (Appendix B) showed useful features for our purposes. One of the first kinetic models used for bioaccumulation calculations is the model of Gobas et al. [48] and has served as a basis for other models. Hendriks et al. [12], for instance, used the principles of the Gobas model and extended them in the OMEGA model. Traas et al. [13] also used the Gobas model as a basis, however, his group also incorporated effect levels into the model in order to derive environmental quality standards for bioaccumulating substances. More recently Alonso et al. [35] proposed to use a tiered approach in calculating bioaccumulation. In an example they implemented a simple kinetic model, which could be extended if necessary. The four models serve as candidates for bioaccumulation modelling in the intended risk assessment tool. In the following sections each model will be discussed separately in more detail, after which the models will be compared.

#### 3.7.2 Gobas: Kinetic Model Combined with a Trophodynamic Food-Web

Gobas et al. [48] presents a steady-state model for estimating concentrations of hydrophobic organic substances in various organisms of aquatic food-webs, including fish, benthos, macrophytes, aquatic plants and others, from chemical concentrations in the water and sediments. The model combines the toxicokinetics of chemical uptake, elimination and bioaccumulation in individual organisms and the trophodynamics of food-webs to estimate chemical concentrations in different organisms of food-webs. This food-web model differs from simple four-level generic food-chain models by incorporating multiple feeding interactions, including benthic and pelagic food-chains, and its ability as a generic model to apply to food-webs on a site-specific basis. The model is relatively simple and requires only few input data, which makes it easy to apply for practical use. Data requirements are regarding the types of species of the food-web, weights, lipid contents and trophic interactions (see Appendix C



for an overview for most important factors used in the model). Thus with little effort, predictions of chemical concentrations in various organisms can be made from concentrations in water and sediments. However, the model does not include top predators. For simulations of Lake Ontario, the model predictions were accurate within a factor of 2 to 3.

The model assumes that chemical uptake in phyto- and zooplankton are predominantly from the water and that uptake from food consumption is insignificant. This assumption is based on the rapid organism-water exchange (due to small size of the organism) which reduces the effect of dietary magnification. Food-web accumulation of organic chemicals therefore occurs predominantly in fish. The model includes:

- Bioavailability
- Growth
- Chemical uptake (water, gill, diet)
- Chemical elimination (water, gill, faecal)
- Lipid content
- Metabolic rate

The chemical concentration in the predator is related to that in its prey. The trophodynamics therefore play an important role in the transfer of chemicals through the food-chain and the accumulation of chemicals in the organisms of the food-web [48].

### 3.7.3 Hendriks: Kinetic Model Combined With a Generic Food-Chain (OMEGA)

The OMEGA model considers influx and efflux to depend on several variables: the octanol–water partition ratio of the substance and the weight, lipid content, and trophic level of the species [12]. OMEGA distinguishes up to four trophic levels in marine and fresh water food chain (Figure 11). Phytoplankton is the first level of the food chain, followed by the zooplankton (herbivores), which feeds on the phytoplankton. The zooplankton is eaten by small fish (primary carnivores) that are consumed by large fish (secondary carnivores). The mass of organisms and populations is governed by four basic flows (Figure 12). The model was calibrated and validated on aquatic species. OMEGA describes more processes than the Gobas model, using more parameters (see Appendix C). Depending on the parameter, about 30 to 71% of the variation was explained by the model.

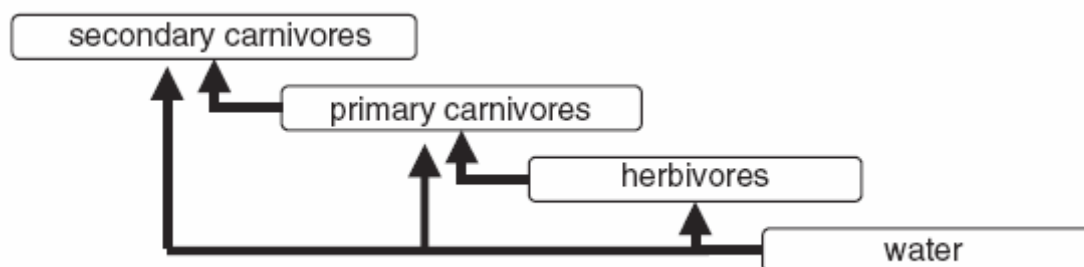


Figure 11 *Scheme of food chain applied in OMEGA [25].*

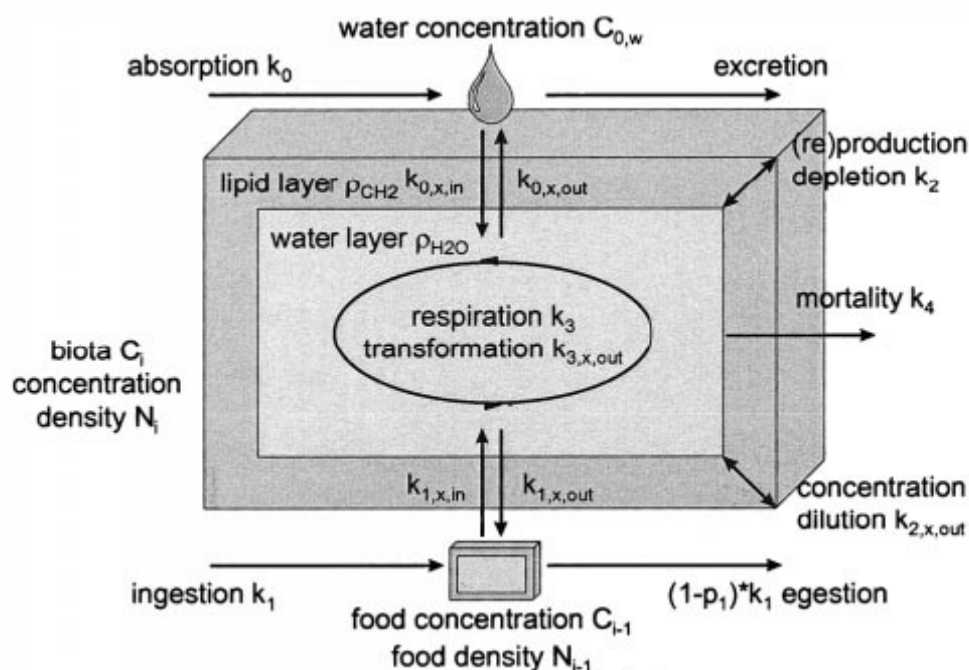


Figure 12 The densities of organisms  $N_i$  and of their food  $N_{i-1}$  are determined by metabolic flows at rate constants  $k_0$  for absorption and excretion of water,  $k_1$  for ingestion and egestion of food,  $k_2$  for (re)production,  $k_3$  for respiration, and  $k_4$  for mortality of mass. The concentrations in organisms  $C_i$  and their food  $C_{i-1}$  are determined by the lipid  $\rho_{CH2}$  and water  $\rho_{H2O}$  resistance as well as by the metabolic flows that carry substances  $X$  into and out of organisms. This occurs at rate constants  $k_{0,x,in}$  for absorption from water,  $k_{1,x,in}$  for assimilation from food,  $k_{0,x,out}$  for excretion with water,  $k_{1,x,out}$  for egestion with food,  $k_{2,x,out}$  for dilution with biomass, and  $k_{3,x,out}$  for transformation [12].

Smítková et al. [25] compared three fish bioaccumulation models: EUSES (European Union System for the Evaluation of Substances), CalTOX (a seven compartment's multimedia human exposure model) and OMEGA (a mechanistic bioaccumulation model). Main difference between the models is that OMEGA includes food as an additional pathway compared to EUSES and CalTOX, which are based on water exposure only. The study showed that all models are virtually similar up to a  $Kow$   $10^6$ . At higher  $Kow$  calculations based on water exposure only (EUSES, CalTOX) decreased parabolically whereas calculations including food exposure are almost linear. They also compared the modeled data to field measurements indicating that uptake from food is an important contribution, as implemented in the OMEGA model.

### 3.7.4 Traas: Food-Web Model Combined with Effect Concentrations

Traas et al. [13] applied Internal Effect Concentrations (IECs) (IEC is identical to the CBR, with that difference that CBRs usually imply mortality, while IECs indicate any effects) (see Chapter 4) in conjunction with a food-web model. First, the EICs were established using data from the online database ERED. Five different classes were established (see below). By combining the matrix framework for bioaccumulation with efficient estimation of rate constants and the concept of internal effect concentrations, bioaccumulation can be incorporated easily in risk assessment and derivation of EQCs (Environmental Quality Criteria) for narcotic compounds [13]. A shallow lake food-web was used in this study. The food-web derived quality criteria depend on specific food relations and thus are ecosystem specific.

Table 1. Statistics for internal effect concentrations (IECs) based on selected entries from the environmental residue and effect database (ERED). Appendix 2 contains the names of the chemicals for each mode of action. CNS = Central nerve system, AChE = Acetylcholinesterase, AhR = Aryl-hydrocarbon receptor, NS = Not significant, *n* = Number of data

Difference between IEC50 and IEC100 <sup>a</sup>	<i>n</i>	Differences between chemical classes		Normal distribution of log IEC, log (mol/g wet wt)	
		Median IEC (mol/g wet wt)	Indication for significant difference <sup>b</sup>	Mean	Standard deviation
Mode of toxic action					
Narcosis (NS)	63	5.75E-6	A	-5.24	0.53
Polar narcosis (NS)	96	1.64E-6	B	-5.92	0.62
AChE inhibitors (***)	26	3.82E-7	C	-6.53	1.2
Uncouplers (NS)	15	1.6E-7	C	-6.76	0.34
CNS-convulsants (NS)	21	2.60E-8	D	-7.91	1.13
Respiratory blockers <sup>c</sup>	4	7.64E-10	D	-9.13	0.13
AhR-disruptors (NS)	16	9.32E-12	E	-11.49	0.94

<sup>a</sup> NS =  $p > 0.05$ ; \*\*\* =  $p < 0.001$ .

<sup>b</sup> Classes with different letters are significantly different groups.

<sup>c</sup> Only IEC100 data available.

### 3.7.5 Alonso: Tiered Model

Alonso et al. [35] reviewed kinetic models that cover species from different trophic levels and ecological behaviour. Based on this study, the authors proposed the development of a tiered conceptual biomagnification model, starting with a simplified food chain which can be refined to more realistic and complex models in successive levels.

Alonso et al. [35] have developed a conceptual model for estimating the biomagnification potential in a generic food-web, which was mathematically implemented through system dynamic models developed under data software. The authors conclude that the mathematical implementation of the conceptual model offers tools for estimating the potential for bioaccumulation and biomagnifications of chemical under very different conditions. The versatility of the model can be used for both comparative estimations and for validating the model. The presented model covers the time variation (daily time segments) of bioaccumulation using just a few toxicokinetic parameters. For validation of the model, the food chain structure of the Barent Sea has been used for simulating the biomagnification of PCB153 which was compared with measured concentrations. The approach for implementing time as well as predicting the concentrations within a food chain were proved to be acceptable [35].

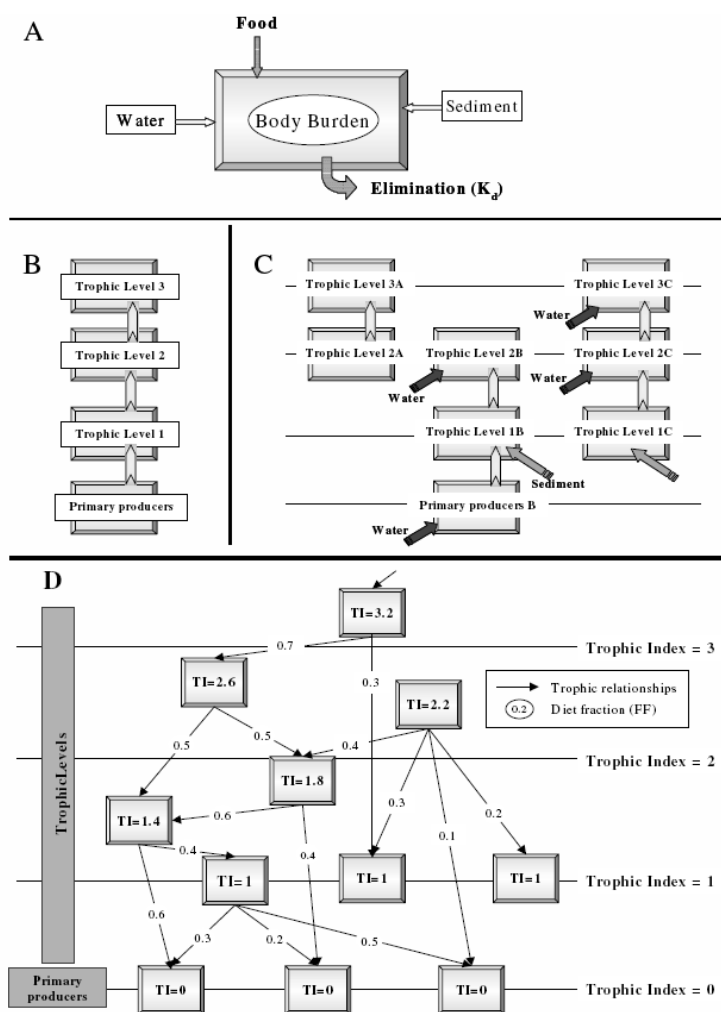


Figure 13 Schematic diagram of biomagnification model steps. A: Kinetic model regulating the mass balance for each organism considering several exposure routes (food, sediment and water). B, C and D are consecutive framework model steps which correspond to three complexity levels of the model: simplified food chain, aggregation of generic food chains and realistic food chain where each organism have an associated Trophic Index (TI) [35].

### 3.7.6 Summary and evaluation

The four available models can be evaluated according to the criteria as provided in Table 7. The models considered in this report all include some important aspects, i.e. they are based on kinetics, include multiple uptake routes, biotransformation and lipid size. The model of Alonso et al. [35] includes not as many aspects compared to the other models. Traas et al. [13] already includes effect concentrations in the model, which is part of the objectives of the proposed model.

Table 7

*Aspects of some available bioaccumulation models*

<b>Aspect</b>	<b>Gobas (1993)</b>	<b>Hendriks (2001)</b>	<b>Traas (2004)</b>	<b>Alonso (2008)</b>
Based on kinetics	X (assuming steady state)	X (assuming steady state)	X	X
Multiple compartments	-	-	-	-
Multiple uptake routes	X	X	X	X
Toxicodynamics	-	-	-	-
Organism's body size/growth	X	X	X	-
Lipid content	X	X	X	-
Habitat use	-	-	-	-
Reproductive status	-	-	X	-
Seasonality	-	-	-	-
Biotransformation	X	X	X	-
Trophic position	X	X	X	X
Effect concentrations	-	-	X	-
Validation	X (PCB in lake)	X (several organic and inorganic substances in aquatic environment)	X (PCB in lakes)	X (PCB in open marine environment)

### 3.8 Conclusions and recommendations

From the text above, the following is concluded:

- Kow (equilibrium approach) is a simple and widely accepted approach to assess bioconcentration. However, for our purpose the approach is unsuited as bioaccumulation is not solely a lipid-water partitioning process. It is even indicated that the key factor controlling bioaccumulation in marine mammals is metabolism, and not equilibrium partitioning. Furthermore for surface active substances (e.g. PFOS), the Kow is an irrelevant parameter as no partitioning will take place.
- The assumption of equilibrium may not be valid in a highly fluctuating system as the Arctic environment.
- Incorporation of lipid content improves exposure assessment for ecological risks substantially
- The basis of expressing concentrations (e.g. wet weight, dry weight, lipids, target organ) should be considered for each group of chemicals (e.g., µg/kg lipid for lipophilic substances).
- There are multiple routes for assimilation and elimination (e.g., uptake through gills, metabolism, ingestion from food, etc.).
- Dynamic food-web interactions contributed to only a small fraction of the total variance in a bioaccumulation model.
- The model of Gobas et al. [48] provides a basis for the other three selected models., where the models of Hendriks et al. [12] and Traas et al. [13] use more sophisticated processes to describe their model. Traas et al. [13] in addition includes effect levels in their model. Alonso et al. [35] use a more simplistic kinetic model (which can be extended) in an useful tiered approach.

Based on the conclusions and the text above, we suggest the following:

- Use a kinetic model to calculate internal concentrations. The tiered approach of Alonso et al. [35] is a useful approach as one starts with a simple food chain and require little data/parameters. However, in this case, the kinetic model needs to be extended as it is most likely over simplistic (all elimination routes are described by a single parameter). Hence elements from the other three models (Gobas et al. [48], Hendriks et al. [12] and Traas et al. [13]) should be included. Parameter values need to be collected based on the level of complexity of the model. See appendix C for an overview of the most important parameters used in each of the four models.
- The complexity of the food-web also depends on whether (top-predators) need to be included. The screening of the petro-industry related substances (Chapter 2) will reveal up to which trophic level these substances accumulate. Trophic levels that are not reached by those substances don't need to be included in the bioaccumulation model.
- Seasonal fluctuations are relevant, for the arctic environment, however required data to properly model these fluctuations are unavailable. Hence a single situation needs to be selected based on worst case assumptions (the moment where the exposure to background contaminants and the ecosystem sensitivity is the highest).

## 4 Effects

### 4.1 Introduction

This chapter describes different aspects of effect assessment of bioaccumulating substances. Effect assessment of bioaccumulating substances differs from the conventional effect assessment as it is based on internal exposure instead of external. Therefore, internal effect concentrations are required, such as the Critical Body Residue (CBR), described in the following paragraph. Other important aspects of effect assessment of bioaccumulating substances are:

- Exposure duration (paragraph 4.3)
- Effect models (paragraph 4.4)
- Species extrapolation (paragraph 4.5)

Furthermore, some known effect concentrations and data availability are described in paragraph 4.6.

### 4.2 Critical Body Residue (CBR) concept

The CBR concept can be used as an alternative for external exposure-based toxicity criteria. It can be related to external exposure (e.g. PEC) through the bioconcentration factor (BCF or BAF) and therefore used in risk assessment (Figure 14 and Figure 15). The main assumption is that exposure to an external concentration is related to the concentration within the organism through the BCF. Hence, the internal effect concentration (CBR) is a function of exposure and bioconcentration [3]. The CBR can be determined from laboratory data, but can also be established using field observations for species that for instance cannot be kept in the laboratory.

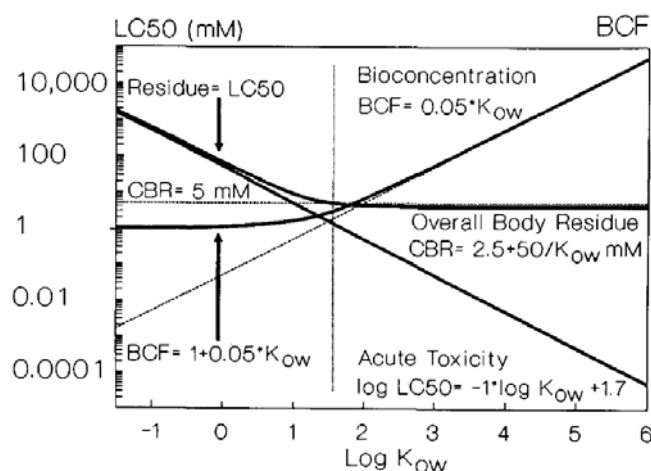


Figure 14 *Idealised relationships between acute toxicity (LC50), bioconcentration (BCF), critical body residue (CBR) and log Kow for small aquatic organisms exposed to narcotic organic chemicals [49].*

An important assumption within the CBR concept is that substances with the same mechanism of toxicity exhibit similar CBR [3], where internal concentrations of substances with the same mode of action need to be summed. Several modes of action can be discerned [13]: nonpolar and polar narcosis; Unspecific reactivity; Specific action. Indeed, an evaluation of a wide range of chemicals on the applicability of the critical body residue (CBR) approach indicated that empirical data do not support broad application of the CBR concept across chemical classes [50].

The concept of CBR appears promising but many questions need an answer before it can be used in the risk assessment of substances. In 1995, the main gaps for the use of CBR were summarised as [3]:

- the need for research to establish threshold tissue concentrations;
- the need to expose a range of taxa to ensure that sensitive species are included;
- data requirements to handle proportionality issues between whole-body and target concentrations.

Limitations found in literature to the tissue thresholds/residue approach are:

- toxicodynamics must be considered [34];
- may not work for very short exposures (compound distribution among the tissues must be at steady state) [34];
- for each compound the CBR should be established [51];
- it is necessary to understand how chemicals interact [51];
- speciation of metals should be taken into account [51];
- metabolic transformation complicates the approach [51].

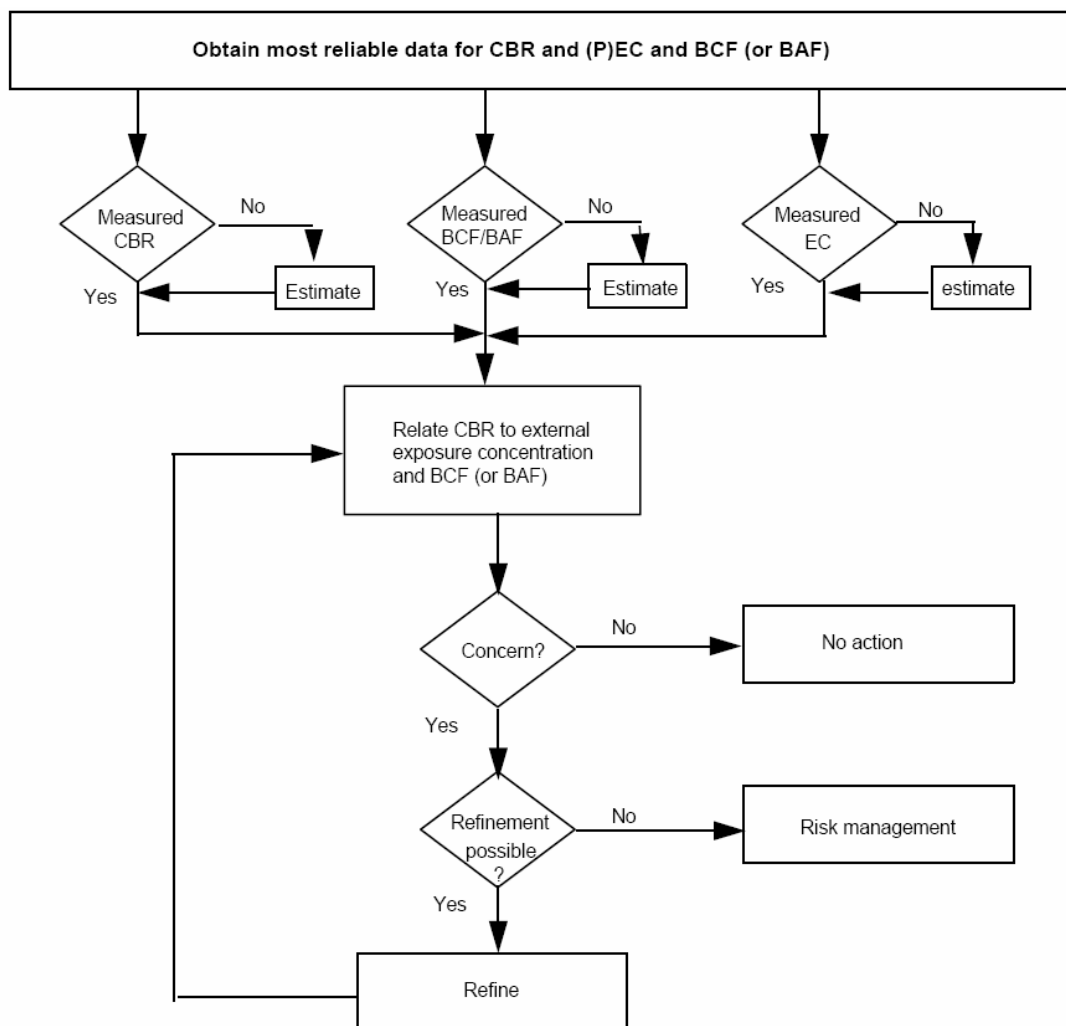


Figure 15 *Process map indicating a way to introduce the CBR concept in the environmental risk assessment of a substance [3].*

Next to the tissue-based approach, exposure thresholds can also be expressed as dietary intake [52]. In this case the diet composition and concentration of the substance in the diet needs to be known. As this generally requires more extensive studies, these data are scarce. Furthermore, waterborne exposure is not considered in oral thresholds. However, if available and waterborne exposure is negligible, they can be used next to tissue-based threshold levels.



### 4.3 Exposure duration

Standard assays of ecotoxicological effects usually provide information about the direct toxic effects of a substance. For those substances which reach steady-state within the organism within the duration of the toxicity test, direct effects of bioconcentration are included, and thus the effect value derived from this testing is appropriate for use in the risk assessment. However, for lipophilic substances which are taken up and depurated very slowly by fish or other organisms, the "time to reach steady-state" should be considered in evaluating effect values for these substances.

Lee et al. [53] studied the bioaccumulation and toxicity of PAH in the freshwater Amphipod *Hyalella azteca*. With exception of naphthalene, the PAHs were in approximate equilibrium between lipid and water at steady state and results were in reasonable agreement with a first-order kinetic model with no biotransformation. However, after the CBR reached a steady state, the toxicity still increased with time. Thus, the first-order kinetic model with  $LC_{50}$  and CBR constant at steady state (the constant CBR model), described and predicted the bioconcentration process but failed to relate the bioconcentration process to the time course of toxicity of PAH in *H. azteca* [53]. The constant CBR model underestimates the  $LC_{50}$  value at short exposure times and overestimates the  $LC_{50}$  as time increases [54]. The toxicity time course is determined not only by bioconcentration kinetics. Therefore, to describe and predict the time course of toxicity, toxicodynamic models must be developed as well as toxicokinetic models. The toxicodynamics process is independent of the toxicokinetic process. Toxicokinetics is what the organism does to a chemical and toxicodynamics is what a chemical does to the organism.

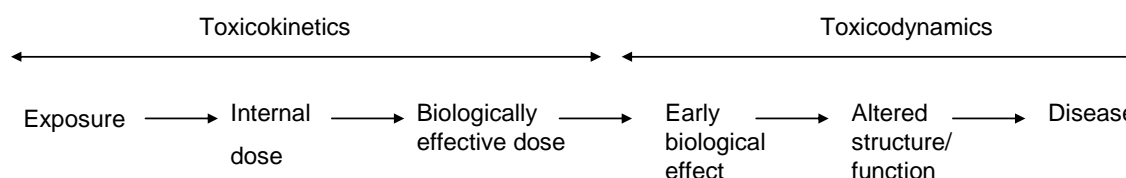


Figure 16 *Toxicokinetics and toxicodynamics [55], where toxicokinetics basically describes the process of bioaccumulation and toxicodynamics describe the processes involved in the expression of the ultimate effect*

### 4.4 Toxic effect modelling

One of the key issues for applying CBR in the environment is the endpoint considered (among others lethal, neurological, immunological, reproductive, biochemical effects) [3]. Toxic effect models tend to differ in the used single-species toxicity data, the effects they consider and the concentration-effect function used [56]. In a study of De Laender et al. [56] different toxic effect sub-model were incorporated in an ecosystem model based on a planktonic system (two phytoplankton objects, one macrophyte object and three zooplankton objects). The planktonic ecosystem model with a toxic effect sub-model incorporating mortality effects using a logistic-effect function made accurate predictions for most populations. Additional incorporation of sub-lethal effects did not result in better predictions. Predictions based on linear concentration-effect functions were less accurate.

Effects are related to the target site concentration (see also Figure 3 in Chapter 3.1). Target site concentrations are considered more suitable in comparisons of the toxicity of chemicals as well as comparisons of species sensitivity [24], compared to total concentrations. However, most toxic effect models are based on the total (internal) concentration.

Figure 17 shows sensitivity distributions expressed as toxic ratio (TR), the ratio of the EC for baseline toxicity to the experimental EC. It is obvious from these plots that TRs of baseline toxicants are close to 1 and interspecies differences are small. In contrast, TRs are several orders of magnitude higher and vary strongly for reactive and specifically acting chemicals and differences in sensitivities are much larger.

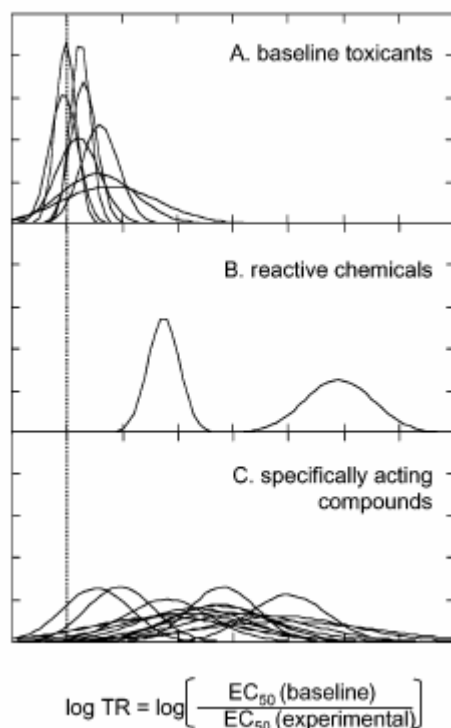


Figure 17 Species sensitivity distribution (SSD) of toxicities of various chemicals according to classes of mode of action. Each of the bell-shaped curves corresponds to a normal distribution with a median corresponding to the 50th percentile of the experimental SSD and a standard deviation derived from the experimental SR95:5-ratio, which is defined as the ratio of 95th to 5th percentile. A. SSD of the baseline toxicants acetone, *o*-cresol, ethyl acetate, heptanol, phenol, propanol, pyridine, and trichloroethylene. For the derivation of the toxic ratio (TR), Kow values given in ref 113 were converted to Kmw values. EC-values for baseline toxicity were estimated with the QSAR for *Poecilia reticulata*. B. SSD of two reactive chemicals, salicylaldehyde and propenal. TRs were taken directly from ref 113, i.e., derived from Kow-based QSARs. C. SSD of specifically acting compounds, including AChE inhibitors and neurotoxic agents: methomyl, carbaryl, parathion, dibrom, fenthion, malathion, dichlorvos, diazinon, aldrin, dieldrin, endrin, heptachlor, lindane, and toxaphene [24].

#### 4.5 Species extrapolation

When CBRs or other effect thresholds are not available for specific species, values might need to be extrapolated from other data. Luttik describes several options to extrapolate toxicity values for birds and mammals from laboratory species [57]. It is recognised that obtaining PNEC values for predatory marine birds and mammals in estimating food chain effects is difficult [32]. The EU Scientific Committee on Toxicity, Ecotoxicity and the Environment notes that caution should be taken in extrapolating too readily from laboratory animals since there are major differences in physiology between these and the marine species that ought to be taken into account [32]. They describe the following: 'This is also true for freshwater species, but it is probably much more important for marine species. Major differences occur between laboratory species like rats, mice and chicken and marine mammals and seabirds. For example, marine mammals during a year undergo dramatic changes in their nutritive condition, and have a very unique fat dynamics. Seals undergo long periods of fasting during moulting and pup production. Also delayed implantation is practiced.'

#### 4.6 Effect concentrations

Internal effect concentrations are available for polar and nonpolar narcotics [13]. The tissue residue for a wide range of neutral narcotics ranges from 2 to 6 mmol kg<sup>-1</sup> wet weight for small fish and invertebrates to yield 50% mortality for acute exposures [34]. For 50% mortality, the residue concentration for chronic exposure to nonpolar

narcotics is about 10% of that required to produce the acute response. Poorly metabolised nonpolar narcotic chemicals are assumed to have a contact threshold for exposure concentration and time. The CBR is relatively constant at 2-8 mmol/kg wet weight for acute mortality [53].

PAH are considered to be typical nonpolar narcotic (anesthetic) compounds. The LR50 (lethal body residue concentration for 50% mortality) values for PAH congeners (naphthalene, phenanthrene, pyrene, fluorene) at different length of exposure (10-28d) were essentially constant and averaged  $7.5 \pm 2.6 \mu\text{mol g}^{-1}$  [14]. The bioconcentration factor declined with increasing exposure concentration and was driven primarily by a lower uptake rate with increasing dose, while the elimination remained essentially constant for each compound.

Internal effect concentrations can be found to some extent in open literature, as described above in the examples of narcotics. There is also an online database available: ERED (Environmental Residue-Effects Database), which comprises a large amount of data (a total of 13981 results on September 5<sup>th</sup> 2008). The U.S. Army Corps of Engineers/U.S. Environmental Protection Agency ERED is a compilation of data, taken from the literature, where biological effects (e.g., reduced survival, growth, etc.) and tissue contaminant concentrations were simultaneously measured in the same organism. Currently, the database is limited to those instances where biological effects observed in an organism are linked to a specific contaminant within its tissues. It includes the results of 446 different species and 404 different substances based on 2180 studies. The database includes factors such as: effect class (e.g. mortality, behavior, growth, etc.), toxicity measure (e.g. ED100, NOED, LOED, etc.), the exposure route, the body part analysed and the age of the species studied. The website can be accessed at: <http://el.erd.c.usace.army.mil/ered/Index.cfm>

The US-EPA developed the PCB Residue Effects (PCBRes) Database to assist scientists and risk assessors in correlating PCB and dioxin-like compound residues with toxic effects. The database contains PCB critical residue values for fish, mammals and birds, especially as these relate to aquatic and aquatic-dependent species. The database also includes expression of critical residue values based upon PCB Aroclors and total PCB-based congener specific methods because PCBs occur as complex mixtures. Because PCB toxicity occurs via the arylhydrocarbon-receptor (AhR), PCB toxicity has also been expressed using the sum of the dioxin-like PCBs after adjustment using toxicity equivalence factors (TEF). Limited dioxin and furan compounds in single and mixture studies are also included. The database hence provides useful information on some relevant background contaminants and should be included in further research. The database is publically accessible on the site of the US-EPA: [http://www.epa.gov/med/Prods\\_Pubs/pcbres.htm](http://www.epa.gov/med/Prods_Pubs/pcbres.htm)

## 4.7 Conclusions and Recommendations

From the text above, the following is concluded:

- The CBR concept describes toxicokinetics (uptake, elimination and other processes). It has been indicated that toxicodynamics (toxicokinetics is what the organism does to a chemical and toxicodynamics is what a chemical does to the organism) should also be included in the CBR concept. Threshold tissue concentrations are required for a range of taxa to ensure that sensitive species are included.
- Online databases (ERED and PCBRes) with internal effect concentrations are available.
- The databases don't contain data for all arctic species (especially (top-)predators) nor for all substances
- Target site concentrations are considered more suitable than total external or internal concentrations.
- Model calculations based on a planktonic system showed that mortality endpoints were sufficient; using additional sub-lethal endpoints did not increase accuracy. For the Arctic environment, which has long food chains, the opposite is to be expected. Especially for (top-)predators, sub-lethal effects such as reproduction, are important endpoints to consider.
- Caution should be taken in extrapolating too readily from laboratory animals to marine birds and mammals since there are major differences in physiology between these and the marine species that ought to be taken into account.
- Target modes of action need to be identified for all substances of interest.
- Metabolites or intermediates can also exert effect on biota. Metabolism should therefore not only be considered as an elimination route, the toxicity of the metabolites should be involved as well.
- Uncertainty in effect threshold is expected to be high.

Based on the conclusions and the text above, we suggest the following:

- The CBR can be used as an internal threshold value, which is related to the target mode of action. This means that all substances that need to be modelled (both background contaminants and contaminants from the petro-industry) are to be grouped into specific target modes of action.
- Target sites should correspond with the site at which the internal concentration is calculated. If the CBR is for instance available for the liver, the bioaccumulation model needs to calculate the concentration in that specific organ. Therefore, in some cases, an extra compartment for the target organ needs to be incorporated in the bioaccumulation model.
- As there are no standardised protocols for deriving CBRs, collected data from literature need to be checked for quality on a case by case basis. In the experimental setup toxicodynamics should be involved such that the observed effect is actually the result of the measured internal concentration.
- It is anticipated that for number of species and target mode of action, no threshold value can be derived directly from literature data. Several techniques are available to extrapolate toxicity values. The uncertainty of the extrapolated value is usually unknown. It is advisable to discuss and finalise derived threshold values in a workshop with a panel of experts.
- It should be stressed that experimentally determined CBRs are prevail over extrapolated values.
- For all substances and species metabolic routes should be studied in literature. When the study indicates that metabolites or intermediates appear to be toxic, these metabolites need to be involved in risk calculations.
- An uncertainty and sensitivity analysis of the intended model will indicate which parameters contribute to which extend to the total variance in model output. This will provide a basis for further refinement of parameter estimation.

## 5 Environmental Risk

### 5.1 Introduction

Environmental risk is characterised by both the exposure level of the contaminant and the sensitivity of the ecosystem to that contaminant. For bioaccumulating substances, risk should be determined for internal exposure levels and internal effect thresholds. Chapter 3 described the options to obtain internal concentrations, chapter 4 dealt with internal effect levels, where the Critical Body Residue (CBR) is the most useful for our purpose. This chapter will discuss how both exposure and effect can be translated into 'risk'. We will start, however, with the current implementation of bioaccumulation in the EIF for produced water.

### 5.2 Current Implementation of Bioaccumulation in the EIF for Produced Water

The existing EIF for produced water implements bioaccumulating properties in a simplified manner. The intended risk assessment tool should be able to operate within the existing EIF framework that is currently used by StatoilHydro. In order to illustrate how the current EIF deals with bioaccumulation, the EIF itself needs some elaboration first.

In environmental risk assessment, a tiered approach usually is applied. In the first tier, worst case exposure levels of a contaminant are compared with worst case sensitivity of the ecosystem for the contaminant. In a second tier, statistical distributions of both exposure levels and species sensitivity are used to refine the risk assessment. In higher tier assessment, field realistic aspects, such as population dynamics and food-web interactions, are involved by performing mesocosm studies or model simulations. Most EIFs are standardised on the probabilistic risk assessment used in the second tier.

The current EIF for produced water is expressed as the water volume in which the multi-substance Potentially Affected Fraction (msPAF) is equal to or greater than 5% (see section 5.2). When a similar approach is to be developed for bioaccumulating substances, we need sufficient spatial (2D or 3D) resolution in concentration calculations (Figure 18).

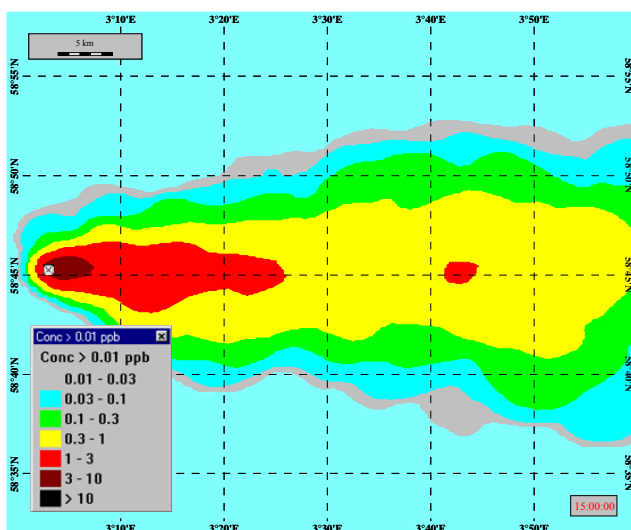


Figure 18 *The spatial distribution of concentrations as calculated by the particle tracking module in the current EIF tool for produced water*

The current EIF for produced water takes bioaccumulation of substances to some extent into account [7]. Figure 19 shows the process scheme for calculating this EIF, this approach uses the universal risk characterisation paradigm (shown in Figure 1 adjusted for bioaccumulating substances). In more detail, the EIF expresses environmental impact as follows:

- $EIF_t = \text{SUM}(\text{water volumes with msPAF} > 5\%) * 10^5$
- EIF = maximum  $EIF_t$  over 1 month
- msPAF = combined risk from concentrations of toxicants in the produced water mixture after discharge
- EIF PW = Indicator for the volume of water around a discharge point where adverse effects to biota might occur as a result of the discharge

Where risk is described by the following aspects:

- Probability = 1, emission and exposure take place
- Target = marine biota
- Effect = adverse (toxic) effects (not specified)
- Severity = percentage of species being adversely affected (PAF)
- Extend = Water volume
- Duration = Maximum value that might occur during a month of the discharge

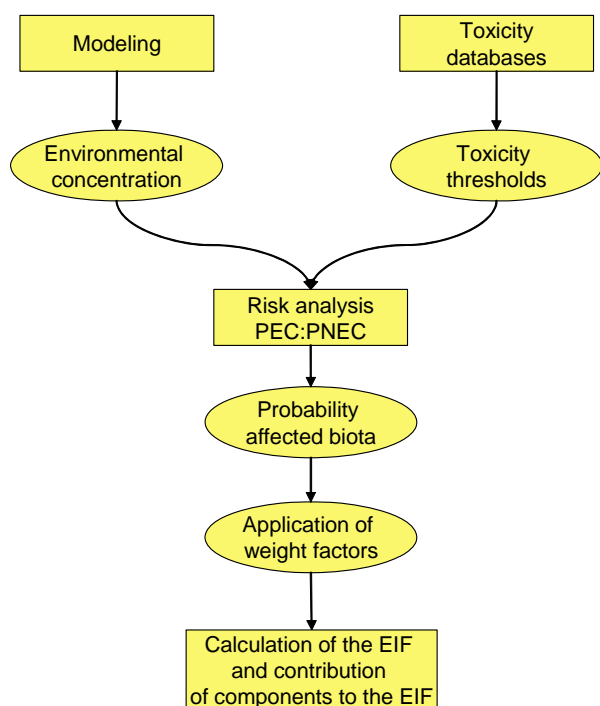


Figure 19 *Process scheme of the calculation of the EIF for produced water discharges [7].*

Figure 19 shows that a weighting factor is applied for the persisting and bioaccumulating properties of a substance. Table 8 shows the weighting factors that are used are a pragmatic way of implementing bioaccumulating substances in the risk management tool. Adjusting the EIF tool to the Arctic environment, where accumulated substances might already pose a risk, requires a more refined approach. This report aims to describe such an approach, based on available knowledge found in literature.

Table 8 *Weighting criteria and weight factors in the EIF [7]*

Biodegradation (BOD, 28 days test)	Bioaccumulation potential (Log Kow)		
	<3	3 – 5	>5
>60%	1	1	1
20 – 60%	1	2	2
<20%	2	2	4

### 5.3 Risk Based on Dynamic Exposure

The risk can be expressed based on dynamic exposure. In fact, this is the approach that has been used in the Dose Related Risk and Effects Assessment Model (DREAM) [7]. The effect is quantified by both the amount the Critical Body Residue (CBR) is exceeded and the time the threshold is exceeded (Figure 20). Although including exposure dynamics reflects the real world better, it comes with a serious down side. This methodology requires the model to incorporate routines to describe species behaviour. That is, the exposure of a species depends on its migration through the spatial concentration distribution. This approach therefore requires large amounts of data, which is generally not available. This is why the DREAM model only includes the dynamic exposure of theoretical fish larvae and was eventually abandoned for the more simplistic EIF approach. The EIF assumes a generic sensitivity of the ecosystem with no spatial distinction. A similar approach is described in the next sections and is recommended for the intended risk assessment tool.

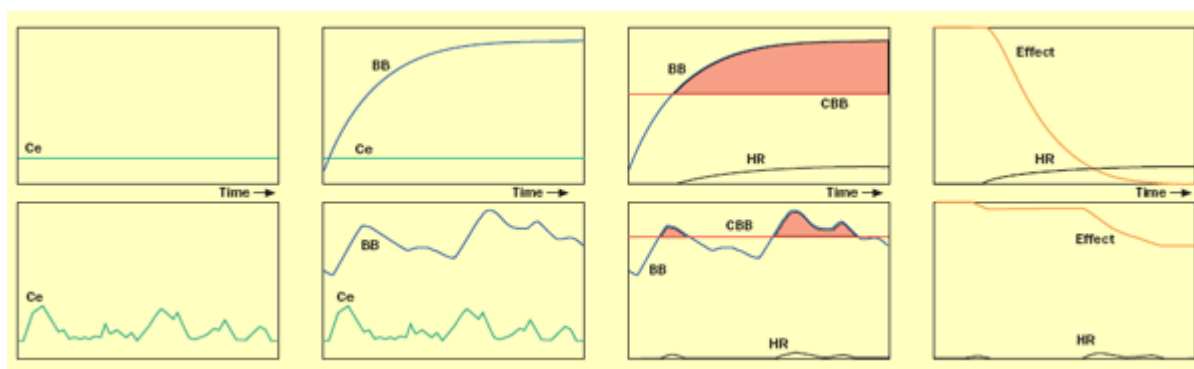


Figure 20 *Critical Body Burden (CBB = CBR) used as a threshold in a dynamic exposure situation.  $C_e$  is the external concentration, BB is the Body Burden (= internal concentration), HR is the hazard rate. The surface between the BB and CBB determine the magnitude of the effect. Top plots show an example with a constant external exposure; bottom plots show an example with a fluctuating external exposure.*

### 5.4 Risk Based on Static Thresholds

In contrast to the DREAM approach we propose to use CBRs as a static threshold. As shown in section 4.2, each species has a CBR for each target mode of actions. When the sum of concentrations of the substances with the same target mode of action exceed the corresponding CBR an effect is anticipated. In this static approach the effect is not quantified, the fact that an effect (no matter the magnitude) is expected is sufficient and considered to be a risk. Figure 21 shows the principle in a theoretical food-web.

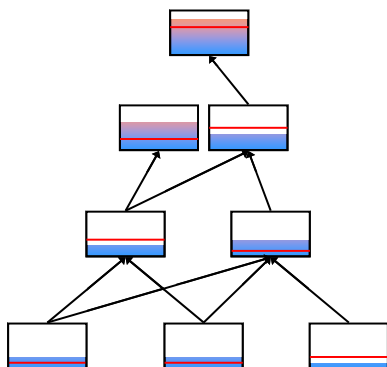


Figure 21 A theoretical food-web as an example. Boxes indicate species in the food-web, where arrows indicate the diet. Where blue areas indicate internal concentration, and red lines indicate Critical Body Residues. In this example 5 out of 8 (62.5 %) of the species are potentially affected.

Although the exceeding of an effect threshold (CBR) indicates a risk, it does not quantify the risk. There are several options to quantify risk. It can be done on a single species basis. In that case the risk characterisation ratio can be calculated by dividing the internal concentration through the CBR (much like the PEC:PNEC ratio).

The risk can also be quantified based on multiple species. The fraction of species in the food-web that are potentially affected can be determined. This will provide an indicator of risk that is very similar to the Potentially Affected Fraction (PAF) of the Species Sensitivity Distributions (SSDs). It might be desirable to determine this affected fraction per trophic level, as bioaccumulation may vary among the trophic levels.

The current EIF for produced water uses Species Sensitivity Distributions (SSDs) directly to quantify risk at a specific exposure concentration. Where a SSD is the cumulative probability that species are exposed above their No Observable Effect Concentration (NOEC). Risk is in this case expressed as the Potentially Affected Fraction of species (PAF). Although these SSDs are based on external concentrations, SSDs based on internal concentrations are also available (e.g. Figure 22). The problem with these SSDs is that they don't directly relate the risk to external concentrations in the environment. The intended model needs to work around that as this link is important to relate the risk to activities that need assessment (Figure 22). However, SSDs based on internal concentrations can still be useful in the intended tool. When a single box in Figure 21 does not represent a single species but a (functional) group of species, the internal concentration is modelled for the entire group and equal for all species in that group. The sensitivity for that group can then be expressed with an SSD containing CBRs for the species in that group.

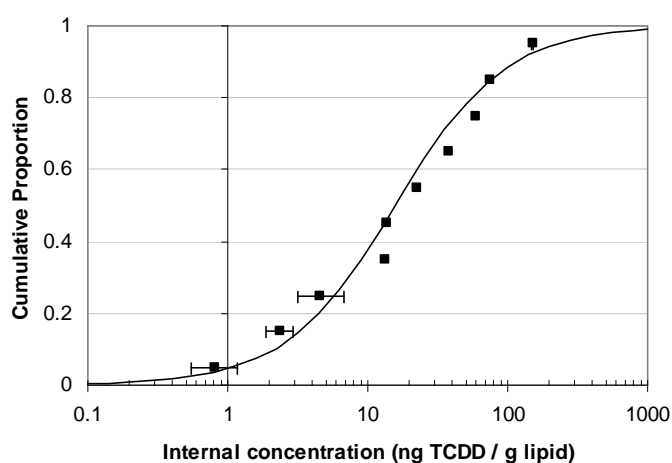


Figure 22 An example of a Species Sensitivity Distribution based on internal concentrations of TCDD based on data from Steevens et al. [58]. As there is no direct link with external concentrations in the environment, this methodology is useful in field monitoring but not for the intended model.



## 5.5 Comparing Risk with the Current EIF

The EIF defines the environmental impact as the water volume where a specific risk threshold is exceeded (section 5.2). Ecosystem sensitivity is assumed to be generic and spatially homogenous, in contrast to the spatial concentration distribution, which is not homogenous. When risk of bioaccumulated substances is to be compared with the current EIF, the impact of those substances needs to be defined in a similar fashion.

The question rises whether the ecosystem sensitivity can be expressed generic for accumulating substances. Migration and spatial distribution of species may result in effects on other locations than the external concentration plume emanating from the petro-industry activities. For instance, when a species migrate through the discharge plume, they are exposed and accumulate some of the contaminants. Effects might only take place after they migrate away from the plume. In addition, when they are preyed upon, away from the discharge plume, the contaminants can accumulate in the predator, while it was not even near the discharge plume. Originally the DREAM model implemented this to some extend as the body burden related effect assessment [7]. This approach led to sophisticated assessment which is hard to interpret and was later replaced by the EIF approach which was easier to comprehend and allowed simple comparison of scenarios for management decisions. The chosen definition for impact of accumulating substances would considerably reduce the complexity of the model, as for each spatial grid the risk is calculated for a generic (but realistic) food-web, where the food-webs in different grid cells don't interact (Figure 23).

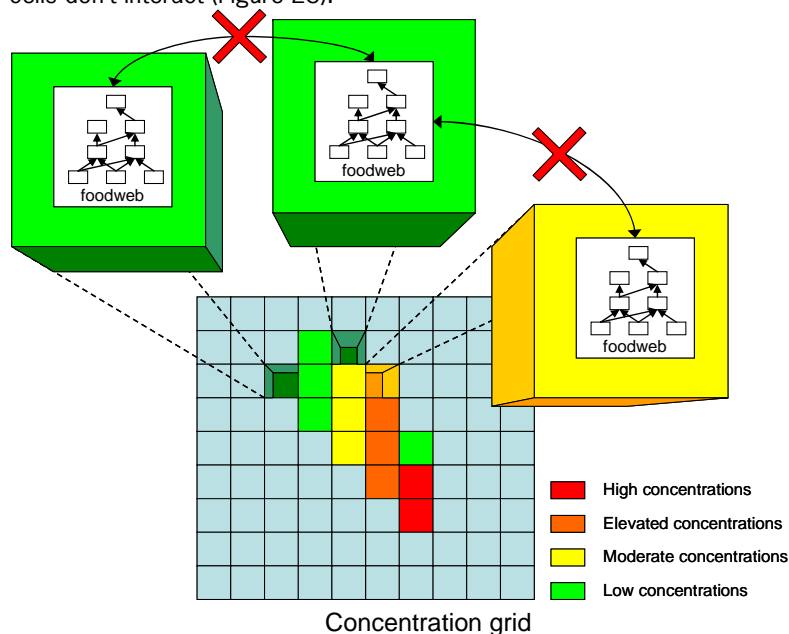


Figure 23 *Spatial distinction will be made in the intended model for external concentrations, which is also the case for the current EIF for produced water. Sensitivity of the ecosystem will be generic in the intended model as explained in the text. For each spatial grid cell a generic bioaccumulation model will be implemented without interaction of the food-webs in different grid cells.*

For a small river floodplain it has been shown that the environmental heterogeneity only contributes only to a minor part of the variation in metal exposure levels in terrestrial vertebrates [59]. Whether this also applies to large marine systems is unknown but unlikely. As the generic food-web used in the model includes all relevant species for each spatial grid cells, the method will be conservative. Each grid cell will for instance contain top-predators like polar bears, while they not necessarily occur at that location. The model will evaluate whether such top-predators (and also the other species) are potentially exposed to internal concentrations above their tolerance limit, as a result of an external concentration in that grid cell.

Using generic sensitivity of the ecosystem, impact factors similar to the current EIF can be produced.

## 5.6 Conclusions and Recommendations

From the above, the following can be concluded:

- Approaches to obtain risk estimates may vary from complex, biological modelling to relative straight forward exposure and sensitivity comparison.
- Most EIFs are standardised on the probabilistic risk assessment used in the second tier.
- An EIF-like approach requires spatial distribution of external concentrations.
- Dynamic exposure modelling requires complex behavioural aspects to be included.

From the conclusion and the text above, the following is recommended:

- Although dynamic exposure modelling is more realistic it is expected to be complex and data demanding for our purposes. It is therefore advised to express risk probabilistically: the fraction of species being potentially affected.
- Following the EIF-approach, spatial variation in risk is determined by the distribution of external concentrations, where the sensitivity of the ecosystem is described generically. The impact in this case is the water volume where external concentrations are such that the accumulation of contaminants can potentially lead to effects in more than a specified fraction of species.

## 6 Proposed Modelling Concept

### 6.1 Introduction

The previous chapters were mainly descriptive, providing background on required elements for environmental risk modelling of bioaccumulating substances. They also provided several alternatives with recommendations for each element. This chapter integrates all the aspects and shows the outline of the proposed modelling tool, thereby providing the direction for future research.

### 6.2 A Framework of the Intended Modelling Tool

Figure 24 schematically shows the intended model, with each of the four modules 'Exposure', 'Bioaccumulation', 'Effect' and 'Risk'. The diagram shows the general processes involved in the proposed modelling tool. The framework presented in Figure 24 will be discussed in this section from the start to the final expression of risk, with special attention to the numbered items in the picture.

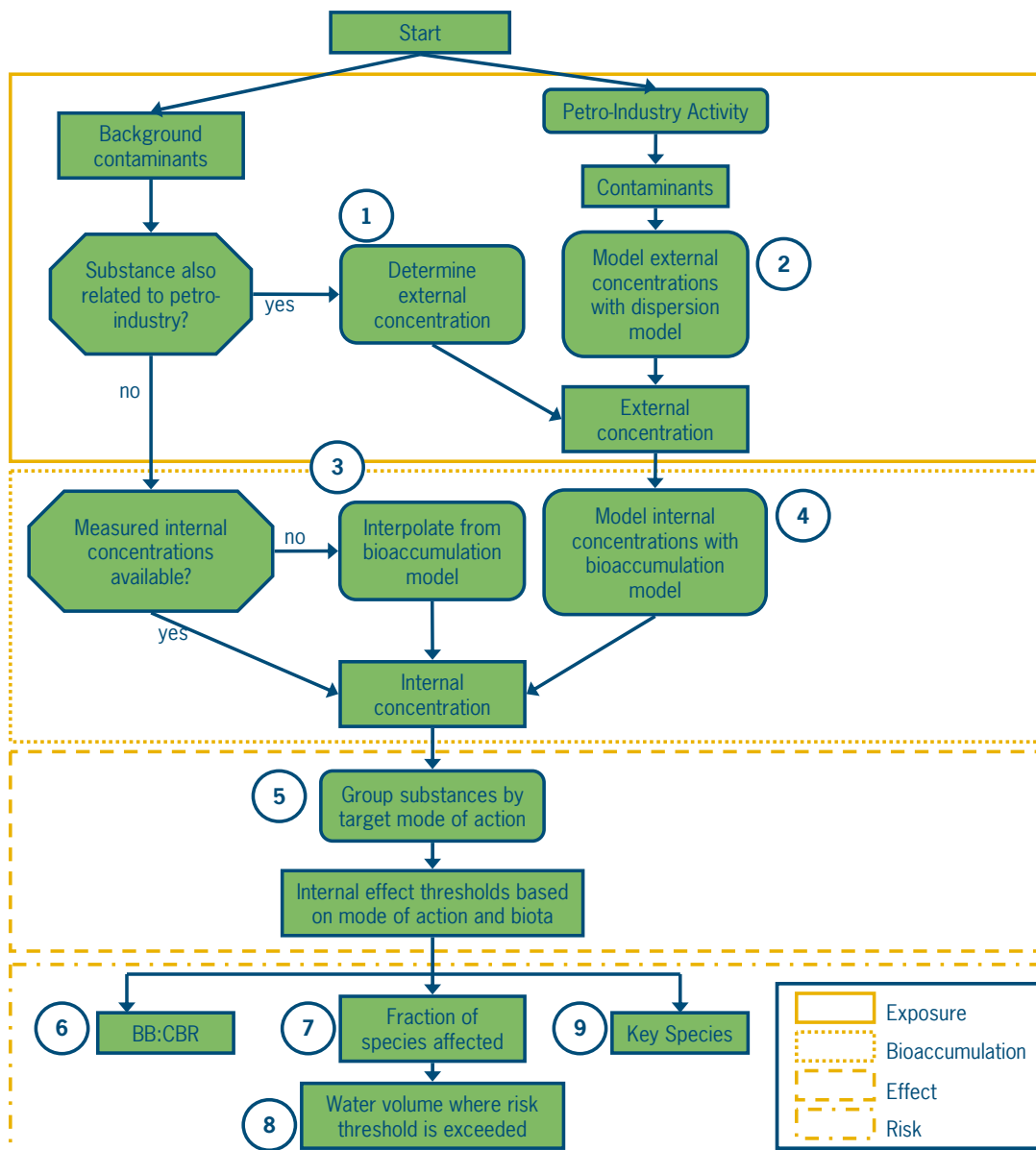


Figure 24 A schematic representation of the intended tool. Rectangles indicate a state, specific information or data. Rectangles with round corners indicate a process or activity. Decisions boxes are indicated by octagons. Large yellow boxes indicate the four modules 'Exposure', 'Bioaccumulation', 'Effect' and 'Risk'. Numbered items are described in text.

### 6.2.1 Exposure

#### (1) Determine external concentration

The framework starts with the exposure where both the background contaminants and contaminants from petro-industry activities are distinguished. Concentration for the petro-industry related substances are only available as external water concentrations, from the dispersion modelling. For background contaminants that are also part of the petro-industry related substances, we also need the external concentrations in order to determine the contribution of both. Many of the background contaminants cannot be measured in water, but are available as internal concentrations in species. In those cases, the external concentration needs to be estimated with a bioaccumulation model (see item 1 in Figure 24). The most pragmatic way of doing this would be to vary the

external concentration as input for the bioaccumulation model and see which external concentration best fits the measured internal concentrations.

#### *(2) model external concentrations*

For the external concentrations of the operation activity related substances, a dispersion model is required. This is illustrated by item 2 in Figure 24, where the dispersion modelling can be performed by existing models (e.g., the particle tracking model currently used for the EIF for produced water). It is imperative to check whether the dispersion model is suitable for bioaccumulating substances. Binding to particulate matter, for instance, is an important process for bioavailability and the exposure level for the benthos. Once the external concentrations are calculated, they should be added to the external concentrations of similar background contaminants. From these external concentrations, internal concentrations then need to be determined.

### 6.2.2 Bioaccumulation

#### *(3) Interpolate from bioaccumulation model*

For background contaminants that are no part of petro-industry related substances, measured internal concentrations are preferred over model estimates. When for specific biota in the food-web, no internal concentration is available, the value needs to be estimated with the bioaccumulation model (see item 3 in Figure 24). In this case we need internal concentrations for the substance of other species in the food-web and vary the external concentration as input for the bioaccumulation model. The missing internal concentration can then be determined from the modelling results that best fit the internal concentrations of the other species.

#### *(4) Model internal concentrations with bioaccumulation model*

The bioaccumulation model has been mentioned a few times now in this chapter. It is primarily needed to calculate internal concentration from external concentrations (see item 4 in Figure 24). To include both biomagnification (exposure from food) and bioconcentration (exposure from water) the model needs to include food-web interactions. The highest trophic level in this food-web has to be determined, using the results of a screening of the petro-industry related substances. It is anticipated that most of those substances don't accumulate in trophic levels higher than fish. If this is truly the case, higher trophic levels don't need to be incorporated in the tool, simplifying the required food-web.

To describe the food-web interactions, it is proposed to use the approach of Alonso et al. [35], where the interaction is described as feeding rates. The trophic index is also used as input in that case. This approach gives a static food-web and doesn't include seasonal variations. The static food-web therefore needs to reflect the most sensitive state in time as a worst case assumption. The kinetic model describing the assimilation and elimination routes of the substances need to be more refined, as Alonso et al. [35] only use a single parameter to describe elimination. Therefore, other models [12, 13, 48] with more refined kinetics which need to be used. Where each elimination and assimilation route needs to be described by a rate constant (see Appendix D). Although most parameters can be estimated from chemical-physical properties, experimental values are preferred. The resulting internal concentration now needs to be translated to a (potential) effect in order to estimate risk.

### 6.2.3 Effects

#### *(5) Group substances by target mode of action*

Potential effects are determined as an exceedance of the Critical Body Residue (CBR) as a static internal effect threshold. In theory, similar target modes of action have a similar CBR in specific species. This is why substances with the same mode of action need to be grouped as indicated by item 5 in Figure 24. Substances with the same target mode of action should then be compared with a single CBR. As a single CBR for a specific target mode of action is mainly a theoretical exercise, it is advisable to further validate this approach with the data collected for the proposed tool.

For all substances of interest we need to extract CBR values from literature for all species in the selected food-web. For many background contaminants (e.g., PCBs and dioxin-like substances) effect data is available in public databases (ERED and PCBRes), however it is anticipated that for many substances and species (especially (top-)predators) the required data is not available. In those cases, values can be extrapolated (e.g. from other

species), but direct experimental data would be preferable. In anyway, collected CBRs need to be assessed for quality and in the end they should be discussed and finalised in a workshop with an expert panel.

Another issue that need to be addressed is metabolism. Not only is it an elimination route, the metabolites themselves can also be toxic. Metabolite profiles should therefore be extracted from literature for all substances and biota.

#### 6.2.4 Risk

##### **(6) BB:CBR**

In the final stage of the modelling approach, the risk is characterised. This is done in a tiered framework, where in the first tier risk is characterised by the ratio between the Body Burden and the Critical Body Burden (analogue to the PEC:PNEC ratio) (item 6 in Figure 24).

##### **(7), (8) Fraction of affected species, Water volume where risk threshold is exceeded**

Using the calculated/measured internal concentrations and the CBR as a effect threshold, species that are potentially affected can be identified (Figure 21). The fraction of affected species provides a probabilistic way (second tier) of quantifying the risk to the ecosystem as indicated by item 8 in Figure 24. To express the impact to the environment in a similar as the EIF, the water volume where a preset risk threshold is exceeded needs to be calculated (item 8 in Figure 24). For this purpose, we propose to use the spatial distribution of external concentrations from the dispersion modelling and assume a generic sensitivity. Meaning that for each spatial grid cell the sensitivity of the ecosystem is equal (the entire generic food-web is modelled separately without a link each grid cell). The impact in this case is the water volume where external concentrations are such that the accumulation of contaminants can potentially lead to effects in more than a specified fraction of species.

##### **(9) Key Species**

The final tier could be relevant to study in more detail the effect on a species that were identified in the second tier as potentially affected. Such approach would include the actual geographic distribution of species, as well as specific population dynamics such as growth, reproduction, survival and length of specific life stages. Many approaches would be possible, many determined by the actual species under study. As such, this tier will not be considered relevant for the development of a (generic) risk assessment model for bioaccumulative species.

This finalises the description of the proposed modelling tool. Of course testing the model also need to be part of the development. The model should be tested with a few scenarios (both in absence and presence of background contaminants) and if possible compared to current EIF values for produced water. It is anticipated that the two will deviate as the definition of environmental impact is slightly different. A sensitivity analysis of the model should be performed in order to identify parameters that potentially need more attention. We expect the effect threshold values (CBRs) to be relatively uncertain and therefore, special attention should go them during development.

## 7 Quality Assurance

IMARES utilises an ISO 9001:2000 certified quality management system (certificate number: 08602-2004-AQ-ROT-RvA). This certificate is valid until 15 December 2009. The organisation has been certified since 27 February 2001. The certification was issued by DNV Certification B.V. The last certification inspection was held the 16-22 of May 2007. Furthermore, the chemical laboratory of the Environmental Division has NEN-AND-ISO/IEC 17025:2000 accreditation for test laboratories with number L097. This accreditation is valid until 27 March 2009 and was first issued on 27 March 1997. Accreditation was granted by the Council for Accreditation, with the last inspection being held on the 12<sup>th</sup> of June 2007.

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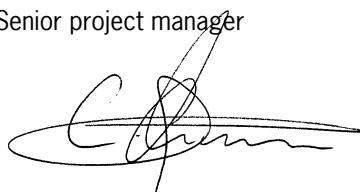
# Justification

Rapport C109a/09  
Project Number: 75003.06

The scientific quality of this report has been peer reviewed by the a colleague scientist and the head of the department of Wageningen IMARES.

Approved: C.C. Karman  
Senior project manager

Signature:



Date: 15 October 2009

Approved: Drs. J.H.M. Schobben  
Department head Environment

Signature:



Date: 15 October 2009

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Number of appendix attachments: 3

## Appendix A. Terminology and definitions

### Bioconcentration

The net result of the uptake, distribution and elimination of a substance in an organism due to waterborne exposure [27]

### Bioaccumulation

The net result of the uptake, distribution and elimination of a substance in an organism due to all exposure routes, i.e. air, water, soil and food [27]

### Biomagnification

Accumulation and transfer of chemicals via the food chain, resulting in an increase of the internal concentration in organisms at higher levels in the trophic chain [27].

### Secondary poisoning

The product of trophic transfer and toxicity [3].

Toxic effects in the higher members of the food chain, either living in the aquatic or terrestrial environment, which result from ingestion of organisms from lower trophic levels that contain accumulated substances [27].

### BCF

The ratio between the concentration in the organism and the concentration in water [27, 35].

### BAF

The ratio between the concentration in the organism and the concentration in its surrounding environment [35].

### BMF

The relative concentration in a predatory animal compared to the concentration in its prey ( $BMF = C_{predator}/C_{prey}$ ) [27].

### Body burden

The total amount of substance an animal has taken up from all sources over time and retained in the body [3].

### Critical Body Residue (CBR)

The concentration of a substance in an organism at the time of death (or any other biological endpoint) [3].

### Trophic transfer

The movement of contaminants through the food web which may result in biomagnification [3].

### Lethal Body Burden (LBB)

Measured body residue at the time of death [53].

### Mean Lethal Residue (MLR)

Average tissue residue of all dead animals from each treatment level.

### Toxicodynamics

Study of toxic actions on living systems, including the reactions with and binding to cell constituents, and the biochemical and physiological consequences of these actions.

### Toxicokinetics

Study of the kinetics of xenobiotics. It refers to the modelling and mathematical description of the time course of disposition of xenobiotics in the whole organism (absorption, distribution, metabolism, excretion).

### Tissue Residue Approach (TRA)

Associates tissue concentrations of chemicals with adverse biological effects in a dose-response fashion that can be used to determine CBRs.

## Appendix B. Literature Search

### *Search engine*

A literature review was performed to find possible approaches to apply within the intended risk management tool and to identify gaps in current knowledge. The literature was searched using the Scopus search engine ([www.scopus.com](http://www.scopus.com)). Scopus is currently the largest abstract and citation database.

### *Search restrictions*

To prevent an unmanageable database of literature, restrictions were applied to the search actions presented in Table 9:

- only publications of the year 2000 and after;
- search the terms only in the title, keywords and abstract;
- only publications in the Scopus subject area 'Environmental Science'.

### *Search terms*

The search terms used are listed in Table 9, which also shows the number of hits as a result of the search action. The search was performed on 21 May 2008.

Table 9 *Search terms and the resulting number of hits in Scopus*

<b>Search term(s)</b>	<b>Resulting number of hits in Scopus</b>
Arctic "food web" bioaccumulation	33
"background concentration" risk assessment	11
"critical body burden"	8
"critical body burdens"	2
"critical body residue"	25
"critical body residues"	13
food web accumulation model	57
food web biomagnification model	23
offshore oil gas Arctic	11
species arctic environmental risk assessment	17

### *Search results*

After the automatic search, duplicates were removed from the results. Relevant documents were then selected manually to be included in this report. Additional documents that were considered relevant by either TNO-IMARES or StatoilHydro were added to the list of literature.

## Appendix C. Parameters of Existing Bioaccumulation Models

This appendix provides an overview of model parameters for the models of Gobas et al. [48], Traas et al. [13], Hendriks et al. [12, 30] and Alonso et al. [35]. This will give an insight into the processes described by the model and thereby also the complexity of the models.

Table C1 *An overview of the most important factors used in the model of Gobas et al. [48]*

Symbol	Description	Unit	Value
BSF	Ratio between truly dissolved and total concentration in water	/	Eqn. 2 from Gobas et al. [48]
$K_{ow}$	Octanol-water partitioning	/	Experimental/Computational
$k_1$	Chemical uptake rate from water	L/(kg·d)	Eqn. 9 from Gobas et al. [48]
$k_2$	Chemical elimination rate to the water	1/d	Eqn. 13 from Gobas et al.
$k_G$	1st order growth rate constant	1/d	Eqns. 18, 19 from Gobas et al. [48]
$k_E$	Faecal elimination rate	1/d	Eqn. 17 from Gobas et al. [48]
$k_M$	Metabolic transformation rate	1/d	0, negligible when compared with other elimination routes
$k_D$	Dietary uptake rate	kg food/kg biota/d	Eqn. 14 from Gobas et al. [48]
$F_D$	Feeding rate	kg food/d	Eqn. 16 from Gobas et al. [48]
V	Weight of biota	kg	Table 1 in Gobas et al. [48]
$L_i$	Lipid content of biota	/	Table 1 in Gobas et al. [48]
$P_{i,j}$	Fraction of species j in diet of species i	/	Table 1 in Gobas et al. [48]
$C_{WD}$	Truly dissolved water concentration	µg/L	Variable
$C_{D,i}$	Internal concentration in species i	µg/kg biota	Variable
$C_p$	Truly dissolved pore water concentration	µg/L	Variable



Table C2 *Factors used in model equations of the OMEGA model of Hendriks et al. [12, 30]*

Symbol	Description	Unit	Typical value or default value
$i$	Trophic level	-	0 = abiotic, 1 = plants, $\geq 2$ = animals
$C_{0,w}$	Concentration in water	$\mu\text{g/L}$	Variable
$C_{i-1}$	Concentration in food	$\mu\text{g/kg}$	Variable, $i \geq 2$
$C_i$	Concentration in organism	$\mu\text{g/kg}$	Variable
$C_i/C_{0,w}$	Organism-water concentration ratio (BCF/BAF)	$(\mu\text{g/kg})/(\mu\text{g/L})$	Eq. 12 in Hendriks et al. [12]
$C_i/C_{i-1}$	Organism-food concentration ratio (BAF/BMF)	$(\mu\text{g/kg})/(\mu\text{g/kg})$	Eq. 12 in Hendriks et al. [12]
$\gamma_0$	Water absorption-excretion coefficient	$\text{kg}^k/\text{d}$	0.2–200 air-breathing, 200 water breathing
$\gamma_1$	Food ingestion coefficient	$\text{kg}^k/\text{d}$	0 ( $i = 1$ ), 0.005 ( $i \geq 2$ )
$\gamma_2$	Biomass (re)production coefficient	$\text{kg}^k/\text{d}$	0.0006 ( $i \geq 1$ )
$\gamma_3$	Respiration coefficient	$\text{kg}^k/\text{d}$	0.0024 ( $i \geq 1$ )
$\kappa$	Rate exponent	/	0.25
$k_0$	Water absorption–excretion rate constant	$(\text{L/kg})/\text{d}$	$k_j = q_{T:c} \cdot \gamma_j \cdot w^{\kappa}$
$k_1$	Food ingestion rate constant	$(\text{kg/kg})/\text{d}$	$k_j = q_{T:c} \cdot \gamma_j \cdot w^{\kappa}$
$k_2$	Biomass (re)production rate constant	1/d	$k_j = q_{T:c} \cdot \gamma_j \cdot w^{\kappa}$
$k_3$	Respiration rate constant	1/d	$k_j = q_{T:c} \cdot \gamma_j \cdot w^{\kappa}$
$k_4$	Mortality rate constant	1/d	$k_2$
$k_{0,x,\text{in}}$	Substance absorption rate constant	$(\text{L/kg})/\text{d}$	Eq. 5 in Hendriks et al. [12]
$k_{1,x,\text{in}}$	Substance assimilation rate constant	$(\text{L/kg})/\text{d}$	Eq. 6 in Hendriks et al. [12]
$k_{0,x,\text{out}}$	Substance excretion rate constant	1/d	Eq. 8 in Hendriks et al. [12]
$k_{1,x,\text{out}}$	Substance egestion rate constant	1/d	Eq. 9 in Hendriks et al. [12]
$k_{2,x,\text{out}}$	Substance dilution rate constant	1/d	Eq. 10 in Hendriks et al. [12]
$k_{3,x,\text{out}}$	Substance transformation rate constant	1/d	Eq. 11 in Hendriks et al. [12]
$K_{ow}$	Octanol–water partition ratio	/	Hydrophilic $< 1 <$ moderately $< 10^3 <$ very hydrophobic
$p_1$	Fraction of ingested food assimilated	$\text{kg/kg}$	0.2 (detriti-), 0.4 (herbi-), 0.8 (grani-carnivores)
$p_{1,x}$	Fraction of ingested substance assimilated	$\text{kg/kg}$	Eq. 7 in Hendriks et al. [12]
$p_{\text{CH}_2,i}$	Lipid fraction of organism (i), food ( $i \geq 1$ )		0.03 [0.02–0.04] $\cdot w^{0.04[0.01-0.07]}$
$q_{T:c}$	Temperature correction factor	$\text{kg/kg}$	1 (cold-blooded), 10 (warm-blooded)
$\rho_{\text{H}_2\text{O},j}$	Water layer diffusion resistance	$\text{d/kg}^{\kappa}$	$2.8[1.4-4.1] \times 10^{-3}$ ( $j = 0$ ), $1.1[0.0-3.9] \times 10^{-5}$ ( $j = 1$ )
$\rho_{\text{CH}_2,i}$	Lipid layer permeation resistance	$\text{d/kg}^{\kappa}$	$4.6[1.3-7.8] \times 10^{-3}$ ( $i = 1$ ), $68[30-110]$ ( $i \geq 2$ )

Symbol	Description	Unit	Typical value or default value
X	Substance		
w	Species weight	kg	$10^{-14}$ - $10^3$
<b>Inorganic substances:</b>			
$K_{sw}$	Dry solids-water partitioning ratio	( $\mu\text{g}/\text{kg dw}$ )/( $\mu\text{g}/\text{L}$ )	$10^{5.1}$ - $10^{2.3}$ (Cd), $10^{4.7}$ - $10^{3.0}$ (Cu), $10^{5.2}$ - $10^{2.2}$ (Hg), $10^{3.9}$ - $10^{2.1}$ (Ni), $10^{5.8}$ - $10^{3.3}$ (Pb), $10^{5.04}$ - $10^{2.2}$ (Zn)
$K_{tw}$	Dry tissue-water partitioning ratio	( $\mu\text{g}/\text{kg dw}$ )/( $\mu\text{g}/\text{L}$ )	$8.0 [5.3-11] \cdot 10^3$
$\kappa_p$	Lipid layer resistance exponent		0.41 [0.28-0.53]
$p_{s,i}$	Dry fraction of organism (i), food (i-1)	kg dw/kg ww	0.20 [0.15-0.26] $\cdot w^{0.03 [0.01-0.05]}$
$K_{e:d}$	Distribution of egested and digested food components		Ag, Cd, Co, Cu, Ni, Zn: 1 (i = 2) or 5 (i > 2). Pb, Hg: 5. Am, Cr: 10. nonmetals: 1
$\rho_{CH2j,in}$	Lipid layer permeation influx resistance	( $\text{d}/\text{kg}^k$ )/( $\mu\text{g} \cdot \text{kg}^{kp}$ )	0.21 [0.10-0.32] (j = 0), 0.006 (j = 1)
$\rho_{CH2,out}$	Lipid layer permeation efflux resistance	d/kg	0.30

Table C3 *Factors used in model equations of Traas et al. [13]*

Symbol	Description	Unit	Value
IEC	Internal effect concentration	$\mu\text{mol/g lipid wt}$	115 (narcotics) 32.8 (polar narcotics) 0.52 (CNS convulsants)
$fD_{\text{AE(DIET)}}$	Food absorption efficiency fraction (of diet)	/	0.2 (detr.) 0.4 (herb.) 0.8 (pred.) (or weighted over diet)
$fD_{\text{OC}}$	Organic carbon (OC) fraction in diet		Food-web-sepcific (see table below)
$fD_{\text{BIOTA}}$	Biota fraction in diet		Food-web-sepcific (see table below)
$fL_{\text{(DIET)}}$	Lipid content fraction (weighted over diet)	$\text{m}^3 \text{ lipid}/\text{m}^3 \text{ biota}$	Food-web-sepcific (see table below)
$fS$	Structural volume of species		0.02; 0.1
$fW_{\text{DIET}}$	Water volume of diet		0.2
$fX_{\text{AE}}$	Toxicant absorption efficiency	/	Eq 10 in Traas et al. [13]
$k_W$	Uptake rate from water	1/d	Eq 4 in Traas et al. [13]
$k_{\text{CL}}$	Clearance rate to water	1/d	Eq 5 in Traas et al. [13]
$k_C$	Consumption rate	1/d	Eq 6 in Traas et al. [13]
$k_G$	Growth rate	1/d	Eq 7 in Traas et al. [13]
$k_E$	Egestion rate	1/d	Eq 11 in Traas et al. [13]
$k_M$	Biotransformation rate	1/d	0, for conservative assessment
$k_{\text{RE}}$	Reproductive growth rate	1/d	Eq 8 in Traas et al. [13]
$k_F$	Uptake rate from food	1/d	Eq 9 in Traas et al. [13]
$K_{\text{OC}}$	OC–water partition coefficient	$\text{L}/\text{kg}_{\text{OC}}$	Eq 1 in Traas et al. [13]
$K_{\text{OW}}$	Octanol–water partition coefficient	$\text{m}^3 \text{ oct}/\text{m}^3 \text{ water}$	Eq 1 in Traas et al. [13]
$K_{\text{pBW}}$	Biota–water partition coefficient		Eq 3 in Traas et al. [13]
$K_{\text{pFW}}$	Feces–water partition coefficient		Eq 12 in Traas et al. [13]
$R_W$	Water layer diffusion resistance		$2.8\text{E-}3$
$R_L$	Lipid layer diffusion resistance		$4.6\text{E}3$ (algae) 68 (other organisms)
$\gamma$	Water absorption coefficient		200
$W^a$	Weight raised to the power	$\text{kg}^a$	[kg]

Table C4 *Individual weights (W), fraction of lipids (FL) and fractions in diet used in the model of Traas et al. [13]*

Parameter	Phyt	Zoo	Chiro	Tubi	Bivalves	Roach	Bream	Pike	Eel	Whitebream	Perch/ruffe
<b>W (g)</b>	1E-08	1E-04	1E-04	1E-05	0.1	20	600	1200	30	150	20
<b>FL (%)</b>	1.2	6.5	0.7	1	1.5	1.3	1.8	0.5	11.4	0.3	1.8
<b>Fraction in diet</b>											
<b>Detritus</b>	0	0	0.5	1	0.1	0	0	0	0	0	0
<b>Phyt</b>	0	1	0.5	0	0.9	0.2	0	0	0	0	0
<b>Zoo</b>	0	0	0	0	0	0.2	0.01	0	0.3	0.2	0.7
<b>Chiro</b>	0	0	0	0	0	0.2	0.7	0	0.6	0.21	0.22
<b>Tubi</b>	0	0	0	0	0	0	0.28	0	0	0	0
<b>Bivalve</b>	0	0	0	0	0	0.4	0.01	0	0.1	0.77	0
<b>Roach</b>	0	0	0	0	0	0	0	0.25	0	0	0.02
<b>Bream</b>	0	0	0	0	0	0	0	0.25	0	0	0.02
<b>Pike</b>	0	0	0	0	0	0	0	0	0	0	0
<b>Eel</b>	0	0	0	0	0	0	0	0.1	0	0	0.02
<b>Whitebream</b>	0	0	0	0	0	0	0	0.2	0	0	0.02
<b>Perch/ruffe</b>	0	0	0	0	0	0	0	0.2	0	0	0

Table C5 *An overview of the most important factors in the model of Alonso et al. [35]*

Symbol	Description	Unit	Value
$BB_{\text{organism},t}$	Body burden of biota at time t	kg/kg	Variable
$C_{\text{food}}$	Concentration in food	kg/kg	Variable
F	Feeding rate	kg food/kg organism/d	0.2 (fish) 0.15 (birds)
$k_d$	Elimination rate (all possible routes and mechanisms)	1/d	0.0095 (fish) 0.0049 (birds)
$k_a$	Absorption rate from water	(L/kg)/d	0 (absorption from water was not considered by Alonso et al. [35])
$\alpha$	Assimilation efficiency	/	0.58 (fish) 0.82 (birds)
T <sub>li</sub>	Trophic index for biota i	-	$1 + \sum (T_{lj} \times FF_{i,j})$ 0 (primary producers)
FF <sub>i,j</sub>	fraction of species j in the diet of species i	/	See Table C6

Table C6 *Trophic Indices (TIs) and fractions in diet (FF<sub>i,j</sub>) used in the model of Alonso et al. [35]*

Parameter	Copepods	Amphipods	Krill	Polar cod	Atlantic cod	Brunnich Guillemont	Black Guillemont
T <sub>I</sub>	1	1	1	2	2.25	2.2	2.9
FF <sub>i,j</sub>							
Copepods	0	0	0	0.8	0.25	0	0
Amphipods	0	0	0	0.1	0.25	0.4	0.1
Krill	0	0	0	0.1	0.25	0.4	0.1
Polar cod	0	0	0	0	0.25	0.2	0.4
Atlantic cod	0	0	0	0	0	0	0.4
Brunnich Guillemont	0	0	0	0	0	0	0
Black Guillemont	0	0	0	0	0	0	0