A literature survey on selected chemical compounds

Literature survey of polyfluorinated organic compounds, phosphor containing flame retardants, 3-nitrobenzanthrone, organic tin compounds, platinum and silver

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Summary

As a consequence of increasing concentrations of anthropogenic organic compounds in a wide range of environmental samples, the Norwegian authorities have begun to consider the need to restrict the import and use of several polyfluorinated compounds (PFC). In addition other potential environmental pollutants, such as phosphor-containing flame retardants, 3-nitrobenzanthrone, organic tin compounds, platinum and silver; have recently come into focus of the Norwegian authorities.

The Norwegian Pollution Control Authority (SFT) commissioned a literature survey of 14 compound groups, overviewing the available literature on polyfluorinated compounds, phosphor containing flame retardants, 3-nitrobenzanthrone, tin-organic compounds and the noble metals platinum and silver until December 2006. The survey provides the foundation on which decisions for the future needs for further screening will be made. Suggestions for geographical sampling locations and important sample compartments were also part of the study.

As a result of their manufacture over a period of decades, and release into the environment following production and use polyfluorinated compounds (PFCs) are now acknowledged to be widespread environmental contaminants. The unique chemical properties of PFCs make them important ingredients in numerous industrial and consumer products. PFCs repel both water and oil, and are therefore ideal chemicals for surface treatment of for example textiles. In addition to their presence in various perfluorinated products, the most important PFC perfluoroocitane sulphonate (PFOS) and perfluoro carboxylic acids (PFCAs) are also stable degradation products/metabolites of neutral PFC. These precursor compounds are more volatile and therefore more likely to undergo long-range atmospheric transport, with sufficient atmospheric lifetimes to reach remote locations, where they can break down.

Good analytical methods are available for the perfluoroalkyl sulphonates (PFS) and perfluorocarboxylacids (PFCA) for most matrices. Interlaboratory comparison showed that the comparability and sample pre-treatment and analytical determination is reasonably good for the analyses of PFCA, PFS and fluortelomer sulphonates (FTS) in biota and sediment matrices, but poor in some sewage sludge, cod liver and water samples. Blank contamination is still an issue for all PFC and should be carefully monitored.

Because of the reasonable robustness of the analytical methods, FTS, PFOS and PFOA are suggested as target compounds for screenings. Besides being very stable, bioaccumulative and final products of degradation of fluorinated precursors, perfluoroocetyl sulphonate (PFOS) and perfluoroocitanoic acid (PFOA) exhibit toxic properties. FTS are used as substitutes for
PFOS and pose emerging threats for the environment and therefore are important to monitor as well. All three compounds can be analysed by using the same analytical method and instrumentation. In future, when robust analytical methods are available, other neutral PFCs can be included in the screening.

Possible precursor compounds for PFCAs and PFOS are fluorotelomer alcohols (FTOHs). Fluorotelomer alcohols are manufactured as a raw material used in the synthesis of fluorotelomer-based surfactants and polymeric products. The manufacture of FTOHs usually results in a mixture containing six to twelve fluorinated carbon congeners, the 8:2 FTOH being the dominant one. Release of the volatile FTOH may occur all along the supply chain from production, application into consumer use and disposal.

Phosphor containing flame retardants (PFR) are not used in the same variety of applications as their brominated counter parts (BFR), but within a much more specific operational area. Because of their physical-chemical characteristics they also function as plasticisers, broadening their field of application, especially in the production of polyurethane foam. A wide range of biological effects of organophosphate esters has been reported, indicating substantial differences between the various organic phosphates. They are subject of several national risk analysis initiatives and the risk assessment by EU with finalisation 2006/2007.

Because of high production numbers we recommend the screening of tris(1-chloro-2-propyl)phosphate (TCPP) and of elevated toxicity we recommend screening of tetrakis(2-chloroethyl)-dichloroisopentyldiphosphate (V6) especially in the vicinity of PUR-foam producing and applying industry. In view of the uncertain toxicological implications and the ubiquitous distribution of PFR substances, screening of indoor air samples is suggested.

The 3-nitrobenzanthrone (3-NBA) abundantly exists in the particulate matters emitted from diesel and gasoline engines and also on the surface of airborne particulates. They are most likely formed during the combustion of fossil fuels as well as by the photoreaction of parent PAH with nitrogen oxides in ambient air. The nitro-PAH 3-nitrobenz-anthrone (3-NBA) is primarily known as a highly mutagenic substance.

Organotin compounds are amongst the most widely used organometallic compounds. Due to the widespread use considerable amounts of these compounds have entered the different ecosystems. To date, most attention has been given to tributyltin (TBT) and its degradation products in water and sediments due to TBTs toxic effect on aquatic life at low concentrations. Antifouling agents, containing TBT and triphenyltin (TPT), are not longer permitted in Norway. It appears that the tri- and tetra- substituted tin compounds are more toxic than the mono-and di- substituted compounds. The slow degradation of organotins in historically contaminated sediments poses a risk of contamination of water and biosphere due to remobilization or desorption processes. Harbours are areas where high organotin concentrations still are expected. It is recommended to keep tinorganic compounds within the already existing Norwegian monitoring programmes.

Platinum (Pt) and silver (Ag) belong to the noble metals. The toxicity of Pt compounds depends considerably on their water solubility. In its metallic state Pt can be regarded as non-toxic. However, halogenated Pt-salts are primarily known as powerful sensitisers inducing allergic responses. Vehicle traffic is the main source of contamination with platinum to the urban environment. Metallic silver and insoluble silver compounds appear to pose minimal
risk to human health. On the other hand, water-soluble silver compounds, such as AgNO₃, are well known to have anti-bacterial properties, and are increasingly used as anti-bacterial agents. Silver in its ionic form is highly toxic to aquatic animals and plants and should therefore be prioritized in screening efforts of the aquatic environment. Especially, the unknown fate of lately introduced silver-containing nano-particles is reason for concern.

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1 Introduction

As a consequence of recent studies, indicating increasing concentrations of fluorinated organic compounds in a wide range of environmental samples, the Norwegian government has begun to consider the need to restrict the import and use of several polyfluorinated compounds (PFC). Many other countries have also initiated studies to provide more information on this group of compounds regarding their significance as environmental contaminants and to assess the need for further regulatory action.

In addition other potential environmental pollutants, such as phosphor-containing flame retardants, 3-nitrobenzanthrone, organic tin compounds, platinum and silver; have come into focus of the Norwegian authorities.

This literature study, commissioned by the Norwegian Pollution Control Authority (SFT), of 14 compound groups, gives an overview of the available literature on the substances and it provides the foundation on which decisions for the future needs for further screening will be made. Suggestions for geographical sampling locations and important sample compartments are also part of the study.

This report consists of fact sheets for the most important compounds from the 14 compound groups of interest. These include:
- Perfluoroalkyl sulphonates,
- Perfluoroalkyl carboxylates,
- Fluorotelomer sulphonates,
- Fluorotelomer carboxylates (saturated and non-saturated),
- Fluorotelomer alcohols,
- Fluorotelomer olefins,
- Fluorotelomer aldehydes,
- Teflon,
- Incineration products of fluoropolymers,
- Phosphor containing flame retardants,
- 3-nitrobenzanthrone,
- Organic tin compounds,
- Platinum and silver.

The document is split into 6 chapters, I) Introduction, II) Polyfluorinated compounds including Teflon and incineration products of fluoropolymers, III) Phosphor containing flame retardants, IV) 3-nitrobenzanthrone, V) Organic tin compounds and VI) Noble metals.

The aim of this paper is to document the research undertaken on the chemicals of interest. Most of the data presented here are derived from databases provided or supported by national and international administrative institutions and literature reviews conducted with integrated web-based software (e.g., ISIWeb of Knowledge). Published data were taken into account until December 2006. The paper focuses on:
- Characteristics of the compound,
- Toxicological data,
- Degradation in the environment,
- Use in Norway
- Emissions
- Monitoring data
- Evaluation of need for screening
- Analyses
2 Polyfluorinated organic compounds (PFC)

2.1 Introduction

As a result of their manufacture over a period of decades, and release into the environment following production and use, polyfluorinated compounds (PFCs) are now acknowledged to be widespread environmental contaminants. The different toxicological, chemical and physical behaviour of PFCs, some of which are used as technical mixtures (formulations) containing a number of individual compounds, makes it difficult to fully assess their impact on humans and the environment. Currently, worldwide research is mainly focused on the perfluorinated alkyl sulphonates and carboxylates (PFS, PFCA), but studies of the more volatile compound groups, fluorotelomer alcohols (FTOH) and sulphonates (FTS), are also underway.

The unique chemical properties of PFCs make them important ingredients in numerous industrial and consumer products. PFCs repel both water and oil, and are therefore ideal chemicals for surface treatment of for example textiles. Polytetrafluoroethylene (PTFE)-based membranes are often used due to their water resistance and ability to “breathe”.

There are two main production processes for PFCs: electrochemical fluorination and telomerisation. In the electrochemical fluorination process, a technical mixture of hydrocarbons (different carbon chain lengths including branched isomers) with a functional group is subjected to fluorination, leading to a mixture of perfluorinated products with the same homologue and isomer pattern. Telomerisation involves coupling tetrafluoro-ethene, which leads to straight-chained products with an even number of carbon atoms. Fluorotelomer products often possess two carbon atoms adjacent to the functional group that are not fluorinated, but also perfluorinated compounds can be synthesised through the telomerisation process.

Since PFCs are generally persistent in the environment, bioaccumulation occurs and they have been found in mammals, birds and fish as well as humans (Giesy and Kannan, 2001; Kannan et al., 2002a; Kannan et al., 2002b; Kannan et al., 2004). To describe bioaccumulation properties, the commonly applied octanol-water-partition coefficients \( K_{ow} \), used for neutral organohalogen compounds, are not suitable in the case of PFC. As PFCs act both oleophobically and hydrophobically these models cannot be used in order to describe their fate. Both PFS and PFCA bind to the serum albumin rather than to lipids in living organisms.

In general there is a lack of data on physicochemical properties and due to the lack of agreement for measurements made by different methods, confidence in existing data is low. Data for physical-chemical properties given in this report, should therefore to be used as estimates only (Houde et al., 2006).
Barber et al. summarises the hypothesis about how are PFC transported from densely populated application areas to remote places? A likely possibility is the so-called ‘precursor’ hypothesis (Ellis, 2004): in addition to their presence in various perfluorinated products, PFOS and PFCAs are also stable degradation products/metabolites of neutral PFC. These precursor compounds are more volatile (Lei, 2004), and therefore more likely to undergo long-range atmospheric transport (LRAT), with sufficient atmospheric lifetimes to reach remote locations (Wallington, 2006), where they can break down. Possible precursor compounds for PFCAs and PFOS are fluorotelomer alcohols (FTOHs) (Ellis, 2004) and fluoro-octane sulfonamides/ethanols (FOSAs/FOSEs), (Martin, 2006; D’eon, 2006) respectively, and it has also been suggested that fluorinated telomer olefins (FTolefins) will degrade to form PFCAs (Prevedouros, 2006). However, a more recent hypothesis predicts that the atmospheric transport of precursor PFC is insignificant in comparison to direct oceanic transport, (Prevedouros, 2006). A third hypothesis is that PFOS and PFCAs may be emitted from primary sources in association with particulate matter, and be directly transported long-distances in the atmosphere attached to particles (Simcik, 2006). A fourth, as yet untested, hypothesis suggests that PFC concentrated at ocean and river surfaces may be transported into the air in the form of marine aerosols (Prevedouros, 2006). Once here, they could partition onto the surface of particles when spray droplets evaporate, and thus be transported long-distances in the atmosphere. Given the lack of consensus in the scientific community, transport pathways and environmental fate of all fluorochemicals need further investigation (Barber, 2007).

Analysis of per- and polyfluorinated alkyl compounds is a relatively new topic in the field of environmental chemistry. The special properties of PFCs make reliable measurements by trace analysis a challenge.

Recent interlaboratory studies demonstrated good reproducibility for the analysis of PFOS, PFOA and PFOSA in standards, fish and human plasma. However, reproducibility for the same compounds is poor for water as matrix (Houde et al., 2006; de Voogt, 2006). The EU project, PERFORCE (Perfluorinated Organic Compounds in the European Environment, FP6-NEST INSIGHT” from the 6. Framework Programme; Contract no. 508967) investigated several PFC in the abiotic environment of Europe (de Voogt, 2006).

Martin et al. (2004) summarized the key challenges in environmental trace analysis of the target compounds. They include blank contamination issues, purity of reference standards and matrix effects in the ionisation process of the mass spectrometer. The following general conclusions can be drawn (Powley et al. 2006; de Voogt and Sáez, 2006):

**Blank contamination** is most problematic for perfluorinated carboxylates, especially PFOA. It is associated with fluoropolymer materials used in the laboratory (e.g. PTFE) or in the analytical instrument, rather than field contamination. These materials must be avoided in sampling, storage and trace analysis of perfluorinated carboxylates. In terms of environmental matrices, the biggest challenges with blank values are encountered when analysing water. This is due to the very low levels of perfluorinated compounds in environmental water samples (low ppt to ppq) relatively to biological matrices and hence the need for a high concentration factor during sample preparation. Furthermore, water samples are usually extracted using solid phase extraction and a vacuum manifold, which commercially contains PTFE parts. Another challenge is the need of water free from the target compounds that could be used as blank control water.
Purity of reference standards used as internal standards or in external calibration solutions is still an issue. These standards may contain homologues of different chain lengths or branched isomers. Response factors of different isomers of a given compound vary greatly in MS-detection (see Martin et al. 2004; Powley et al. 2006). Commercial standards must therefore be carefully characterised before use, and uncertainties in analytical results have to be reported also considering standard purities.

Matrix effects are known to be present especially when applying weak ionization techniques, such as electrospray ionisation used in mass spectrometry of perfluorinated compounds. Furthermore, due to the amphiphilic properties of the target compounds and due to blank problems, a short and crude clean-up is usually performed, leaving many matrix compounds in the final extract. Measures have to be taken to control matrix effects in MS. For example matrix extract dissolved external calibration standards, matrix spike experiments and determination of suppression/enhancement factors, standard addition methods or the use of authentic mass labeled internal standards for all analytes of interest. The method developed by Powley proved to be virtually free from matrix effects (Powley et al. 2005).

The water method does not always perform properly for the perfluorinated sulfonates. This might be due to irreversible adsorption of these compounds to surfaces like polyethylene. However, this phenomenon is under investigation, and the water method has to be considered as still under development. For water samples with high particle content, the particle phase has to be analysed separately, due to the tendency of PFOSA and long-chain perfluorosulfonates and –carboxylates to bind to particles. As much as 20% of the extracted PFOS and 30% of the extracted PFNA from a sewage water sample were found in the particle phase. Recoveries for the water and the particle phase are 60–100% (lower for long-chain compounds) and 60–90%, respectively. Method detection limits range between 20 and 200 pg/L for the water and the particle phase, but are often elevated for dissolved PFHxA and PFOA due to blank contamination. Repeatability depends on the concentration of the analytes and the matrix in the water (purity of the water), but is usually excellent (Berger and Haukas, 2005; de Voogt, 2006b).

A promising easy to use, less time and solvent consuming method for analyzing air-samples for FTOHs with the use of commercial solid phase extraction cartridges is under development. This method is also less susceptible to blank contamination (Barber, 2007; Jahnke, 2007).

The PERFORCE consortium concluded in following remarks concerning quality assurance of PFC analyses (Berger and Haukas, 2005; de Voogt, 2006b):

- For specific matrices such as cod liver, where matrix effects were observed it should be noted that the methods are not yet sufficiently robust to provide accurate results.
- Interlaboratory comparison by co-analysis of selected samples within the consortium showed that the comparability and sample pre-treatment and analytical determination is reasonably good for the analyses of PFCA, PFS and FTS in biota and sediment matrices, but poor in some sewage sludge and water samples.
- The worldwide interlaboratory study on a fish tissue, fish liver extract and a water sample showed large variation in the between-laboratory results, showing that participating laboratories were not yet able to generate comparable results. Poor accuracy of individual laboratories is most likely caused by improper choice of (internal) standards, non-
selective extraction methods and non-selective final detection.
- QA/QC should be carefully considered when generating and interpreting the results of PFAS analyses.

More interlaboratory calibration activities are needed in order to ensure and define quality of the produced data.

The Norwegian Pollution Control Authority (SFT) commissioned a study investigating the use of PFCs in Norway in 2002. According to the report, PFCs are not produced in Norway although they are imported, either as chemical products or constituents in manufactured products. Approximately 20 t PFCs were used in Norway for a variety of purposes. Aquatic fire fighting foam was the main application, with 65% of the total use, followed by textile coating at 30% (SFT, 2004).

Perfluorinated compounds have a very unique chemistry and their toxicological properties are presently not well understood and although clearly the presence of different length (perfluorinated) carbon chains and functional groups are likely to influence toxicity. It is not clear at this time whether the hazard concerns of PFOS can be extrapolated to other perfluorinated compounds.

Alexander et al. (2003), performed an investigation of mortality of employees in a PFOS manufacturing facility. They found a small increase in mortality from bladder cancer among workers with a high exposure to PFOS. However, one could not rule out the possibility of coincidence and it is therefore difficult to make certain conclusions about the findings.

Different PFCs are considered inert and there is so far no evidence for PFCs to be chemical carcinogens or mutagens. Concerns have arisen from their similarities to cellular phospholipids, with a long hydrophobic tail and a hydrophilic head moiety making it likely that they may affect cellular lipid homeostasis. Further more, it is likely that PFCs may affect cellular membrane properties, which for example may have consequences for the distribution of oxygen in lung cells, or disrupt inter- or intracellular communication (Hu et al., 2002; Hu et al., 2003).
2.2 Perfluoroalkyl sulphonates (PFS)

The technical formulations and composition are often not accessible to the public. The congeners addressed in this literature study, are listed below.

<table>
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<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
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<td>PFBS</td>
<td>Perfluorobutane sulphonate</td>
<td>375-73-5</td>
</tr>
<tr>
<td>PFHxS</td>
<td>Perfluorohexane sulphonate</td>
<td>432-50-7</td>
</tr>
<tr>
<td>PFOS</td>
<td>Perfluorooctane sulphonate</td>
<td>1763-23-1</td>
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The general chemical structure of PFS contains a perfluorinated carbon chain connected to a sulphonate group. The length of the carbon chain determines the nomenclature of the alkyl sulphonate.

Information on the toxicity of PFC is highly varied. The most studied PFC is perfluorooctanesulphonic acid (PFOS). Less is known about the toxicity of shorter chain PFCs, but in general they can be regarded to have similar mechanisms of toxicity, but are less toxic and bioavailable.
2.2.1 Perfluorobutane sulphonate (PFBS)

The 3M Company replaced PFOS with PFBS in their Scotchgard brand in June 2003 (Poulsen et al., 2005). Since then PFBS has been increasingly used as PFOS-substitute.

![Chemical structure of perfluorobutane sulphonate.](image)

In addition to intentional production, PFBS is shown to be a degradation product of anthropogenic N-methyl perfluorooctane sulphonamidoethanol (NMeFOSE) in the atmosphere (D’Eon et al., 2006).

**Characteristics of the compound**
- Molecular formula: C₄F₉SO₃⁻
- Melting point: not applicable
- Vapour pressure: 0.29 mm Hg at 20°C
- Water solubility: dispersable in all proportions
- Log K<sub>ow</sub>: not applicable

**Toxicological data**
- LC₅₀: 96-hr fathead minnow > 1000 mg/L ([www.m3.com](http://www.m3.com))
- LC₅₀: 96-hr fathead minnow 1938mg/kg (NICNAS, 2005)
- LD₅₀ (oral; rats) > 2000 mg/kg ([www.m3.com](http://www.m3.com))

A thorough assessment of potassium perfluorobutane sulphonate was performed by the Australian authorities (NICNAS, 2005) who showed that PFBS has a low toxicity. No lethal concentrations were assigned and acute lethal concentration was higher than 2000 mg/kg in rats. There was no indication that PFBS was either as a developmental toxin or toxic to reproduction, fertility or lactation. It was, however, found to be an eye irritant.

In a 28-day oral study on rats a significant increase in liver and kidney weight was observed in animals receiving 900 mg/kg/day. In a 90 days study on rats a NOAEL was assigned to 200 mg/kg/day based on microscopic changes in the stomach. In developmental study on rats a NOEL of 300 mg/kg/day was indicated based on reduced maternal body-weight gain. Some reductions in fetal body weight were observed in 1000 mg/kg/day treatment groups. PFBS is rapidly excreted by the kidneys in cynomologus monkeys. Approximately 34-87% of the dose, administered intravenously, was recovered from the urine within 24 hours. It was shown that PFBS tended to be highly bound to human albumin.

In addition, a range of tests has been performed on birds, aquatic invertebrates and fish showing that PFBS is non-toxic. A LD₅₀ of 1938 mg/L was evaluated on fathead minnow (NICNAS, 2005).

**Degradation in the environment**
PFBS is considered as stable in the environment; PFBS is one of the degradation products of N-methyl perfluorobutane sulphonamidoethanol in the atmosphere (D’Eon et al., 2006).

**Use in Norway**
No known use.

**Emissions**
Possible from consumer products.

**Monitoring data**
No data available.

**Evaluation of need for screening**
Considered as high, due to an increasing use as a substitute for PFOS. There is relatively little known about the
toxicological effects and environmental fate of PFBS. We recommend to analyse for the shorter chain PFS in the same cases when PFOS analyses are considered (see PFOS chapter).

**Analyses**

The analyses of PFBS can be carried out with higher PFS and PFCAs. Different methods can be applied:

a) Extraction with water/methanol; HPLC/ESI-ToF-HRMS.
b) Ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.
c) Extraction with ethylacetate; treatment with ENVICarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley (2005), recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005). The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005).
2.2.2 Perfluorohexane sulphonate (PFHxS)

![Chemical structure of perfluorohexane sulphonate (PFHxS).](image)

**Characteristics of the compound**
- Molecular formula: C₆F₁₃SO₃
- Melting point: not applicable
- Vapour pressure: no data
- Water solubility: dispersable in all proportions
- Log K_{ow}: not applicable

**Toxicological data**

No information on LC₅₀ or LD₅₀ was found.

PFHxS was found to inhibit gap junctional intercellular communication (GJIC) in a dose-dependent fashion, and this inhibition occurred rapidly and was reversible. Indications show that the inhibitory effect is determined by the length of fluorinated tail and not by the nature of the functional group (Hu et al., 2002; Verreault et al., 2005).

**Degradation in the environment**

PFHxS is considered as stable in the environment and is regarded as degradation product of other perfluorinated compounds.

**Use in Norway**

No known use.

**Emissions**

Possible from consumer products (Kubwabo et al., 2005).

**Monitoring data**

PFHxS was detected with a range of 2-4300 ng/g in dust samples from Canada (Kubwabo et al., 2005) as well as a median of 2 ng/mL and 6 ng/mL in human plasma (Olsen et al., 2005; Karrman et al., 2006). No substantial difference was found in levels of PFSs between the urban and rural regions (Karrman et al., 2006). In the marine ecosystem PFHxS was found in fish from Japan and sediments collected from shallow water (Taniyasu et al., 2003; Nakata et al., 2006). Verreault et al. detected up to 2.7 ng/g ww PFHxS in plasma of glaucous gull from the Norwegian Arctic (Verreault et al., 2005).

**Evaluation of need for screening**

Considered high, as it is a possible degradation product of other polyfluorinated compounds. We recommend to analyse for the shorter chain PFS in the same cases when PFOS analyses are considered (see PFOS chapter).

**Analyses**

The analyses of PFHxS can be carried out with higher PFS and PFCAs. Different methods can be applied:

a) extraction with water/methanol; HPLC/ESI-ToF-HRMS.

b) ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.

c) extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley (2005), recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005).
The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005).
2.2.3 Perfluorooctane sulphonate (PFOS)

PFOS is considered as the most important PFC because of its intentional industrial production and global distribution. PFOS and its homologues are used commercially for numerous applications. However, its potential toxicity, extreme persistence and accumulation potential have resulted in PFOS-containing products being prohibited for new use or importation by chemical regulatory authorities in the US and elsewhere. 3M, the major manufacturing company of PFOS, voluntarily began phase out of the PFOS chemistry in 2001 (3M, 2000; U.S. Environmental Protection Agency, 2001).

PFOS was still manufactured in Germany (20–60 tonnes) and Italy (< 22 tonnes) in 2003. The total global production volume today is not known but was estimated to 5,000 tonnes per year in 2000. This was followed by a considerable decrease in the recent years due to phasing-out (3M, 2000; Poulsen et al., 2005; Houde et al., 2006). An Environmental Risk Assessment on PFOS was performed by the UK Environment Agency in the context of the EU Existing Chemicals Legislation (Brooke et al., 2004).


Due to the persistent nature of PFOS, there is a need to take into account the total accumulated production of PFOS since the middle of the 20th century, as well as the different uses in the past. (“Exploration of Management Options for PFOS” The fifth meeting of the LRTAP Task Force on Persistent Organic Pollutants, Proposal submitted by Sweden; Tallinn, 29 May-1 June 2006).
the experiments, although the animals showed symptoms of toxicity, indicating a critical exposure dose (OECD, 2006). In a study by Seacat et al. (2002) Cynomolgus monkeys were exposed to 0.03-0.75 mg/kg/day for 182 days. Adverse effects were only observed in the high exposure group of which two animals died. The most profound findings were lower serum cholesterol levels, lower triiodothyronine levels (without evidence for hypothyroidism), and lower estradiol levels. The monkeys also experienced decreased body weight and increased liver weights. The authors suggested a no-observed-effect-level (NOAEL) of 0.15 mg/kg/day (Seacat et al., 2002; Seacat et al., 2003).

The most profound effect of PFOS in rodent studies is as peroxisome proliferators. Characteristics for peroxisome proliferators are hepatomegaly, proliferation of smooth endoplasmatic reticulum and peroxisomes in association of enzyme induction, and inhibition of mitochondrial beta-oxidation. Biochemical characteristics are decrease in serum lipids, such as triglycerides and cholesterol and induction of CYP4A. Isseman and Green (1990) identified a receptor, which was activated by peroxisome proliferators. This receptor is known as the peroxisome profilator activated receptor (PPAR). This receptor belongs to the steroid/thyroid/retinoid superfamily of nuclear receptors, and is involved in the regulation of carbohydrate and lipid-metabolism as well as in and cell-regulation (Issemann and Green, 1990; Suga, 2004). Endogenous ligands for PPAR are polyunsaturated fatty acid. There are 3 isoforms of the receptor, PPAR-α, -β, -γ, which are coded by 3 different genes. PFOS is known to be a PPAR-α agonist. PARP inducers are recognized as non-genotoxic carcinogens, or tumour promoters. Only a few studies have evaluated the carcinogenic potential of PFOS.

In a study performed by 3M, refereed in an OECD-report, male and female rats were exposed to PFOS in diet for 104 weeks (0.5 ppm-20 ppm). The study showed that PFOS induced a small increase in the incident of tumours in liver, and thyroid and mammary glands (OECD, 2006). The NOAEL for male and female was considered to be 0.5 ppm and 2 ppm in diet respectively, which corresponds to approximately 0.03 mg/kg/day and 0.15 mg/kg/day. In this study and in a work by Seacat et al. (2002), there was no evidence for hepatocellular peroxisomal or cellular proliferation, measured as hepatic palmiotyl-CoA activation, at the doses tested. However, similar to the monkey study the animals had increased liver weight and decreased serum cholesterol, which is indicative of PFOS induced alterations in protein synthesis and/or lipid metabolism.

The findings that is a species difference in hepatic response to PPAR-inducers, of which rodents are especially sensitive, raises the question if this mechanism of action is relevant to human exposure (Suga, 2004).

It was recently discovered that PFOS induced a high mortality among developmentally exposed rodents (Lau et al., 2004; OECD, 2006). Pregnant Sprague-Dawley rats and CD-1 mice were given 1-20 mg/kg/day from gestation day (GD) 2 to GD 20 and GD 1 to GD 17 respectively. The major findings on the mothers were a reduction in serum thyroxine (T4) and triiodothyronine (T3), without effects on thyroide-stimulating hormone (TSH). Maternal rats exposed to high dosages (>5 mg/kg/day) experienced a reduction in serum triglycerides and cholesterol. The mice dam experienced a reduction in serum triglycerides and an elevation in liver weight at a dose of 1 mg/kg/day. The most pronounced effects were seen on the newborn rodents. At high doses (10 mg/kg/day) an increase in the prevalence of birth
defects, such as cleft palate, anasarca, ventricular septal defects and enlargement of the right atrium, occurred (Lau et al., 2003). Of more concern was the observation that 50% of the newborn rats and mice died within 24 hours when prenatally exposed to 3 mg/kg/day and 10 mg/kg/day respectively. In a more detailed study by Luebker et al. (2005), it was shown that maternal exposure up to 1.6 mg/kg/day was a critical dose leading to approximately 50% mortality among prenatally exposed pups within 4 days after delivery. No long-term permanent effects were observed in pups, which survive the first 4 days after delivery (Lau et al., 2004; Luebker et al., 2005). To indicate the most critical period in gestation Grasty et al. (2005) exposed pregnant rats at certain time intervals of 4 days to 25 mg/kg/day. Mortality of offspring was observed independently of exposure period, but was highest when the dams were exposed late in gestation (Lau et al., 2003; Grasty et al., 2005; Grasty et al., 2006).

The mechanisms for the high mortality of pups are not elucidated, and appear unclear. Luebker et al. (2005) could not provide evidence that the high mortality was due to the lipid status, utilisation of glucose or thyroid hormones. The most plausible hypothesis is a PFOS induced effect on the lungs of the neonates. Grasty et al. (2005) showed that exposed neonates had morphological changes in lungs that were indicative of immaturity. However, by co-exposure of protective agents and a more detailed investigation of the pulmonary surfactant profile failed to make certain conclusions. Since Grasty et al. (2005) achieved mortality even when dams were exposed only twice at gestation day 19 and 20 it is reasonable to believe that PFOS may influence the surface properties of the lungs making them less efficient to absorb oxygen. Other possible mechanisms of PFOS toxicity are the inhibition of gap junctional intercellular communication (Hu et al., 2002), disruption of calcium homeostasis by changing membrane surface properties (Harada et al., 2005a) and its PPAR activating properties which may influence several processes in cells, as stated above, but also induce oxidative stress and mitochondrial dysfunction.

The toxicologists working on the EU project “PERFORCE” found that PFOS primarily downregulated gene expression in pathways related to cholesterol and steroid biosynthesis whereas functions related to protein kinase regulator activity were upregulated. With regard to the bacterial gene expression profile after exposure to PFOS, different stress genes are significantly induced. This suggests that PFOS is targeting the membrane, causing oxidative damage and resulting in interference with DNA metabolism. The membrane related stress is most probably a direct consequence of the detergent like nature of the compound (de Voogt, 2006).

In Norway, Bioforsk conducted eco-toxicological tests in earthworms (*Eisenia fetida*) with PFOS, PFOA and 6:2 fluorotelomer sulfonate. Reproduction studies were performed for the three compounds in agreement with OECD guideline 222. Results indicated that PFOS is harmful to earthworm reproduction when the soil concentration levels exceeded 10. Observed effects were reduced number of cocoons, reduced hatchability, and reduced number and weight of juveniles. The soil-to-earthworm bioconcentration factor (BCF) was 2.3 for PFOS. The chemical level in earthworms was more than doubled compared to the environment. This indicates that the bioconcentration of PFOS already starts at a low level of the food chain (SFT, 2007).

**Degradation in the environment**

Since several identified and unidentified precursors can degrade to PFOS, they will all contribute to the environmental load for PFOS. PFOS has been classified as a persistent, bioaccumulative and toxic,
PFOS is a degradation product of f.ex. neutral perfluorinated compounds as N-methyl perfluorooctane sulphonamide and N-ethyl perfluorooctane sulphonamide (Tomy et al., 2004).

**Use in Norway**

In a recently published report from the Norwegian Pollution Control Authority (SFT) an inventory was made of the remaining quantities and historic emissions of fire fighting foams still containing PFOS in Norway. The quantities of PFOS were estimated to approximately 22 tonnes and the dominant use was in offshore installations. Historic emissions of PFOS and related compounds were estimated to a minimum of 58 tonnes, with offshore platforms as the main contributor (90%) (Kartlegging av PFOS i brannskum, SFT report, TA-2139/2005, ISBN 82-7655-275-7, (Summary available in English), http://www.sft.no/publikasjoner/kjemikalier/2139/ta2139.pdf).

The Norwegian Ministry of Environment is preparing to ban PFOS in fire fighting foam, textiles and waterproofing agents.

**Emissions**

It is not possible to provide detailed emission scenarios for all PFOS potential precursors as much information is not available;

Releases of PFOS and its related substances are likely to occur during the whole life cycle of the product containing them. They can be released during production, at assembly, during distribution and disposal, from landfills and waste incineration.

Emissions from manufacturing operations prior to the PFOS phase-out were primarily in the form of waste process water discharged to industrial or municipal treatment facilities.

Paper recycling facilities may continue to be a source of PFOS emissions as there may also be releases of PFOS during the recycling process (Moriwaki et al., 2003).

The use of fire fighting foam containing PFOS on offshore oil platforms can be a direct water pollution route.

**Monitoring data**

More than 200 publications were available in November 2006 describing the fate, bioaccumulation, distribution and transport of PFOS. PFOS is the predominant PFC-compound detected in biota. In order to assess the amount of accumulated information several overviews of the levels and trends of PFC in environmental samples, including those in Europe, published 2005/2006, were used as a starting point (3M, 2000; Poulsen et al., 2005; Loewen et al., 2005; Houde et al., 2006; Prevedouros et al., 2006; de Voogt and Saez, 2006).

**Long-range-transport (LRT):**

PFOS is considered as not easy accessible for LRT because of its chemical-physical properties. However, findings in Arctic air as well as Arctic biota lead to the hypothesis that volatile precursors are transported to the Arctic, followed by degradation to the stable product PFOS. Transport by ocean currents may lead to elevated concentrations in the Arctic as well (Jahnke et al., 2007b; Yamashita et al., 2005; Prevedouros et al., 2006).

**Air:**

Air is not an important sink for PFOS because of its very low volatility. However, there is a high tendency for PFOS to be absorbed on particulate material can lead to elevated concentrations in airborne dust. PFOS was detected in dust samples...
of Japanese homes (11-2500 ng/g) (Moriwaki et al., 2003).

PFOS was also present in the particular phase of air samples from the UK and Japan, suggesting potential air transport via particles (Harada et al., 2005b; Harada et al., 2006; de Voogt, 2006).

Water:
Ground water around fire-training areas has been proven to contain elevated levels of PFOS (Moody et al., 2003). Surface water in Japan, and Canada contained PFOS at levels between 2-150 ng/L and in the Pacific Ocean at low levels (Yamashita et al., 2005). During PERFORCE PFOS was detected in several rivers samples in the Netherlands up to 56 ng/L (de Voogt, 2006). In a Norwegian study of PFC in the Norwegian environment PFOS was found in elevated concentrations in cleaned landfill effluents and sediments. In the aquatic environment, PFOS was also found in freshwater samples and sediments (SFT, 2005b). These concentrations have to be considered low and primarily caused by long-range-transport. In fish samples from the same freshwater locations PFOS dominated the PFC pattern with up to 4 ng/g ww (SFT, 2005b).

Sediments:
The recent Nordic screening project found PFOS as the dominating fluorinated substance with e.g. 1020 pg/g ww in Norwegian samples (Kallenborn et al., 2006; Prevedouros et al., 2006). A Norwegian screening found PFOS in all marine sediment samples at concentrations between 170 and 5900 pg/g dry weight (SFT, 2005b).

PERFORCE detected 1.5 ng/g dw PFOS in sediment samples from the Western Scheldt, Netherlands (de Voogt, 2006). PFOS was detected in sediments in the USA together with substances that may be transformed to PFOS, such as 2-(N-ethylperfluorooctanesulphonamido) acetic acid (N-EtFOSAA) and 2-(N-methylperfluorooctanesulphonamido) acetic acid (N-MeFOSAA). Both were present in sediments and sludge at levels often exceeding PFOS (Higgins et al., 2005).

Biota:
The global contamination of PFOS in wildlife was reported by Giesy et al. (2001) and has since then been further examined in numerous international studies (Key et al., 1997; Giesy and Kannan, 2001; Kannan et al., 2002b; Kannan et al., 2005; Houde et al., 2006). In both aquatic and terrestrial organisms PFOS was found over a broad concentration range and within the most parts of the food web. For high levels of PFOS in the aquatic food web, sediments are suspected as main source, mediated through accumulation of PFOS precursors in the sediment (Martin et al., 2004b; Higgins et al., 2005). Elevated concentrations of >100 ng/g ww in fish were detected at different locations (Taniyasu et al., 2003; Houde et al., 2006). Discharge from municipal wastewater, fire fighting operations and landfill leakage may be responsible for elevated levels in urban areas (Taniyasu et al., 2003).

Samples of marine biota analysed under the Norwegian screening in 2004, showed PFOS contamination in all blue mussel and cod liver samples, with concentrations up to 0.2 ng/g ww and 6.3 ng/g ww respectively (SFT, 2005b). In a study of PFC in Northern Fulmars from Bjørnøya, PFOS was detected in median concentrations of 3.4 ng/g ww which is 30 times lower compared to PFOS concentrations found by Verreault et al. (2005) in liver samples of glaucous gull from the same region (Gabrielsen et al., 2005; Verreault et al., 2005; SFT, 2005b).

Similar to fish, PFOS levels in birds and mammals living close to industrialized areas are higher compared to rural locations. Highest observed levels are
between 800 and 1200 ng/g ww PFOS (Houde et al., 2006).

Only a few studies have investigated the PFOS contamination of terrestrial mammals. However, results indicate that PFOS is readily bioavailable (Giesy and Kannan, 2001; Hoff et al., 2004).

Marine mammals have been studied quite thoroughly during recent years. Harbour seals from the Dutch Wadden Sea showed a concentration order in the tissues analysed as: kidney > spleen > liver > blubber > skeletal muscle (Van de Vijver et al., 2005). The highest PFOS levels were observed in polar bears (> 3000 ng/g ww), harbour seals (2700 ng/g ww) and bottlenose dolphins (1400 ng/g ww) (Houde et al., 2006). Smithwick et al. (2006) found evidence for an exponential increase of PFC concentrations in polar bears between 1972 and 2002 at two different Canadian locations. Doubling times of 13.1 +/- 4.0 years for PFOS were calculated from the data.

**Human Exposure:**

Since PFOS is ubiquitous in freshwater and saltwater fish, humans are readily exposed via food intake (Houde et al., 2006). Consequently, PFOS has been detected globally in human samples. However, higher PFOS concentrations were measured in blood and serum from North Americans compared to people from Asia, Europe and the Southern Hemisphere indicating that marine food may not be the main source of PFOS contamination in humans (Houde et al., 2006).

Personal care products, cleaning detergents in addition to indoor dust may be other exposure routes (Moriwaki et al., 2003; Harada et al., 2005a; Calafat et al., 2006a; Calafat et al., 2006b). PFOS-precursors such as PFOSA, can potentially migrate from food packaging. Recent studies indicate that men have a higher exposure to PFOS compared to women, and findings of PFOS in umbilical cord blood also suggests that human foetuses are exposed (Calafat et al., 2006a; Calafat et al., 2006b).

A study comparing blood samples from Norwegian and Russian people found PFOS in the majority of all samples. The median concentration varied between 3.7 and 1.6 ng/g plasma in the Norwegian and Russian samples respectively (SFT, 2005a).

**Time trends:**

Levels of PFOS have been shown to increase over time in different studies until the end of 1990s reaching a plateau after 2000 in parallel to the decrease of production in the numbers of PFCs (Holmstrom et al., 2005; Bossi et al., 2005; Smithwick et al., 2006; Calafat et al., 2006a; Calafat et al., 2006b). Both the direct uptake of PFOS or subsequent metabolisation of precursors are possible uptake routes. Since PFOS is extremely persistent and distributed globally both indoors as well as in the environment, exposure will continue for a long time to a certain degree.

**Evaluation of need for screening**

Considered as high, because of persistent, ubiquitous and toxic characteristics.

We recommend measuring PFOS in sediments from STPs, wastewater from textile and paper industry as well as organisms living near PFOS-consuming industry (fish, birds etc.).

The lack of data concerning terrestrial abundance and transport of PFOS leads to a recommendation of a screening of terrestrial biota along a north-south trajectory (birds, rodents etc.).

Human exposure, both occupational and background, should be monitored on a regular basis by analyses of human plasma.
Longer chained PFS (C9-C15) are relevant as well for human exposure in terms of toxic characteristics, accumulation potential and recent findings.

**Analyses**

The analyses of PFOS can be carried out with higher PFS and PFCAs. Different methods can be applied:

a) extraction with water/methanol; HPLC/ESI-ToF-HRMS.

b) ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.

c) extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley (2005) recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005). The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005; de Voogt and Saez, 2006).
2.3 Perfluoroalkyl carboxylates (PFCA)

The technical formulations and composition are often not accessible to the public. The main congeners found in the environment however are listed below.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFB</td>
<td>Perfluorobutanoate</td>
<td>375-22-4</td>
</tr>
<tr>
<td>PFO</td>
<td>Perfluorooctanoate</td>
<td>335-67-1</td>
</tr>
<tr>
<td>PFN</td>
<td>Perfluorononanoate</td>
<td>375-95-1</td>
</tr>
<tr>
<td>PFUn</td>
<td>Perfluoroundecanoate</td>
<td>2058-94-8</td>
</tr>
</tbody>
</table>

The general chemical structure of PFCA contains a perfluorinated carbon chain connected to a carboxilates group (RCO2⁻). The length of the carbon chain determines the nomenclature of the alkyl sulphonate.

![Figure 5: Perfluorooctane carboxylate.](image)

Direct sources of PFCA result from their manufacture and use. PFCA have been used as processing aids in the manufacture of fluoropolymers such as Teflon since the 1950s.

They have been manufactured as salts by four distinct synthesis routes:

i) Electrochemical fluorination (ECF)

ii) Fluorotelomer iodide oxidation

iii) Fluorotelomer olefin oxidation

iv) Fluorotelomer iodide carboxylation.

Commercial PFCA products consists mainly of linear C8- and C9-PFCA. Homologues with a chain length between C4 and C13 can also be found. From 1947 until 2002, the ECF process was used to produce the majority of PFO (Prevedouros et al., 2006).

In a study of extractable PFC of waterproofed textile two patterns of PFCA, were found (Figure 6). They probably represent the two main production processes of PFCA. Figure 6A shows a “common” almost symmetric distribution of PFCA homologues around PFNA, which leads to the conclusion that this PFCA mixture origins from an electro-chemically produced PFNA.

![Figure 6A: PFCA pattern in the extract from a textile (A) (Berger and Herzke, 2006).](image)

In contrast, Figure 6B shows predominantly even carbon numbered homologues, dominated by PFOA, as could be expected from a telomerisation production of PFOA. Surprisingly, in both textile extracts PFCA up to C15 could be detected, which might point to direct sources of long-chain perfluoro-carboxylates (Berger and Herzke, 2006). So far it was hypothesised, that these must be degradation products of other long-chain fluorochemicals, such as FTOHs (Martin et al., 2004a).

![Figure 6B: PFCA pattern in the extract from a textile (B) (Berger and Herzke, 2006).](image)
Bioaccumulation of PFCAs is a function of carbon chain length. PFCAs with less than 8 carbons were shown not to bioaccumulate in fish. Studies of PFCAs in polar bears confirm this assumption as the seven carbon chained perfluoroheptanoate was absent (Smithwick et al., 2005; Smithwick et al., 2006). This knowledge has led to a shift in the production towards more short chain length perfluorinated compounds.

In the same study, Smithwick et al. (2006) found evidence for an exponential increase of PFCA concentrations in polar bears at two different Canadian locations between 1972 and 2002. Doubling times ranged from 3.6 +/- 0.9 years for perfluorononanoic acid in the eastern group to 13.1 +/- 4.0 years for PFOS in the western group.

There is limited information about toxicity of PFCA with the exception of the longer chain moieties, such as perfluoro-octanoic acid (PFOA) and perfluorodecanoic acid (PFDA). The available literature suggests that there is a common assumption that the lower chain PFCA is less toxic than the longer chain PFCA. This is based partly on the knowledge that the bioaccumulation of PFCA is a function of carbon chain length. Further, Kudo et al (2001) showed that the elimination rate of PFCA in rats, with chain length from hepta to deca, decreased as a function of chain length. In another work by Kudo et al. (2000) it was claimed that the peroxisome proliferation activity of PFCA in rats was not governed by their chain length, but the rate of elimination (Kudo et al., 2000; Kudo et al., 2001).
2.3.1 Perfluorobutanoate (PFB)

There are no known commercial manufacturers of PFB, although it is available as a research chemical. It is known as a by-product from the electrochemical fluorination processes (ECF) used to manufacture perfluorohexanoate and perfluorohexane sulphonate. There are manufactures in North America, Europe and Japan using the ECF process to manufacture perfluorinated substances (de Voogt and Saez, 2006; de Voogt, 2006).

Toxicological data
No data available.

Degradation in the environment
No information is known about the chemical or biological stability of PFB.

Use in Norway
No data available.

Emissions
No data available.

Monitoring data
No data available.

Evaluation of need for screening
PFBA should be included in screening efforts in order to monitor its appearance in the environment as a degradation product or as substitute for PFOA. We recommend analysing for the shorter chain PFCA in the same cases when PFOA analyses are considered (see PFOA chapter).

Analyses
The analyses of PFB can be carried out with higher PFS and PFCAs. Different methods can be applied:

a) extraction with water/methanol; HPLC/ESI-ToF-HRMS.

b) ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.

c) extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005; de Voogt and Saez, 2006).

If one is interested only in the PFCA, without measuring PFS, a method applying gas-chromatography/mass spectrometry can also be used (Alzaga et al., 2005).

Characteristic of the compound
- Molecular formula: F(CF$_2$)$_3$CO$_2^-$
- Melting point: no data
- Vapour pressure: no data
- Water solubility: no data
- Log $K_{ow}$: not applicable
2.3.2 **Perfluorohexanoate (PFHx)**

As with PFB there are no known commercial manufacturers of PFHx, although it is available as a research chemical. It is a known by-product from electrochemical fluorination processes (ECF) used to manufacture perfluorohexanoate and perfluorohexane sulphonate. There is one manufacturer each in North America, Europe and Japan using the ECF process to manufacture perfluorinated substances (de Voogt, 2006).

<table>
<thead>
<tr>
<th>Characteristic of the compound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula: F(CF₂)₅CO₂⁻</td>
</tr>
<tr>
<td>Melting point: no data</td>
</tr>
<tr>
<td>Vapour pressure: no data</td>
</tr>
<tr>
<td>Water solubility: no data</td>
</tr>
<tr>
<td>Log Kᵪₐₜₜ: not applicable</td>
</tr>
</tbody>
</table>

**Toxicological data**

There is a lack of toxicological data for PFHxA. However, PFHxA induces hepatomegaly, peroxisomal beta-oxidation and microsomal 1-acyl-GPC acyltransferase (Kudo et al., 2006).

**Degradation in the environment**

No data available.

**Use in Norway**

No data available.

**Emissions**

No data available.

**Monitoring data**

High water concentrations were detected in several European rivers resulting in an approximated emission of 10 t PFHxA annually (de Voogt, 2006).

**Evaluation of need for screening**

The aquatic ecosystem is suggested for screening because of the reported elevated levels in river water and the lack of data. We recommend analysing for the shorter chain PFCA in the same cases when PFOA analyses are considered (see PFOA chapter).

**Analyses**

The analyses of PFHx can be carried out with higher PFS and PFCAs. Different methods can be applied:

a) extraction with water/methanol; HPLC/ESI-ToF-HRMS.

b) ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.

c) extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley, 2005, recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005). The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005; de Voogt and Saez, 2006).

If one is interested only in the PFCA a method applying gas-chromatography/mass spectrometry can be used as well (Alzaga et al., 2005).
2.3.3 Perfluorooctanoate (PFO)

The perfluorooctanoic acid (PFOA) is the free acid of the salt perfluorooctane. It is used as processing aid in the manufacture of fluoropolymers, like PTFE (e.g. Teflon). Commonly the abbreviation PFOA describes both the perfluorooctanoic acid and its salts. The ammonium, sodium, potassium and silver salts of PFOA belong to the list of substances which are of primary interest for risk assessment by the US EPA (US EPA, 2004). Recently, a number of global companies who manufacture or use PFOA have committed to a voluntary stewardship program to reduce manufacturing emissions and product content (US EPA, 2006).

A sealed vial experiment demonstrated that perfluorooctanoic acid sublimes at room temperature (Kaiser et al., 2005).

**Toxicological data**

Mice/Rat, LD$_{50}$ PO: 400 mg/kg (Kennedy et al., 2004)

Rat LD$_{50}$ IP: 198 mg/kg (Olson and Andersen, 1983)

Guinea pig, LD$_{50}$ PO: 178 mg/kg (Kennedy et al., 2004)

Fathead minnow, LC$_{50}$ 96h: 300 mg/L, (Hekster et al., 2002)

As for PFOS, PFOA primarily accumulates in liver and plasma due to its high affinity to proteins (Martin et al., 2003a; Martin et al., 2003b; Kennedy et al., 2004). In fish PFOA does not biomagnify, but bioconcentrates by uptake through water probably across the gills (Martin et al., 2003a; Martin et al., 2003b). In humans and other animals the major pathway for exposure is probably by food or inhalation.

There is no evidence for PFOA to be chemical carcinogens or mutagens and it is unlikely that PFOA represent any significant human cancer risk (Kennedy et al., 2004; Butenhoff et al., 2004a). Biegel et al. (1995) performed a two-year feeding study in rats and found increases in liver, Leydig cell and pancreatic acinar cell tumors in PFOA treated rats. This observation was attributed to PFOA as a potent PPAR inducer. Bearing in mind that humans and primates are poor PPAR inducers it is unlikely that PFOA is carcinogenic by this pathway.

When administered repeatedly the effect is cumulative and male rats appears more susceptible than the female due to differences in the elimination rate (Kennedy et al., 2004). In feeding studies with rhesus monkeys Griffith and Long observed mortality at 100 mg/kg (2-5 weeks) and at 30 mg/kg (7-12 weeks) indicating that monkeys are more susceptible than rodents (Griffith and Long, 1980).

This was also shown in a later study by Butenhoff et al. (2002) who observed that cynomologus monkeys poorly tolerated repeated exposure of 30 mg/kg/day. The dose was adjusted to 20 mg/kg/day, but still resulted in serious weight loss and increased liver weight which was followed by serious hepatocellular necrosis. Even at the lowest dose (3 mg/kg/day) an increase in liver weight was observed, which was attributed to increased mitochondrial proliferation (Butenhoff et al., 2002). Rats also experience increased liver weight as a consequence of PFOA exposure, which is a typical effect for PPAR inducers. In a 13 week oral feeding study on rats by Perkins
et al. (2004), a no effect level of 0.06 mg/kg/day was estimated.

The most profound effects on rat liver are influences on the fatty acid levels (Olson and Andersen, 1983), reduction in serum triglycerides and cholesterol (Haughom and Spydevold, 1992) increase in mitochondrial proteins and microsomal content of Cyp 450 (Permadi et al., 1992) and rat liver triglyceride accumulation (Kudo and Kawashima, 2003). This observation was attributed to PFOA as a potent peroxisome proliferator (PPAR-inducer), which can be detected by liver enlargement. Several investigations further revealed that PFCAs with longer carbon chain length, such as the perfluorordecanoic acid, are even more potent PPAR inducers than PFOA. Some PARP inducers are recognized as non-genotoxic carcinogen or tumour promoters. Bearing in mind that human and primates are poor PPAR inducers makes it not likely to believe that PFOA is carcinogenic by this pathway (Suga, 2004).

In a study by Biegel et al. (1995) PFOA was shown to reduce serum and testicular interstitial fluid levels of testosterone and increase estradiol levels in exposed rats after peroral exposure to 25 mg/kg/day for 14 days. This effect on endocrine functions in rats led to a more thorough investigation of employees at 3M workplace, however no certain association between PFOA exposure and hormonal changes was achieved (Kennedy et al., 2004). There is, however, a slight correlation between serum PFOS/PFOA levels and an increase in serum triglyceride level, alkaline phosphatase and T3 levels (Kennedy et al., 2004; Olsen et al., 2005). Another PFOA induced effect, not related to PPAR inductions, are inhibition of gap junctional intercellular communication (Upham et al., 1998).

Bearing in mind the decreased survival of the developmentally PFOS exposed rat puppies, some concern was raised if PFOA acted in a similar way. The effect of PFOA on rodents is clearly species dependent and dependent on their ability to eliminate the compound. Pregnant CD-1 mice were exposed by gavage to PFOA daily from GD1 to GD17 (1 to 40 mg/kg/day) (Lau et al., 2006). Shortly after delivery approximately 25% of the litters in 5 mg/kg/day group died, whereas only 25% of the pups in 10 and 20 mg/kg dose groups survived. The observation was reported to be similar to the developmental effects previously observed for PFOS. A similar study was performed on rats of which only a small effect was observed on the post weaning mortality at the high exposure group (maternal exposure to 30 mg/kg/day 70 days prior to mating until weaning) (Butenhoff et al., 2004b). No effects on offspring were observed in the lower dose groups.

PFOA has low toxicity towards aquatic organisms (Hekster et al., 2002, 2003). Fish appear to be more sensitive than invertebrates and algae and approximate lethal concentration on fish (LC50, Fathead minnow) is 300-700 mg/L in 96h studies.

The overall PFOA toxic mechanisms for bacteria is rather similar to that of PFOS although other genes are affected (MicF – membrane related damage; KatG and Nfo – oxidative damage). Also here the observed membrane damage could be linked to the oleophobic properties of the chemical (de Voogt, 2006).

In Norway, Bioforsk conducted ecotoxicological tests in earthworms (*Eisenia fetida*) with PFOS, PFOA and 6:2 fluorotelomer sulfonate. Reproduction studies were performed for the three compounds in agreement with OECD guideline 222.

Results indicated that PFOA is harmful to earthworm reproduction when the soil concentration levels exceeded 16 mg/kg.
Observed effects were reduced number of cocoons, reduced hatchability, and reduced number and weight of juveniles. BCF for PFOA was 1. This means that no indications of bioconcentration of PFOA between earthworms and the environment were observed in this study (SFT, 2007).

**Degradation in the environment**

PFOA is regarded as the final degradation product of perfluorinated precursors (Mabury, 2004; US EPA, 2004; Poulsen et al., 2005; Martin et al., 2006; Andersen et al., 2006). For example, 0.6% of the sulphonamide alcohol N-EtFOSF transform to PFO in a biodegradation study (D'Eon et al., 2006; Prevedouros et al., 2006). Smog chamber/Fourier transform infrared (FTIR) techniques were used to investigate the chemical reactions taking place in 700 Torr of N₂ or air at 296 +/- 2 K. The Cl initiated oxidation of CF₃CH(OH)(2) in 700 Torr of air gave CF₃COOH in a molar yield of 101 +/- 6%. The results suggest that OH radical initiated oxidation of fluorotelomeralkohol hydrates could be a significant source of perfluorinated carboxylic acids in the environment (Andersen et al., 2006). In general, the concrete chemical reactions in the atmosphere leading from fluorinated precursors to PFCA are not quite understood.

However, Gauthier et al. (2005) photodegraded 8:2 fluorotelomer alcohol (8:2 FTOH) in aqueous hydrogen peroxide solutions, synthetic field water (SFW) systems, and Lake Ontario (Canada) water samples. It was found to undergo indirect photolysis, with the data suggesting that the hydroxyl radical was the main degradation agent and that nitrate promoted photolysis whereas dissolved organic carbon inhibited it. The half-lives of 8:2 FTOH were 0.83 +/- 0.20 h (10 mM H₂O₂), 38.0 +/- 6.0 h (100 μM H₂O₂), 30.5 +/- 8.0 to 163.1 +/- 3.0 h (SFW systems), and 93.2 +/- 10.0 h (Lake Ontario). No significant loss of the parent compound by direct photolysis could be observed. The major monitored products were the 8:2 fluorotelomer aldehyde, the 8:2 fluorotelomer acid (8:2 FTCA), and perfluoro-octanoate (PFOA); the minor monitored products were the 8:2 fluorotelomer unsaturated acid (8:2 FTUCA) and perfluorononanoate (PFNA). The intermediates, 8:2 FTCA and 8:2 FTUCA, were photodegraded to verify the degradation pathway, and a mechanism for the photolysis was proposed whereby the end products of the photolysis pathway were PFOA (major) and PFNA (minor) (Gauthier and Mabury, 2005).

Dinglasan et al. (2004) examined the aerobic biodegradation of the 8:2 telomer alcohol using a mixed microbial system. The initial measured half-life of the 8:2 FTOH was similar to 0.2 days/mg of initial biomass protein. Telomer acids and PFOA were identified as metabolites during the degradation, the unsaturated telomer acid being the predominant metabolite measured. The overall mechanism involves the oxidation of the 8:2 FTOH to the telomer acid via the transient telomer aldehyde. The telomer acid via a beta-oxidation mechanism was further transformed, leading to the unsaturated acid and ultimately producing the highly stable PFOA. Telomer alcohols were demonstrated to be potential sources of PFCAs as a consequence of biotic degradation. Biological transformation may be a major degradation pathway for fluorinated telomer alcohols in aquatic systems (Dinglasan et al., 2004).

The thermolysis of PFOA in quartz ampoules was studied by ex situ heating in the temperature range 355–385°C and produced moderate amounts of perfluoro-1-heptene and SiF₄ in addition to 1-H-perfluoroheptane (Krusic et al., 2005).

**Use in Norway**

No data available.
Emissions

Emissions of PFOA can occur during production, product use and as product impurities or degradation products. In 2000 about 20t of PFO was emitted from the largest production plant in the US. The estimated historical global emissions by industry from PFO production (1951-2004) are estimated between 400 and 700t, with the main part emitted via water (Prevedouros et al., 2006). As a result of the termination of PFO production using the ECF-based technology, global manufacturing emissions have decreased from 45t in 1999 to about 15t in 2004 and to an expected 7t in 2006 (Prevedouros et al., 2006).

An exposure assessment and risk characterisation was conducted to better understand the potential human health significance of trace levels of perfluoro-octanoate detected in certain consumer articles. While there are considerable uncertainties in the assessment, it indicates that exposures to PFO during consumer use of the articles evaluated in the study are not expected to cause adverse human health effects in infants, children, adolescents, adult residents, or professionals nor result in quantifiable levels of PFO in human serum (Washburn et al., 2005).

Sewage treatment plants (STP) are major vectors of diffuse releases of PFCs into the aquatic environment. The particulate phase of the influent contributes significantly to the overall influent concentration of PFCA. STP sludge was shown to contain high amounts of PFOA and STP can serve as point sources for PFC both for the aquatic ecosystem (effluent discharges) and the terrestrial (sewage sludge application) (US EPA, 2004; de Voogt and Saez, 2006).

Monitoring data

PFOA is dissociated in water and does not evaporate from the water phase. Water is regarded as the sink compartment for PFOA. A study of spatial distribution of PFOA in European river water calculated emissions of 20 t annually (de Voogt, 2006). Concentrations of PFOA were determined in nine major water bodies (n = 51) of New York State, US (NYS). PFOA was ubiquitous in NYS waters. PFOA was typically found at higher concentrations than were PFOS and PFHS. Elevated concentrations of PFOA were found in the Hudson River (Sinclair et al., 2006).

PFOA is expected to dissociate in the environment almost entirely to the ionic PFO. With its negligible vapour pressure, high water solubility and moderate sorption to solids, accumulation in surface waters is likely (Mabury, 2004). Experiments indicate that perfluorooctanoate is concentrated in the surface foam whether alone in an aqueous solution or with another co-surfactant. This result suggests that foam (marine aerosol) transport should be considered an important transport mechanism (Kaiser et al., 2006). Yamashita et al. (2004) detected PFOA as the major perfluorinated compound detected in oceanic waters, followed by PFOS.

Within the EU project PERFORCE several marine mammal samples were investigated (de Voogt and Saez, 2006; de Voogt, 2006). PFCA concentrations detected were fairly low in all species and tissues analysed.

In general, a positive correlation between high PFOS concentrations and PFCA concentrations was observed. In the same study air samples were also analysed for PFCA. PFOA was present as the predominant compound, in the particle phase, indicating atmospheric transport potentially via particles (de Voogt, 2006).
Herzke et al. (2006) investigated two seabird species, European shag (Phalacrocrrax aristotelis) and common eider (Somateria mollissima) from the Norwegian coast. The samples were collected at a remote bird colony (Island Sklinna, 65°12'N 11°00'E, ca 35 km from the Norwegian coast). Eggs and blood samples were taken from breeding shag females. Livers of juveniles were collected. Eggs from common eider were taken from the same site, one from each nest. PFOA was found in all plasma samples in concentrations between 2-6 ng/g ww, whilst it was not detectable in the shag eggs and only occasionally found in the liver and egg samples of the common eider (Herzke et al., 2006).

The concentrations of PFOS and PFOA in the vacuum cleaner dust collected in Japanese homes were measured. The compounds were detected in all the dust samples and the ranges were 11-2500 ng g(-1) for PFOS and 69-3700 ng g(-1) for PFOA. It was ascertained that PFOS and PFOA were present in the dust in homes, and that the absorption of the dust could be one of the exposure pathways of the PFOS and PFOA to humans (Moriwaki et al., 2003). Particle samples collected along a road in Japan reviled considerable concentrations of PFOA adsorbed to inhalable particles (Harada et al., 2005b; Harada et al., 2006).

Larsen et al. (2005) detected small amounts of PFOA (max 140 ppb) in extracts of polytetrafluoroethylene (PTFE; Teflon), obtained after applying pressure and increased temperatures to the material.

PFOA was found in sewage sludge samples and dominate in landfill effluent during a screening programme financed by the Nordic Council of Ministers (NMR) in 2004. Lake water, seawater and rainwater (precipitation) samples had relatively low contamination with greatest concentrations in rainwater samples from Sweden and Finland (11 ng/L and 11-17 ng/L, respectively). Six Nordic countries participated in the screening study (Denmark, Faroe Islands, Finland, Iceland, Norway and Sweden) (Kallenborn et al., 2006).

In a Norwegian study of PFC in the Norwegian environment, carried out in 2004, PFOA was found in elevated concentrations in cleaned landfill effluents and sediments, mainly as dominating compound. In the aquatic environment, PFOA was also found in freshwater samples and sediments (SFT, 2005b).

PFOA was the dominant compound in a comparative study, investigating Russian and Norwegian human plasma samples in 2004 (median 6.8 ng/g plasma in Norway and 9.9 ng/g plasma in Russia respectively) (SFT, 2005a)

Evaluation of need for screening

Pending the improvements in analytical methodologies, assessment of the fluxes to the environment requires further work on levels of PFC in STP matrices. Further sampling of river water and spill water from textile and paper industry is required to quantify loadings and identify sources (de Voogt, 2006).

More environmental measurements of PFOA in the Arctic are necessary for pathway and sink verification.

Since PFOA is one of the agents used under the production of Teflon, wastewaters and STP sediments from Teflon processing plants have to be screened for PFOA and its lower and higher congeners (Metall coating: IITTALA, Moss; Norwegian Coating Technology, Notodden; Otto Olsen, Lillestrøm; Belegningsteknikk, Drammen); (Textile and ski wax companies: Swix; Skogstad, Bergans and furniture producing companies).
Analyses

The analyses of PFO can be carried out with higher PFS and PFCAs. Different methods can be applied:

a) Extraction with water/methanol; HPLC/ESI-ToF-HRMS.
b) Ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.
c) Extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley (2005) recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005). The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005; de Voogt and Saez, 2006).

If one is only interested in the PFCA a method applying gas chromatography/mass spectrometry can also be used (Alzaga et al., 2005).
2.3.4 Perfluorononanoate – perfluorotridecanoate (PFN - PFTr)

PFN has been manufactured since about 1975 and has been used mainly in the form of the ammonium salt as a surfactant (de Voogt, 2006). Long chained PFCA are formed during the production of PFO and PFN and, depending on the production process, even (ECF) or odd-numbered (Telomer olefin) carbon chains are formed (D'Eon et al., 2006; Prevedouros et al., 2006).

A dynamic method was used to determine the vapour pressures of perfluorooctanoic, -nonanoic, -decanoic, -undecanoic, and -dodecanoic acids (C8-C12) (Kaiser et al., 2005):

<table>
<thead>
<tr>
<th>PFCA</th>
<th>Temperature range (°C)</th>
<th>Vapour pressure range (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFOA</td>
<td>59.2 - 190.8</td>
<td>0.128 - 96.50</td>
</tr>
<tr>
<td>PFNA</td>
<td>99.6 - 203.1</td>
<td>1.120 - 99.97</td>
</tr>
<tr>
<td>PFDoA</td>
<td>127.6 - 247.4</td>
<td>0.856 - 99.96</td>
</tr>
</tbody>
</table>

Toxicological data

LC50 Rat, 4h IH*: 820 mg/ m³ (Kinney et al., 1989).

The acute lethal concentration (LD50) of perfluorodecanoic acid (PFDA), administered intraperitoneally (IP), is 41 mg/kg. The LD50 of PFOA, administered IP, is 198 mg/kg indicating that the longer chain acid is considerably more toxic than PFOA (Olson and Andersen, 1983). PFNA and PFDA are both peroxisome proliferators (Kudo et al., 2000; Lau et al., 2004) and PFDA is reported to produce hepatotoxicity, anorexia, alteration of fatty acid metabolism and reduction of circulating thyroid hormones in the rat (Lau et al., 2004). PFNA induces hepatomegaly, peroxisomal beta-oxidation and microsomal 1-acyl-GPC acyltransferase (Kudo et al., 2006).

Degradation in the environment

The degradation of perfluorinated precursors is a potential indirect source of PFCA in the environment. For example 0.6% of sulphonamide alcohol N-EtFOSE transformed to PFO in a biodegradation study (Prevedouros et al., 2006). For further details on potential degradation routes refer to chapter 2.3.3.

Use in Norway

No data available.

Emissions

Emissions of PFNA can occur during production, product use and as product impurities or degradation products.

Monitoring data

If present in the atmosphere, ionic PFCA are expected to be associated with particles since the vapour pressure is so high. They are expected to either bind to the organic phase in aerosols and/or dissolve in present water, accumulation in surface waters is likely (Mabury, 2004).

As free acids they are more volatile and may be present in the air as well (Prevedouros et al., 2006).

A study conducted by the Norwegian Society for the Conversation of Nature, found PFCA, as the PFC group, showing highest extractable concentrations next to FTOHs, in waterproofed textiles (Berger and Haukas, 2005; Berger and Herzke, 2006).
PFNA was found as the dominating PFCA in fish samples from North America and Asia (Houde et al., 2006). Perfluorocarboxylic acids (PFCAs) with 8-15 carbon (C) atoms were found in glaucous gulls (Larus hyperboreus) caught at Svalbard, Norway, with the highest concentrations found in plasma, compared to liver, brain and egg (sum PFCA: 42-260 ng/g ww) (Verreault et al., 2005).

**Evaluation of need for screening**

Because of lack of knowledge it is recommended that measurements in air are taken. Due to findings of elevated PFNA levels in waterproofed textiles, sampling of wastewater from the textile and furniture industries is also recommended, as well as effluent from STPs. In terms of differences in toxic behaviour and accumulation potential it is recommended analysing for the other PFCAs with carbon chain length from C4 to C15 as well.

**Analyses**

The analyses of PFCA can be carried out with higher PFS. Different methods can be applied:

a) extraction with water/methanol; HPLC/ESI-ToF-HRMS.

b) ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.

c) extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley (2005), recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005). The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005; de Voogt and Saez, 2006).

If one is interested only in the PFCA a method applying gas chromatography/mass spectrometry can be used as well (Alzaga et al., 2005).
2.4 Perfluorinated alcohols

Perfluorinated alcohols are interesting building blocks for pharmaceuticals and agrochemicals (Rosen et al., 2006).

Perfluoroalcohols are not known as a general class of compounds. The CF$_2$OH- and CFOH- groups are unstable and decompose into hydrogen fluoride and perfluorinated carbonyl compounds. This is especially true for primary perfluoroalcohols (Cheburkov and Lillquist, 2002).

However, formation of stable perfluoroalcohol adducts with an amine group are described (Cheburkov and Lillquist, 2002).

$$\text{RFCF}_2\text{O}^+ \text{HNEt}_3^-$$ (RF : F; C$_2$F$_5$; i-C$_3$F$_7$)

Apart from their use in research laboratory no large-scale use is known to the authors.
2.5 Fluorotelomer sulphonates (FTS)

The fluorotelomerisation process, used by the industry, results in an ethyl group being inserted between the fluoroalkyl chain and the end-group. With reference to fluorotelomer sulphonates, the number of fluorocarbons (X) and hydrocarbons (Y) are designated in a ratio X:Y. So the 1H, 1H, 2H, 2H Perfluorooctane-sulphonate is referred to as 6:2 FTS since it has 6 fluorinated carbons and 2 hydrocarbons in the fluoroalkyl chain (Schultz et al., 2004).

Fluorotelomer sulphonates are commercial surfactants mainly applied in aqueous formulations. They lower surface tension and improve wetting and levelling. The fluorotelomer sulphonate (6:2 FTS, also known as THPFOs) is a result of the telomer manufacturing process and has been found in the abiotic environment (de Voogt, 2006). Odd numbered fluorotelomer sulphonates are unlikely to be formed during the telomere manufacturing process since only even-numbered homologues are produced (Banks, 1994).

### Abbreviation | Compound | CAS-Nr.
--- | --- | ---
6:2 FTS | 1H,1H,2H,2H-Perfluorooctanesulphonate | 27619-97-2
8:2 FTS | 1H,1H,2H,2H-Perfluorodecanesulphonate | 

Figure 7: 6:2 Fluorotelomer sulphonate.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
</tr>
</thead>
<tbody>
<tr>
<td>6:2 FTS</td>
<td>1H,1H,2H,2H-Perfluorooctanesulphonate</td>
</tr>
<tr>
<td>8:2 FTS</td>
<td>1H,1H,2H,2H-Perfluorodecanesulphonate</td>
</tr>
</tbody>
</table>

The chemical structure of telomere sulphonates is characterised by a per-fluorinated carbon chain, which is followed by a CH₂-CH₂-group connected to the sulphonate group.

<table>
<thead>
<tr>
<th>Characteristic of the compound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula: F(CF₂)₆₋₁₀CH₂CH₂SO₃⁻</td>
</tr>
<tr>
<td>Melting point: no data</td>
</tr>
<tr>
<td>Vapour pressure: no data</td>
</tr>
<tr>
<td>Water solubility: no data</td>
</tr>
<tr>
<td>Log Kow: not applicable</td>
</tr>
</tbody>
</table>

### Toxicological data

**LD50:** > 500mg/kg

6:2 FTS has a low pH (l) and is therefore considered to be a severe skin and eye irritant, in addition to being corrosive. A NOAEL liver weight of 30 ppm based on increase of liver weight and decreases in body weight was defined by DuPont (Norwegian Institute of Public Health, 2006).

In Norway, Bioforsk conducted ecotoxicological tests in earthworms (*Eisenia fetida*) with PFOS, PFOA and 6:2 fluorotelomer sulfonate. Reproduction studies were performed for the three compounds in agreement with OECD guideline 222. However, in the test using 6:2 FTS the juveniles from the reproduction test were further exposed to 6:2 FTS until they reached sexual maturity, in order to reveal possible effects on offspring after prolonged exposure.

6:2 FTS was less toxic to earthworms than PFOS and PFOA. Harmful effects on reproduction were not observed until soil concentration of 6:2 FTS exceeded 21 mg/kg. Reduced number of cocoons and juveniles, and reduced juvenile body weight was also observed in this experiment. By extending the experiment so that juveniles were followed until they reached sexual maturity, a tendency of delayed growth and development was observed at the highest concentrations (250 mg/kg and 500 mg/kg) during the whole exposure.
period (16 weeks in total). However, the difference in growth and development was not significantly different from the control. BCF were determined for both adults and offspring in this study, and was 3.0 and 2.7, respectively. The bioconcentration of 6:2 FTS in earthworm was similar to that of PFOS. However, it is not certain that 6:2 FTS is as persistent in the environment as PFOS (SFT, 2007).

Degradation in the environment

Degradation of fluoroalkylthioamido-sulphonates into FTS is suggested and 6:2 FTS is susceptible to biodegradation under sulphur-limiting and aerobic conditions (Banks, 1994; Key et al., 1998).

Use in Norway

No data available.

Emissions

Emission of FTS from STP effluents is proven (de Voogt, 2006). As 6:2 FTS is used in fire fighting foams as substitute for PFOS, FTS can be expected in the aqueous environment.

Monitoring data

To our knowledge FTS have not so far been detected in biota. However, during the EU-project PERFORCE, FTS were detected in several environmental samples. 6:2 FTS was present in the particle phase of UK air samples and therefore it is possible that non-volatile ionic FTS might directly undergo atmospheric transport on particles from source regions (de Voogt, 2006).

6:2 FTS (15-300 ng/L) was abundant in the dissolved phase of STP influents and effluents in the Netherlands and in the sludge of STP from the Netherlands, Sweden and United Kingdom (de Voogt, 2006).

Schultz et al. found FTS in groundwater collected near Air Force Bases in the US influenced by heavy use of aqueous fire fighting foam (AFFF) (Schultz et al., 2004). 4:2, 6:2 and 8:2 FTS were also detected in water samples from several air force bases. Odd numbered fluorotelomer sulphonates were not detected.

Evaluation of need for screening

A better characterisation of industrial uses for 6:2 FTS is necessary in addition to the screening of training areas for fire fighting, effluents from sites of fire and effluents from the textile industry.

Analyses

The analyses of FTS can carried out with higher PFS and PFCAs. Different methods can be applied:

a) Extraction with water/methanol; HPLC/ESI-ToF-HRMS.

b) Ion-pairing procedure; extraction with methyl-tert-butylether; HPLC/ESI/MS/MS.

c) Extraction with ethylacetate; treatment with ENVIcarb; HPLC/ESI-ToF-HRMS.

After using the method described by Powley (2005) recoveries for the target analytes extracted from biological samples were typically between 80 and 100%. For fish spleen tissues, recovery ranged between 50 and 70%. Method detection limits were typically around 0.5 and 0.05 ng/g wet weight, respectively. This method was also compared to both the screening and the ion pair extraction method and results were given in Berger et al. (2005). The modified Powley method is recommended as the method of choice for trace analysis of perfluorinated compounds in biological samples (Hansen et al., 2001; Powley et al., 2005; Berger and Haukas, 2005; de Voogt and Saez, 2006).
2.6 Fluorotelomer acids; saturated and unsaturated (FT(U)CA)

The 8:2 fluorotelomer alcohol (8:2 FTOH) was found to undergo indirect photolysis to the 8:2 fluorotelomer acid (8:2 FTCA), and perfluorooctanoate (PFOA); the minor products monitored were the 8:2 fluorotelomer unsaturated acid (8:2 FTUCA) and perfluorononanoate (PFNA). 8:2 FTCA and 8:2 FTUCA are intermediates which photodegrade further to PFOA (major) and PFNA (minor) (Gauthier and Mabury, 2005).

### Abbreviation | Compound | CAS-Nr.
--- | --- | ---
6:2 FTCA | 1H,1H,2H,2H-Perfluorooctane-carboxylate | 84-10-3
8:2 FTCA | 1H,1H,2H,2H-Perfluorodecane-carboxylate | 861-67-0
8:2 FTCA | | 87-85-0
8:2 FTUCA | | 851-35-5

![Figure 8: 1H, 1H, 2H, 2H- Perfluorodecane-carboxylate.](image)

### Toxicological data

- **LC$_{50}$:** no data
- **LD$_{50}$:** no data

The toxicity of the 4:2, 6:2, 8:2 and 10:2 saturated (s) and unsaturated (u) forms of the FTCAs were assessed on crustaceans (*Daphnia magna*), midge (*Chironomus tentans*) and duckweed (*Lemna gibba*). Acute toxicity studies indicated that all three species were most sensitive to FTCAs with chain lengths $\geq$ 8 fluorocarbons (FCs). *L. gibba* was the most sensitive of the three to FTCAs of chain lengths $\leq$ 8 FCs, with EC50 values for growth ranging from 0.71-10.04 mg/L for the 8:2 and 6:2 u-FTCAs, respectively. *D. magna* was the most sensitive to FTCAs of chain lengths $>$ 8 FCs, with EC50 values for immobility of 0.025 and 0.279 mg/L for the 10:2 s-FTCA and u-FCTA, respectively. In all three species, toxicity increased with increasing chain length from 6 to 8 FCs. This trend continued for *D. magna* through FC chain lengths of 10, but not for *C. tentans* or *L. gibba*. The s-FTCAs were generally more toxic than corresponding u-FTCAs with the exception of the 8:2 FTCA for *L. gibba*, and the 10:2 FTCA for *C. tentans* and *L. gibba*. A 60-d life cycle assay with *C. tentans* and the 8:2 s-FTCA resulted in toxicity thresholds for growth and mortality 5-6 times smaller than those measured in the acute study. The chain-length trends observed in the acute studies agree with those previously reported for the PFCAs, but toxicity thresholds were 1-4 orders of magnitude smaller for the FTCAs (MacDonald et al., 2005).

### Characteristic of the compound

- **Molecular formula:** F(CF$_2$)$_{6-10}$CH$_2$CH$_2$CO$_2$
- **Melting point:**
  - 87.8°C (8:2 FTCA)
  - 106.6°C (8:2 FTUCA)
- **Vapour pressure:**
  - 0.187 kPa (8:2 FTCA)
  - 0.39 kPa (8:2 FTUCA) (Kaiser MA et al., 2006)
- **Water solubility:** no data
- **Log K$_{ow}$:** not applicable

The chemical structure of fluorotelomere acids is characterised by a perfluorinated carbon chain, which is followed by a CH$_2$-CH$_2$-group connected to the carboxilate group. FTCAs are not commercially produced substances. FTCAs are the degradation product of FTOHs in the atmosphere (Ellis et al., 2004; Scott et al., 2006) and via occur biodegradation via the unsaturated FT(U)CAs as precursors (Dinglasan et al., 2004; Wang et al., 2005).
Degradation in the environment

Biotic and abiotic oxidation of the FTOHs yield saturated and unsaturated fluorotelomer carboxylic acids (FTCAs) (MacDonald et al., 2005). Dinglasan et al. (2004) found that FTCA can be transformed via a beta-oxidation mechanism, leading to an unsaturated acid and ultimately producing the highly stable PFOA.

Aresenault et al. (2006) found that saturated FTCAs are readily degraded to the corresponding unsaturated acids in the presence of a base in methanol, but not in water.

Use in Norway

No data available.

Emissions

No data available.

Monitoring data

To our knowledge, there are no reports on the detection of FTCAs or FTUCAs in biota. However, Loewen et al. detected low levels of C10- and C12-FTCA and FTUCA in rainwater collected in Winnipeg, Canada (Loewen et al., 2005).

Evaluation of need for screening

A better characterisation of findings of FT(U)CAs as degradation products of FTOHs is necessary.

Analyses

The analyses of FT(U)CAs can be carried out together with PFS and PFCAs. The lack of isotope labelled internal standards reduces the certainty of the results.

Lower volatility and higher water solubility of FTCAs compared with their FTOH counterparts suggest surface waters as a likely a repository for FTCAs however no fate data exist for this environmental matrix (MacDonald et al., 2005).
2.7 Fluorotelomer alcohols (FTOH)

Fluorotelomer alcohols are manufactured as a raw material used in the synthesis of fluorotelomer-based surfactants and polymeric products.

For example, fluorotelomer-based acrylic polymers (FBAPs) are chemicals used for the coating of textiles, paper and carpet to achieve oil, stain and water repellent properties. The major building block of these high molecular polyacrylates is the fluorotelomer alcohol 2-perfluorooctylethanol (8:2 FTOH).

The manufacture of FTOHs usually results in a mixture containing six to twelve fluorinated carbon congeners, the 8:2 FTOH being the dominant one. Fluorotelomer alcohols are present in the consumer products as residual raw materials. The estimated global production of fluorinated telomer alcohols is ca. 11-14 x 10^6 kg/yr and increasing (Dinglasan-Panlilio and Mabury, 2006).

On the basis of their volatility, polyfluorinated telomer alcohols are expected to occur predominantly in the atmospheric gas phase. However, given their low solubility in water and high sorptivity to organic solvent or sorbent, the 8:2 fluorotelomer alcohol is expected to partition to the air compartment only under conditions where no sorptive medium is present (Kaiser et al., 2006).

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>6:2 FTOH</td>
<td>1H,1H,2H,2H-Perfluorooctanol</td>
<td>647-42-7</td>
</tr>
<tr>
<td>8:2 FTOH</td>
<td>1H,1H,2H,2H-Perfluorodecanol</td>
<td>865-86-1</td>
</tr>
</tbody>
</table>

On the other hand, the K_{WA} (water-air partition coefficient) values of the three fluorinated telomer alcohols extrapolated to 25°C are of a similar order of magnitude (1 < log K_{WA} < 2) and suggest that rain scavenging is not a very efficient atmospheric deposition process (Lei et al., 2004).

Fluorotelomer alcohols have higher calculated vapour pressures than the parent alcohol; e.g., the 10:2 FTOH is 1000 times more volatile compared to dodecanol (Stock et al., 2004a) and in the absence of a solution or adsorbed state, 8:2 FTOH rapidly sublimes at ambient temperature (Kaiser et al., 2006).

The chemical structure of telomere alcohols is characterised by a perfluorinated carbon chain, which is followed by a CH₂-CH₂-group connected to the hydroxy group.

Li et al. (2006) report that the uptake of FTOHs on or into the aqueous component of cloud/fog droplets or aqueous aerosol particles is unlikely to be an important atmospheric sink for FTOH. However, the larger uptake coefficient measured for 1-octanol surfaces indicates that FTOH partitioning to organic-containing cloud/fog droplets and aerosol particles may be an atmospheric loss mechanism.
In recent work by Fasano et al. (2006) rats were administered 8-2 fluorotelomer alcohol (8-2 FTOH). The plasma elimination half-life was estimated to less than 5 hours with no sex related differences. Most of the FTOH was eliminated in faeces of which 37-55% was identified as the parent compound. There are reasons to believe that the different FTOH compounds have a relatively short half-life, both in biota and in abiotic environment. Several studies have shown that FTOH compounds are metabolized to their carboxylic moiety, such as PFOA (Dinglasan et al., 2004; Wang et al., 2005; Wallington et al., 2006; Martin et al., 2005). Administration of 8:2 FTOH to pregnant rats showed transfer of the metabolites PFOA and PFNA to the neonates (Henderson and Smith, 2006). Pregnant rats were administered 8-2 telomer B alcohol from day 6 through 20 of gestation at daily doses of either 0, 50, 200, or 500 mg/kg (Mylchreest et al., 2005). Mortality was observed at 500 mg/kg. The NOAEL for both maternal and developmental toxicity was estimated to be 200 mg/kg/day and was not considered to be a selective developmental toxicant in rats. A recent investigation of Maras et al. (2006) indicated that 8:2 FTOH has oestrogen-like properties as shown by its ability to induce breast cancer cell proliferation.

Treatments with 8:2 telomer alcohol caused liver enlargement in a dose- and duration-dependent manner. Peroxisome proliferation in the liver of mice was confirmed by electron microscopic examination. Peroxisomal acyl-CoA oxidase was induced by these treatments with 8:2 telomer alcohol in a dose and time dependent manner. Five metabolites, namely, perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), 2H, 2H-perfluorodecanoic acid (8:2 telomer acid), and two unidentified metabolites, were present in the liver and serum. PFOA was confirmed to be accumulated in the liver of mice following the administration of 8:2 telomer alcohol in a dose and duration dependent manner. A linear relationship was observed between the concentration of PFOA and the activity of peroxisomal acyl-CoA oxidase in the liver of mice. These results strongly suggest that PFOA, but not 8:2 telomer alcohol itself, caused peroxisome proliferation in the liver Kudo et al. (2005).

Degradation in the environment

The oxidation of fluorotelomer alcohols in the atmosphere by OH-radicals leads quantitatively to the production of the corresponding polyfluorinated aldehyde, being further degraded to PFCA (Hurley et al., 2004; Ellis et al., 2004; Gauthier and Mabury, 2005; Andersen et al., 2005; Sulbaek Andersen et al., 2006).

Atmospheric lifetime of short chain FTOHs was determined to be 20 days, enabling the molecules to be transported upto 7000 km by air (Ellis et al., 2004; Wallington et al., 2006).

However, Gauthier et al. (2005), photodegraded 8:2 fluorotelomer alcohol (8:2 FTOH) in aqueous hydrogen peroxide solutions, synthetic field water (SFW) systems, and Lake Ontario (Canada) water...
samples. It was found to undergo indirect photolysis, with the data suggesting that the hydroxyl radical was the main degradation agent and that nitrate promoted photolysis whereas dissolved organic carbon inhibited it. The half-lives of 8:2 FTOH were 0.83 +/- 0.20 h (10 mM H$_2$O$_2$), 38.0 +/- 6.0 h (100 µM H$_2$O$_2$), 30.5 +/- 8.0 to 163.1 +/- 3.0 h (SFW systems), and 93.2 +/- 10.0 h (Lake Ontario). No significant loss of the parent compound by direct photolysis could be observed.

The major monitored products were the 8:2 fluorotelomer aldehyde, the 8:2 fluorotelomer acid (8:2 FTCA), and perfluorooctanoate (PFOA); the minor monitored products were the 8:2 fluorotelomer unsaturated acid (8:2 FTUCA) and perfluorononanoate (PFNA).

The intermediates, 8:2 FTCA and 8:2 FTUCA, were photodegraded to verify the degradation pathway, and a mechanism for the photolysis was proposed whereby the end products of the photolysis pathway were PFOA (major) and PFNA (minor) (Gauthier and Mabury, 2005).

![Figure 10: Proposed mechanism for the degradation of the 8:2 fluorotelomer alcohol (FTOH) and its impurity, the allylic 8:2 FTOH; taken from (Gauthier and Mabury, 2005).](image)
Dinglasan et al. (2004) examined the aerobic biodegradation of the 8:2 telomer alcohol using a mixed microbial system. The initial measured half-life of the 8:2 FTOH was similar to 0.2 days/mg of initial biomass protein. Telomer acids and PFOA were identified as metabolites during the degradation, the unsaturated telomer acid being the predominant metabolite measured. The overall mechanism involves the oxidation of the 8:2 FTOH to the telomer acid via the transient telomer aldehyde. The telomer acid via a beta-oxidation mechanism was further transformed, leading to the unsaturated acid and ultimately producing the highly stable PFOA. Telomer alcohols were demonstrated to be potential sources of PFCAs as a consequence of biotic degradation.

Biological transformation may be a major degradation pathway for fluorinated telomer alcohols in aquatic systems (Dinglasan et al., 2004).

**Use in Norway**

No data available

**Emissions**

Unreacted telomere alcohols can potentially gas-off during production of the monomer as well as from polymeric materials (up to 4% unbound alcohols detected fluorinated materials) (Andersen et al., 2005; Dinglasan-Panlilio and Mabury, 2006).

The residual fluoro-alcohol contribution to the atmospheric load of FTOH is significant and may be the dominant source. Release of FTOH may occur all along the supply chain from production, application into consumer use and disposal.

For example, a Teflon product, analysed by Dinglasan et al., contained telomere alcohols with chain length ranging from 8 to 14 carbons. The 8:2 alcohol was found in highest concentrations. (Dinglasan-Panlilio and Mabury, 2006).

**Monitoring data**

FTOHs were found in the North American atmosphere (Martin et al., 2002; Stock et al., 2004b). However, present modelling results show that with current estimates of chemistry and fluxes the atmospheric oxidation of 8:2 FTOH can provide a quantitative explanation for the presence of PFCAs in remote regions (Wallington et al., 2006).
During the PERFORCE project, 6:2-, 8:2- and 10:2 FTOH were measured in air samples from several sites in Europe (both rural and urban) and 8:2 FTOH was the dominant alcohol at all outdoor sampling sites, with slightly higher levels compared to data from North America. By the same study FTOHs could be detected in indoor air as well (Jahnke et al., 2007b; de Voogt, 2006; Martin et al., 2006).

Berger and Herzke (2006) detected several FTOH in weather clothing purchased from the Swedish and Norwegian market. Again 8:2 FTOH dominated all analysed PFCs in the sample followed by 10:2 FTOH. In a few samples 6:2 FTOH could be detected, but not 4:2 FTOH (SFT, 2006; de Voogt, 2006; Berger and Herzke, 2006).

**Evaluation of need for screening**

A better understanding of degassing amounts of FTOH is needed for both indoor and outdoor environments. In addition the degradation of FTOH in consumer products to PFCA should be investigated.

More environmental measurements of FTOHs in the Arctic are needed, as well as aldehydes and acids for pathway and sink verification.

Identification of how much of FTOHs originates from:
- Residuals
- Fluorinated polymers (breakdown in use and disposal, including)
- Industrial cleaning effluents and dry cleaning effluents

Identification of the relative importance of abiotic/biotic FTOH \(\rightarrow\) PFCA degradation processes at different trophic levels and geographic regions as well as in human blood.

**Analyses**

The analyses of FTOH can be carried out by active/passive air sampling on PUF or XAD resin, followed by extraction of the samplers with subsequent GC/MS in PCI mode (Dinglasan-Panlilio and Mabury, 2006). Berger et al. developed a LC/MS technique quantifying FTOHs (Berger and Haukas, 2005; Dinglasan-Panlilio and Mabury, 2006; Larsen et al., 2006). A GC/MS method for detection is described by Jahnke et al. (2007a). Extraction from textile samples was performed in ethyl acetate in the ultrasonic bath. GC-MS analysis (Berger and Herzke, 2006).

Barber et al. (2007) describe method quantitation limits (MQLs) of typically around or below 1 pg/m\(^3\) for analyses of FTOH in air. Higher MQLs in the range of 5-120 pg/m\(^3\) were found for indoor studies as a result of the much lower sampling volume. The common method of air sampling with PUF/XAD cartridges will result in breakthrough of the lighter FTOHs, such as 6:2 FTOH and in particular 4:2 FTOH, and thus air concentrations will be underestimated. Breakthrough may be minimised by sampling smaller volumes of air, and the addition of mass labelled analogues to the sampling media prior to sampling will account for these losses (Jahnke, 2007a). During the sample extraction process, some volatilisation losses occur for 4:2 FTOH, 6:2 FTOH, 10:2 FTolefin and almost certainly the other FTolefins. These losses can be accounted for by the use of mass labelled IS where available, and/or odd number fluorinated alcohols such as 5:1 FA, 7:1 FA etc. Currently the extraction process does not include a clean-up stage, and consequently large final extract volumes and daily GC maintenance are required in order to analyse samples (Barber et al., 2007).
2.8 Fluorotelomer olefines (FTolefine) and fluorotelomer iodide

Similarly to the FTOHs, the fluorotelomer olefins (FTolefins) are used in the production process of other fluorinated compounds. The FTolefins have so far not been reported to be of any environmental concern.

FTiodide is an intermediate in the polymer production processes in addition to the synthesis of FTOH and PFCA (e.g. PFOA).

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:2 FTolefin</td>
<td>1H,1H,2H Perfluorodecene</td>
<td>21652-58-4</td>
</tr>
<tr>
<td>10:2 FTolefin</td>
<td>1H,1H,2H Perfluoroundecene</td>
<td>30389-25-4</td>
</tr>
<tr>
<td>8:2 FTiodide</td>
<td>1H,1H,2H,2H-Perfluorodecyliodide</td>
<td>2043-53-0</td>
</tr>
</tbody>
</table>

Figure 12: 8:2 Fluorotelomer olefine.

The chemical structure of telomere sulphonates is characterised by a per-fluorinated carbon chain which is followed by an ethylene (CH=CH2) group. Both compound groups are characterised by high vapour pressure.

Toxicological data

No data available.

Degradation in the environment


Use in Norway

No data available.

Emissions

No data available.

Monitoring data

Through cooperation and financial support the Swedish and Norwegian Societies for Nature Conservation (Svenska naturskyddsföreningen and Norges naturvernforbund), and the Norwegian Pollution Control Authority (Statens forurensningstilsyn; SFT) commissioned the analysis of several all-weather-clothing textiles for, among others, FTolefines. 10:2 FTolefines was detected in low concentrations in some textiles and the only detected olefine in the samples (SFT, 2006; Berger and Herzke, 2006).

Evaluation of need for screening

A better characterisation of industrial uses for FTolefins is needed.

Identification of how much of FTolefins is derived from:
- Residuals
- Fluorinated polymers (breakdown in use and disposal)
Analyses

Extraction of textile samples was performed in 50 mL ethyl acetate in an ultrasonic bath for 30 minutes followed by GC-MS analysis (Berger and Herzke, 2006).

The current method of air sampling with PUF/XAD cartridges is still under development. During the sample extraction process, some volatilisation losses occur for 10:2 FTolefin and almost certainly the other FTolefins. These losses can be accounted for by the use of mass labelled IS. However, these options do not currently exist for FTolefins. Currently the extraction process does not include a clean-up stage, and consequently large final extract volumes and daily GC maintenance are required in order to analyse samples (Barber et al., 2007).
2.9 Fluorotelomer aldehydes (FTAL)

Telomer aldehydes are the transient product of the degradation of FTOH to the telomer acid. The telomer acid via a beta-oxidation mechanism is further transformed, leading to the unsaturated acid and ultimately producing the highly stable PFOA (Dinglasan et al., 2004).

![8:2 FTAL and 8:2 PFAL](image)

Figure 13: 8:2 Fluorotelomer aldehyde and perfluorodecanal.

**Characteristic of the compound**
- Molecular formula: \( \text{F(CF}_2\text{)_6-10CH}_2\text{CH}_2\text{CHO} \)
- Melting point: no data available
- Vapour pressure: 0.067 kPa (8:2 FTAL) (Ellis et al., 2004b)
- Water solubility: no data available
- Log K<sub>ow</sub>: no data available

**Toxicological data**

There are potential toxicological implications from the binding of the unsaturated compound FTUAL, the degradation product of polyfluorinated telomere aldehyde to the glutathione GSH. This is based on knowledge gained for other direct acting alkylating agents, include the impairment of enzymes and genotoxicity (Martin et al., 2005).

**Degradation in the environment**

The oxidation of fluorotelomer alcohols in the atmosphere by OH-radicals leads to the production of the corresponding polyfluorinated aldehyde in which there is a \( \text{CH}_2 \) moiety left intact between the aldehyde functional group and the perfluorinated alkyl chain. This process indicates that the alcohol and the aldehyde have atmospheric lifetimes on the order of 20 days. Wet and dry depositions are expected to be negligible for these compounds in comparison to their OH chemistry and that the predominant oxidative pathway of the first formed aldehyde with OH is the production of a further aldehyde which is perfluorinated. In the absence of NOx, this perfluorinated aldehyde undergoes further oxidation by OH to produce the corresponding PFCA. A second pathway available to the perfluorinated aldehyde is the production of shorter chain PFCAs. It is suggested that although the production of PFCAs by this second route is minor in comparison to the production of carbonyl fluoride it is still environmentally significant (Ellis et al., 2003; Ellis et al., 2004).

Aldehyde metabolites were identified in isolated rat hepatocytes incubated with FTOH (e.g. 4:2, 6:2, 8:2, and 10:2 FTOH in individual experiments) (Martin et al., 2005). 8:2 FTAL was incubated with hepatocytes for 2 h to determine its respective metabolites. No trace of 8:2 FTAL or 8:2 FTUAL was detectable after 2 h, but acid metabolites included small amounts of PFOA, PFNA, 8:2 FTCA, and 8:2 FTUCA. These were quantified but the molar balance of the acid products was low (<10%), suggesting that oxidation to carboxylic acids was not the primary fate for the aldehyde.

Experimental observations suggested that 8:2 FTAL is unstable in water, and dehydrofluorinated (90% in 90 min) at a physiological temperature and pH to yield 8:2 FTUAL. 8:2 FTUAL itself is also transient, however, its fate is unknown and volatilisation cannot be ruled out (Martin et al., 2005).

**Use in Norway**

Not applicable since this is a transient product.
Emissions

No data available.

Monitoring data

No data available.

Evaluation of need for screening

Questions still remain concerning the degree to which wet deposition plays a role in the atmospheric fate of the per-fluorinated aldehyde and how significant these pathways are to the environmental burden of PFCAs with varying amounts of NOX.

More environmental measurements in the Arctic of FTOHs, aldehydes and acids for pathway verification are needed additional to all environments with high exposure to FTOH, for example indoor air and effluents from textile industry etc.

Analyses

Aldehydes can be detected by HPLC/MS/MS as the respective hydrazone derivative following reaction with DNPH. Reversed phase chromatography of the hydrazones can be performed using an acetonitrile: water gradient elution program and with MS/MS detection by multiple reaction monitoring (Martin et al., 2005).
2.10 Polytetrafluoroethylene (Teflon, PTFE)

The fluoro polymer polytetrafluoroethylene (PTFE) is generally known to the public by DuPont's brand name Teflon®. PTFE has the lowest coefficient of friction (against polished steel) of any known solid material. It is used as a non-stick coating for pans and other cookware. PTFE is virtually non-reactive, and so is often used in containers and pipe work for reactive chemicals. According to DuPont its melting point is 327 °C although its properties start to degrade above 260 °C.

DuPont™ Teflon® fluoropolymer products are used in industrial and commercial applications, such as cabling solutions, semiconductor manufacturing, pharmaceutical and biopharma manufacturing, food processing equipment and industrial bakeware (http://www2.dupont.com/Teflon_Industrial/en_US/uses_apps/index.html).

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTFE</td>
<td>Polytetrafluoroethylene</td>
<td>9002-84-0</td>
</tr>
</tbody>
</table>

The world consumption of fluoro-polymers reached a record level of 123 million kg per year in 2001 and is still increasing. PTFE production came to 61% of the overall fluorinated polymer production worldwide in 1988. No more recent data are available, but an increased trend in production numbers of PTFE is very probable.

Traditionally PTFE waste is decomposed by thermal treatment over 500°C (Zou et al., 2004). Refer to chapter K. in order to find more information concerning thermo degradation and combustion of fluoropolymers.

Additional pyrolants based on Magnesium/Teflon®/Viton® (MTV) have been in use since the 1950s as payloads in infrared decoy flare applications for military purposes. Titanium, PTFE and Viton pyrolants, which generate high temperature products, are used in partition type igniters (Kuwahara et al., 2005).

Viton is a trademark of vinyliden-fluoride-hexafluoropropene-copolymer (\(CH_2CF_2\)\(_n\)(CF\((CF_3)CF_2\)\(_n\). In MTV compositions the PTFE, acts as fluorine source. Magnesium fluoride is formed under the process of combustion, resulting in intense heat and flame formation (Koch, 2002).

**Characteristic of the compound**
- Molecular formula: \((CF_2-CF_2)_n\)
- Melting point: 327°C
- Vapour pressure: not applicable
- Water solubility: not applicable
- Log K\(_{ow}\): not applicable

**Toxicological data**

Johnston et al. (2000) documented that PTFE fumes consisting of large numbers of ultrafine (uf) particles can cause severe acute lung injury: (i) uf PTFE fume particles are causally involved in the induction of acute lung injury, (ii) uf PTFE elicit greater pulmonary effects than larger sized PTFE accumulation mode particles, and (iii) preexposure to the uf PTFE fume particles will induce tolerance (Oberdorster, 2000; Johnston et al., 2000).
Studies on rats confirmed the high pulmonary toxicity of inhaled PTFE fumes containing ultrafine particles (Johnston et al., 1998b; Johnston et al., 2000). In addition, mice were exposed to air containing PTFE particles. Median particle size was similar to 20 nm. Lung lavage and total RNA isolation of the lung were performed. Compared to the previous studies in rats with the same exposure concentrations, the inflammatory response elicited in mice was less severe and no deaths occurred. The lowest exposure concentration had no effect on message levels in either group of mice. The mid-exposure level increased mRNA levels for messages encoding IL-1 beta, IL-3, IL-6, MnSOD, and Mt in young C57BL/6 mice. In contrast, at this level less dramatic increases were measured for IL-1 beta, IL-6, and Mt in old mice. After the highest exposure concentration, IL-1 beta, IL-3, IL-6, Mt, and MnSOD were increased. Old mice also demonstrated increased mRNA abundance for IL-1 beta, IL-3, IL-6, Mt, and MnSOD. However, at this concentration TNF alpha mRNA levels were induced 12-fold in old mice, whereas only a threefold induction was measured in 8-wk-old mice. Lung lavage PMN levels were also threefold greater in the old mice compared to young mice. These findings suggest inflammatory response to PTFE fumes with ultrafine particles is altered with age, and that the inflammatory response is greater in the old animals (Johnston et al., 1998; Johnston et al., 2000).

According to DuPonts Safety in Handling and Use-recommendations, Teflon® AF resins contain parts per million of residual hexafluoroacetone (HFA). In addition fluorine-containing monomers form the basis for the production of a large number of commercially important fluoropolymers. Most of the polymerisation occurs as gas-phase reactions, hence the hazards associated with the monomers arises primarily from inhalation. The chemicals include bromotrifluoroethylene (BTFE), chlorotrifluoroethylene (CTFE), hexafluoroacetone (HFA), hexafluorobutylene (HFIB), hexafluoropropylene (HFP), perfluorobutylene (PFBE), tetrafluoroethylene (TFE), trichloropropene (TFP), vinyl fluoride (VF), and vinylidene fluoride (VF2).

In animal models and in humans, these monomers may be absorbed into the body at varying rates and the metabolism ranges from extensive to little in a species, dose, and chemical specific fashion. The major target organ of these materials is the kidney, and the degree of involvement depends greatly on the excretion patterns and metabolic profiles of the monomers (Kennedy, 1990).

In animal tests, HFA has been shown to be a potent reproductive and developmental toxin (Kennedy, 1990; Lee and Kennedy, 1991). HFA hydrates are readily absorbed through the skin and it is necessary to avoid skin contact with the resin during processing. DuPont recommends the use of protective gloves if handling of the resin is required during manufacturing operations. Some residual gases (including HF, COF₂, CO, and HFA), which may be harmful, diffuse from Teflon® AF resins even at room temperature (http://www2.dupont.com/Teflon_Industrial/en_US/assets/downloads/h75334.pdf). Therefore, to avoid exposure, all resin containers should be opened and used only in well-ventilated areas using local exhaust ventilation.

For more information see also the report for the Norwegian Pollution Controll (SFT): “Helserisikovurdering for SFT av Teflon-Polytetrafluoroetyle (PTFE) CAS-Nr. 9002-84-0; utført av Nasjonalt folkehelseinstitutt”.

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Degradation in the environment

Its chemical-structure, consisting only of carbon and fluorine atoms, which form a very strong bonding and molecular structure, PTFE is regarded as extremely stable and inert against all kinds of degradation.

PTFE is highly resistant to chemical and physical degradation, has almost no water absorption and is characterised as weather resistant in 20 years Florida weather (http://www2.dupont.com/Teflon_Industrial/en_US/tech_info/techinfo_compare.html).

The fluoropolymer PTFE is a heat resistant polymeric material characterised by a maximum continuous use temperature of 260°C (Cho et al., 2005).

The photodegradation of PTFE was studied by Ono et al. (2005). Mass analysis identified the carbon containing groups CF$_n^+$ (n = 1–3) in the molecular emission from PTFE, indicating that the polymer C–C backbone undergoes scission upon the photodegradation but only minor C=C double bond generation.

The leading mechanism for degradation of commercially-available PTFE membranes occurs by free peroxide radicals. The degradation is initiated by abstraction of a hydrogen atom from residual carboxylic acid ends and at less active positions as well on PTFE backbones. Such atom abstraction initiates a systematic chain oxidation to carbon dioxide and hydrogen fluoride (Schiraldi, 2006).

Zou et al. (2004) reported a method to degrade PTFE in a potassium bath at 100 and 180°C. PTFE was completely degraded to carbon after 30 hours.

For more information see also the report for the Norwegian Pollution Controll (SFT): “Miljøvurdering av miljø-informasjon vedrørende Teflon og nedbrytningsprodukter fra Teflon; NIVA”.

Use in Norway

Truck & Trailer Industry A/S produces and sells Teflon spray products.

Emissions

Fluoropolymers are films (e.g., on nonstick cookware) or membranes (e.g., in outerwear). The ammonium salt of PFO (ammonium perfluorooctanoate, APFO) is an essential processing aid in the formulation of such fluoropolymers. Thus, residual PFO may be present in fluoropolymer films and membranes used in manufacturing certain consumer articles. However, PFO was not detected in over 40 extraction tests on nonstick cookware, under test conditions simulating cooking and prolonged food or consumer contact. The manufacture of nonstick cookware includes a high temperature step (i.e., sintering) that supposedly degrade residual PFO prior to article use by consumers (Washburn et al., 2005; Krusic et al., 2005).

A Norwegian study, investigating waterproofed textiles for PFC, also included the analyses of a teflon-coated table-cloth. As can be seen from Table 1, FTOHs dominated the PFAS class extracted from the textiles. FTOHs chemically bound to an oligomeric/polymeric backbone structure might cleave off at high temperatures as used in the GC injector. Therefore, the fluorotelomer alcohols were also quantified in the methanol extracts by LC-MS, and the high concentrations of freely extractable FTOHs were confirmed. However, there were big differences in the concentrations found in the different textiles. Whereas FTOHs were not detected in one sample, as much as 11 mg/m$^2$ were extracted from the jacket with the highest FTOH content. Next to FTOHs, PFCAs were the PFAS group showing highest extractable concentrations. Levels exceeded 0.1 and 0.4 mg/m$^2$ for a PTFE table-cloth and a cotton textile, respectively. Only minor amounts of PFS
Table 1: Freely extractable PFAS from different textile samples summarized in groups (μg/m² textile).

<table>
<thead>
<tr>
<th></th>
<th>10:2 FTolefin</th>
<th>Sum FTOHs</th>
<th>Sum FTS/FTCAs</th>
<th>Sum PFS</th>
<th>Sum PFCAs</th>
<th>Sum FOSAs/FOSEs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sandvika Seiersborg – PTFE table-cloth</td>
<td>n.d.</td>
<td>285</td>
<td>5.56</td>
<td>0.04</td>
<td>170</td>
<td>0.03</td>
</tr>
</tbody>
</table>

(including PFOS) were detected, however, they correlated with the values for FOSAs/FOSEs, which are possible precursor compounds for PFOS.

The increase in production and use of PTFE containing material will mean that waste management will have to deal with increasing amounts of non-degradable material. Waste incineration processes will have to be state of the art in order to exclude the formation of toxic fluorinated compounds. Refer to chapter K. for more information about thermodegradation of fluoropolymers.

**Monitoring data**

No data available.

**Evaluation of need for screening**

Increased amounts of waste PTFE containing material in the future will require both waste incineration plants and landfills will have to be monitored in order to assess the emissions of toxic fluorinated compounds.

Occupational exposure with fluorine-containing monomers should be evaluated and reduced to a limit.

**Analyses**

Not applicable for Teflon, but PFCA analyses of Teflon-coated consumer products have been done. In addition volatile fluorinated decomposition products have been studied in air close to Teflon-processing plants.
2.11 Combustion/ thermodegradation of fluoropolymers

Fluorinated plastics possess properties that are beyond the performance of other plastics: they have universal chemical resistance, an extremely low friction value, stability to ageing, and possible application temperatures between -200°C and +260°C.

Thermoplastically processable copolymers, partially or fully fluorinated or combined with non-fluorinated monomers, such as ethylene, broaden the scope. More stringent industrial requirements, a demand for increased safety, freedom of maintenance, and environmental protection open up even more applications. Traditionally, the emphasis on use was in the chemical and pharmaceutical industries, automobile and machine engineering, and electrical engineering (Korinek, 1993).

Gustaffson et al. (2006) studied the time scales involved in the decomposition and combustion of fluorinated polymers tested at temperatures between 760 and 1370°C. Fluorine rich polymers release both hydrogen and fluorine during thermal decomposition, leading to a suppression of combustion promoting hydroxyl radicals. As a result, fire events are suppressed. The polymer containing a similar amount hydrogen and fluorine produced the highest amount of HF in the shortest time, compared to products containing less or no hydrogen. Carbon monoxide, COF₂, H₂O and CO₂ are the main decomposition products.

The thermolysis of fluorinated polymers, such as the commercial polymers Teflon and Kel-F, can produce trifluoroacetate and the homologue chlorodifluoroacetate. This can occur in the atmosphere either directly or indirectly via products that degrade to these haloacetates (Mashino et al., 2000a; Mashino et al., 2000b). Tetrafluoro-ethylene, hexafluoro-propene and cyclooctafluorobutane are the main gases produced during thermolysis of pure fluorinated polymers besides trifluoroacetate. Thermolysis also leads to longer chain polyfluoro- and/or polychlorofluoro- (C₃-C₁₄) carboxylic acids (PFCA) (Ellis et al., 2001).

For example, DuPonts Teflon® AF resins are thermally stable up to about 360°C in air. The isothermal rate of weight loss of Teflon® AF in air at 360°C has been shown to be 0.2 to 0.6%/hr. When Teflon® AF resins are decomposed in air at temperatures from 360 to 450°C, HF from reaction of COF₂ with moist air, COF₂, CO, and hexafluoroacetone (HFA) are released. The amount of HFA evolved upon complete decomposition at 500°C is up to 100–200 mg/g of sample (http://www2.dupont.com/Teflon_Industrial/en_US/assets/downloads/h75334.pdf).

Furthermore, CFCs and fluorocarbons-groups that can destroy ozone and act as greenhouse gases respectively, were detected among thermal degradation products of PTFE, suggesting that waste incineration of fluoropolymers may also exacerbate stratospheric ozone-depletion and global warming (Ellis et al., 2001).

Clarke et al. (1992) reported a temperature related composition of smoke from PTFE combustion. At temperature of 750°C 20% of the smoke consist of octafluorobutene (OFB) and tetrafluoroethylene (TFE), but almost no tetrafluoromethane. After increasing the temperature to 850°C tetra- and trifluoromethane were the dominant fluorocarbons, with only small amounts of OFB and TFE.

Characteristic of the compound

Temperature of 10% weight loss: 160-310°C (Gustavsson et al., 2006)

Toxicological data

LC₅₀: 3 mg/L
(smoke from fire of PTFE cable insulating; (Clarke et al., 1992)).
The smoke of 30 kg of telecommunications cable insulated and jacketed with fluorinated materials (Teflon(R) FEP and/or Teflon(R) PFA) was exposed to fires involving 110 kg wood cribs, or energetically equivalent amounts of diesel fuel or polyurethane foam. An observed fluorine loss was probably attributable to deposition of hydrogen fluoride in the high-humidity conditions associated with combustion. Exposed male Sprague-Dawley rats showed some of the effects to combustion products of fluoropolymers, but also showed near-lethal amounts of blood COHb, attributable to carbon monoxide from the principal fuel.

The lethal smoke concentration of the cable smoke alone, i.e., without the effects of the carbon monoxide contributed by the principal fuel, is estimated to be 1.6 mg/l. This toxicity is within a factor of two of what would be expected if the principal toxic agent (in addition to CO) were hydrogen fluoride or carbonyl fluoride (Clarke et al., 1992).

Kinetic studies on fluorinated dibenzo-p-dioxins and dibenzofurans revealed a rapid elimination, suggesting a much lower toxicity than the corresponding polychlorinated and polybrominated congeners. The concentration in the thymus of several 2,3,7,8-fluorinated PFDD/PFDF exceeded that in hepatic tissue greatly. An organotropy quite different from that of the other polyhalogenated congeners must be expected, immunosuppressive effects presumably being the predominant ones (Herzke et al., 2002).

In rat hepatocytes a primary culture induction of CYP4501A1-catalyzed EROD activity could be demonstrated, indicating that 2,3,7,8-TFDD activates the dioxin receptor. In rat hepatocyte cultures similar EC50 values were found for 2,3,7,8-TCDD and 2,3,7,8-TFDD (Weber et al., 1995).

**Degradation in the environment**
PTFE is highly resistant to chemical and physical degradation and has almost no water absorption and is characterised as weather resistant in 20 years Florida weather (http://www2.dupont.com/Teflon_Industrial/en_US/tech_info/techinfo_compare.html).

**Emissions**
Herzke (1998) described the formation of fluorinated dibenzo-o-dioxins and furans (PFDD/F) during the combustion of PTFE at 800°C. Several PFDD/F were detected in the ash of aluminium production facilities ranging from mono- to hexafluoro dioxins and furans.

**Monitoring data**
Modelling indicates that the thermolysis of fluoropolymers in industrial and high-temperature consumer applications (ovens, non-stick cooking utensils and combustion engines) is likely to be a significant source of trifluoroacetate in urban rain-water (similar to 25 ng/L, as estimated for Toronto, Canada) (Ellis et al., 2001).

**Evaluation of need for screening**
Little is known about the formation of condensed fluorinated compounds during combustion and thermal processes involving fluoropolymers. Aromatic fluorocarbons are more volatile, stable and surface active than their chlorinated or brominated homologues and are therefore expected to be airborne and/or adsorbed to particles. It is therefore suggested to monitor indoor and outdoor air of municipal waste incineration plants, fluoropolymer production sites and aluminium smelters (because of their extensive use of fluorinated compounds under high temperatures.

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Analyses

PFDD/F analysis can be carried out in analogy to PCB analysis with GC/EI-MS as detection method (Weber et al., 1995; Herzke, 1998; Herzke et al., 2002).
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3 Phosphororganic flame retardants (PFR)

3.1 Introduction

By 2001, the world-wide usage of flame retardants was estimated at 1,217,000 tonnes of which 186,000 tonnes were organophosphate based (Davenport et al., 2002). With 15% of the world consumption of flame retardants, organic phosphorus containing flame retardants (PFR) represent a comparable market volume to the brominated flame retardants (BFR) (Danish Environmental Protection Agency, 1999). While the BFR are in the focus of scientific research and public attention little information is available on phosphor containing organic flame retardants and plasticisers.

BFR (Figure 16) are often used as alternatives to brominated flame retardants. Different phosphor containing flame retardants can be either simply mixed into plastics or be reactive, chemically bound into the plastic molecules during polymerisation. This depends on the properties required of the plastic and flame retardancy. We can distinguish halogenated and non-halogenated PFR. The majority of the phosphorus organic compounds have been on the market since the 1950s (Freudenthal and Henrich, 2000). Many of them have consequently not been studied according to modern, more rigorous standards (Hedemalm et al., 2000). Little data exists about degradation and end-of-life issues, like deposition, mobility, long-term effects or bioaccumulation.

PFR are not used in the same variety of applications as BFR, but within a much more specific operational area. Because of their physical-chemical characteristics they also function as plasticisers, broadening their field of application. Halogenated (chlorine, bromine) PFR combine the flame retarding properties of both the halogenated and the phosphorus groups. The insertion of halogen in the PFR compounds results in reduced vapour pressure and water solubility. This contributes to the retention of the retardant in the polymer but also reduces the biodegradability of these compounds (WHO, 1997). To impart durable flame resistance to cellulose in e.g. textiles, PFR are the material of choice (Green, 1996a; Green, 1996b). PFR are used as lubricants for industrial air compressors and gas turbines, and also as pigment dispersants and wood preservatives (Boethling and Cooper, 1985). Chlorinated phosphate esters are mainly used in polyurethane foam whereas aryl phosphates are present in the plastic parts of electrical and electronic equipment and in PVC-based
products working as flame retardants and plasticisers. Car and computer industries represent a growing marked for PFR. The increasing strictness in regulating the use of brominated flame retardants as e.g. brominated diphenylethers, and the stronger demands on fire safety will cause a further increase of the use of PFR (WHO, 1997).

A study by Orango AB (Hedemalm et al., 2000) concluded that the acute toxicity of phosphorus substances is generally higher than that of brominated compounds and that some phosphorus substances have adverse long-term effects such as mutagenicity and/or neurotoxicity. Tris(2,3-dibromo-propyl)-phosphate, for example was the most widely used halogenated PFR, until it was withdrawn from the market in many countries, due to its carcinogenic properties in animals (Green, 1996a). The European Commission Regulation (EC) No 2268/95 of 27 September 1995 concerning the second list of priority substances undertook the evaluation and control of risks of tris(2-chloroethyl) phosphate (TCEP) (Council Regulation (EEC) No 793/93). In addition tris(2-chloroisopropyl) phosphate (TCPP), tris(1,3-dichloro-2-propyl) phosphate (TDCP) and 2,2-bis(chloromethyl)-trimethylbis(2-chloroethyl) phosphate appeared on the fourth priority list established in October 2000 (European Chemicals Bureau. Commission Regulation (EC) No 2364/2000 of 25 October 2000 concerning the fourth list of priority substances as foreseen under Council Regulation (EEC) No 793/93. http://ecb.jrc.it/).

In addition, TCPP, TCDP and TCEP are included in the list of EU High Production Volume Chemicals.

PFR have come under intense environmental scrutiny, due to their acute toxicity to algae, invertebrates and fish (Danish Environmental Protection Agency, 1999). A wide range of biological effects of organophosphate esters has been reported, indicating substantial differences between the various organic phosphates (Carlsson et al., 2000). They are subject of several local risk analysis initiatives such as the National Academy of Science report, Australian Priority Existing Chemicals assessment report, UBA and the risk assessment by EU with finalisation 2006/2007.

There are no known restrictions (other than those based on voluntary eco labelling initiatives) on the use of halogenated phosphate esters as flame retardants anywhere in the world (http://www.cefic-efra.com).

PFR are ubiquitous substances that appear to be present in all types of indoor environments, and exposure to them seems to depend on the construction materials and products used in the buildings.

Identified sources include upholstered furniture, floor coverings and polishes, and acoustic ceilings. In premises that are subject to strict fire safety standards, such as public buildings, the exposure is higher than in other buildings (Hartmann et al., 2004; Marklund et al., 2005a).

Emission of PFR from specific products may lead to significant exposure through inhalation of indoor air and ingestion of dust.
3.2 Tris(2-chloroethyl) phosphate (TCEP)

TCEP is used in the manufacture of polyester resins, polyacrylates, polyurethanes and cellulose derivatives. However, TCEP is currently being replaced by other flame retardants, primarily TCPP, and is no longer produced in Europe (Marklund et al., 2005a).

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
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<tbody>
<tr>
<td>TCEP</td>
<td>Tris-(2-chloroethyl)phosphate</td>
<td>115-96-8</td>
</tr>
</tbody>
</table>

Figure 17: Molecular structure of TCEP.

Toxicological data

No reports are found which indicate that TCEP is associated with human toxicity. TCEP has a moderate acute oral toxicity in mice and rats. Different sources in the literature have reported an oral LD50 range of approximately 400-2000 mg/kg (Table). In a thorough toxicity and carcinogenesis study performed by The National Toxicology Program (Anonymous, 1991) the most pronounced findings were neuronal necrosis in hippocampus and thalamus of rats. This was primarily observed in rats receiving high doses of TCEP (175 and 350 mg/kg/day for 16 weeks or 44 mg/kg/day for 2 years), which also experienced increased mortality. In the 2-year study evidence for carcinogenic activity, as shown by increased incidences of renal tubule adenomas, were detected. TCEP is, however, not mutagenic and regarded as not classifiable as a carcinogen to humans coming in contact with TCEP via consumer products. (WHO, 1997;WHO, 1998).

However, in 2005 the EU risk assessment raised concerns about the carcinogenic properties of TCEP on humans having occupational exposure to TCEP. Dermal contact and inhalation should be avoided. A level below 0.2 mg/m³ TCEP is recommended for carcinogenicity inhalation exposure at the workplace. The establishment of an occupational exposure limit for TCEP is also recommended. Exposure to skin contact should be limited to 2 mg/person/day (EU, 2005).

TCEP is recommended classified in 31 ATP; Carc.cat.3; R40 Repr. Cat 2; R60, Xn; R22 N; R51-53 (SFT, 2007, pers. communication).

Modest to low acute toxicity to fish is indicated in the EU risk assessment, 2005. TCEP concentrations around 10 mg/L are suspected as a threshold level for sublethal effects on sensitive fish like trout (EU, 2005).

In the overall conclusions regarding risk for the all life cycle steps, to all environmental compartments, the EU expressed no need for further information and/or testing and for risk reduction measures beyond those which are being applied already (EU, 2005).
Degradation in the environment

TCEP is considered as very stable both for oxidation and degradation by chlorine attack (Westerhoff et al., 2005). No degradation in sewage treatment plants were detected (Meyer and Bester, 2004). TCEP is considered as non biodegradable by the EU (EU, 2005).

Use in Norway

Norway is listed in the database SPIN (Substances in Preparation in the Nordic countries) as using 1285 tonnes of TCEP in products in 2003, exceeding the other Scandinavian countries by far (EU, 2005). Manufacture of rubber and plastic products and manufacture of chemicals and chemical products were identified as the main fields of application.

Emissions

Releases of TCEP are expected during industrial use and manufacturing. Since TCEP is used as an additive, which is not chemically bound to the polymer, it can migrate to the surface of the products and subsequently be released during use and disposal of products (EU, 2005).

TCEP can be emitted by sewage treatment plants, because of lacking methodology to remove chlorinated PFR from the effluents (Andersen et al., 2004; Meyer and Bester, 2004).

The flame retardant tris(2-chloroethyl)-phosphate (TCEP) is widely used in indoor housing materials and can be detected in household dust and indoor air. Therefore, human exposure to the two organo-phosphates may be expected.

Monitoring data

TCEP was one of the most frequently detected compounds in water samples from a network of 139 streams across 30 states in the USA during 1999 and 2000 (Kolpin et al., 2002). TCEP was detected in surface waters in Japan as well (Fukushima et al., 1992).

TCEP has been analysed in several rivers and sewage treatment plant (STP) effluents in Germany. The concentrations in the River Ruhr were 13-130 ng/L TCEP. The STP effluents exhibit concentrations up to 130 ng/L TCEP. The River Oder on the eastern boarder of Germany, showed mean values of TCEP between 30 to 282 ng/L. The main sources for the load of organophosphates are sewage treatment plants, but not all contribute proportionally to the number of inhabitants they serve (Fries and Puttmann, 2001; Fries and Puttmann, 2003; Andersen et al., 2004; EU, 2005).

TCEP was detected in all precipitation samples collected in Ireland, Poland, Sweden in concentrations up to 20 ng/L (Laniewski et al., 1999; EU, 2005).

TCEP was present in 85% of a total of 983 domestic dust samples. Since TCEP residues in domestic dust are assumed to be condensates arising from primary sources, spot check analysis of various indoor materials was performed. The results show that soft foams, paints and

<table>
<thead>
<tr>
<th>Organism</th>
<th>Test Type</th>
<th>Route</th>
<th>Reported Dose</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouse</td>
<td>LD50</td>
<td>oral</td>
<td>1866 mg/kg</td>
<td>National Technical Information Service. Vol. AD-A067-313,</td>
</tr>
<tr>
<td>Rat</td>
<td>LD50</td>
<td>oral</td>
<td>1230 mg/kg</td>
<td>Eldefrawi AT et al.; Bulletin of Environmental Contamination and Toxicology. Vol. 17, Pg. 720, 1977.</td>
</tr>
<tr>
<td>Fish</td>
<td>LC50</td>
<td></td>
<td>170 mg/L</td>
<td>(Yoshioka and Ose, 1993; EU, 2005)</td>
</tr>
</tbody>
</table>
wallpapers contained TCEP. Moreover, TCEP can also be detected in indoor air in concentrations up to 6,000 ng/m³ (Ingerowski et al., 1975).

Marklund et al. (2005a) discovered TCEP and other PFR in several indoor environments. The total amounts of PFR in the air samples ranged between 36 and 950 ng/m³. TCPP and TCEP being the most abundant (0.4 to 730 ng/m³). Public buildings tended to have about 3-4 times higher levels of PFR than domestic buildings. Based on estimated amounts of indoor air inhaled and dust ingested, adults and children in the sampled environments would be exposed to up to 5.8 µg/kg/day and 57 µg/ kg/day total PFR respectively.

**Evaluation of need for screening**

In view of the uncertain toxicological implications of these substances, indoor air sampling, both active and passive, are suggested, covering the particle bound chemicals as well (Ingerowski et al., 1975).

For further exposure assessments it is important to consider concentrations in dust, especially since children tend to be relatively more exposed through ingestion of dust than adults.

Exposure by inhalation of indoor air is highest for the chlorinated PFR, TCEP and TCPP. It would therefore be important to investigate individuals who are occupationally exposed to PFR, especially since data regarding their health effects are scarce. Such exposure assessments should include both personal air sampling and analyses of blood and urine from the subjects (Swanson et al., 2004; Marklund et al., 2005a).

To evaluate the environmental contamination with TCEP a similar approach as used for screening for brominated flame retardants (BFR) is suggested: a broad screening of (1) some sewage treatment plants and run-off from landfills in order to detect emissions to water, (2) marine and fresh water sediments from potential source area as Drammenselva estuary (car demolition plants), Ålesund area (upholstered furniture industry) and (3) a combination of moss samples and passive air samples taken close to major cities in order to detect emissions to air.

**Analyses**

Air samples were collected on solid-phase extraction (SPE) columns (Isolute NH₂, 25 mg, 1 ml, supplied by IST, Mid Glamorgan, UK), which have been shown to be suitable adsorbents for PFR. After sampling the SPE columns were eluted with dichloromethane. Quantitative analysis can be performed using a GC-NPD or GC/MS system (Carlsson et al., 1997; Marklund et al., 2005a).

Sludge samples were extracted in a Soxhlet apparatus. The resulting extracts were cleaned up and applied to gel permeation column (GPC) (Bester, 2005).

Quantitative analysis can be performed using a GC-NPD or GC/MS systems (Carlsson et al., 1997; Marklund et al., 2005a).
3.3 Tris(1-chloro-2-propyl) phosphate (TCPP)

TCPP is a colourless liquid used mainly as an additive in polyurethane foams as a substitute for TCEP (WHO, 1998). TCPP is manufactured from polyethylene and phosphorous oxychloride. The annual worldwide demand exceeded 40 000 tonnes in 1997 (Bester, 2005). There are four producers of TCPP in Europe (Supresta, Lanxess, BASF and Albemarle). Total EU production in the year 2000 was 36 000 tonnes (EU, 2006c). Most TCPP is used as a flame retardant in the production of polyurethane for use in construction and furniture. TCPP is not a suitable replacement for pentabromodiphenylether but the increase in TCPP consumption is linked mostly with the decline in TCEP use and increase in the market for polyurethane generally (EU, 2006c).

<table>
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<tr>
<td>TCPP</td>
<td>Tris(1-chloro-2-propyl) phosphate</td>
<td>13674-84-5</td>
</tr>
</tbody>
</table>

![Figure 18: Molecular structure of TCPP.](image)

**Toxicological data**

The EU risk assessment 2006 states, that it is unlikely for TCPP to exhibit a chronic toxicity to fish at <1 mg/L and suggests no classification for the compound (EU, 2006c). Inhibition of soil nitrogen transformation by soil microorganisms was examined in a study with TDCP. A 28-day NOEC of 128 mg/kg wet weight was determined in the test. Due to the structural similarity of TDCP to TCPP, the EU risk assessment agreed assume similar long-term effects for TDCP to TCPP (EU, 2006c).

No reports are found which indicate that TCPP is associated with a toxicity to humans. TCPP has a relatively low acute oral toxicity and oral LD50 to rats range between approximately 1000-4000 mg/kg. The aquatic toxicity is also low, with lethal concentrations from approximately 100 mg/L for the fathead minnow, *Daphnia magna* and algae (WHO, 1998). The EU concluded that there was no need for further information and/or testing and no need for risk reduction measures beyond those which are already being applied. Due to the lack of bioaccumulation of TCPP, the EU concluded that there were no risks for secondary poisoning of the marine environment. Regarding human health, the data presented were consistent with the classification R22 (harmful if swallowed). This is based on the fact that the majority of LD50 values determined from acute oral toxicity studies were <2000 mg/kg. It was also proposed to classify with Carc. Cat. 3 R40 (Limited evidence of a carcinogenic effect). This classification is based on the substances structural similarity to TCEP and TDCP (EU, 2006c).

**Characteristic of the compound**

Molecular formula: $C_{9}H_{18}Cl_{3}O_{4}P$  
Melting point: -40$^\circ$C  
Vapour pressure: $2.02 \times 10^{-5}$ mm Hg  
Water solubility: 1200 mg/L  
Log $K_{ow}$: 2.59
Organism | Test Type | Route | Reported Dose | Reference \\
--- | --- | --- | --- | --- \\
Invertebrates | LC50 |  | 63 mg/L | (Marklund et al., 2005a; EU, 2006c) \\
Earthworm | LC50 |  | 97 mg/kg | (EU, 2006c) \\
Mouse | LD50 | Intravenous | 56 mg/kg | U.S. Army Armament Research & Development Command, Chemical Systems Laboratory, NIOSH Exchange Chemicals. Vol. NX#05768, \\
Rat | LD50 | Oral | 1500 mg/kg | National Technical Information Service. Vol. OTS0557521,

Degradation in the environment

TCPP is considered as very stable both for oxidation and degradation by chlorine attack (Westerhoff et al., 2005). No degradation in sewage treatment plants (Meyer and Bester, 2004). Readily metabolism in fish (WHO, 1998). The EU risk assessment evaluates TCPP as: inherently biodegradable, not fulfilling the criteria of the risk assessment. Therefore TCPP is considered to meet the screening criteria as persistent/very persistent (EU, 2006c).

Use in Norway

The SPIN database showed that 50 tonnes of TCPP were used in Norway in 2001 for the manufacture of chemicals, rubber and plastic and also construction (EU, 2006c).

Emissions

Since it is a liquid at room temperature and is used as an additive, TCPP has a high potential of diffusing out of the treated material (EU, 2006c).

Identified sources include upholstered furniture, floor coverings, floor polishes and acoustic ceilings. In premises that are subject to strict fire safety standards, such as public buildings, the exposure is higher than in other buildings (Hartmann et al., 2004; Marklund et al., 2005a).

Kemmlein et al. (2003) found TCPP to be one of the most commonly emitted organophosphate flame retardants in polyurethane foam applications under constant environmental conditions (23°C, 50% RH) using a fixed sample surface area and controlled air flow rates. Depending on the sample type, area-specific emission rates (SERa) of TCPP varied between 20 ng/m²/h and 140 µg/m²/h.

The EU calculated in-service loss rates for TCPP from furniture and automotive foam. A loss to air of 0.25% was calculated for indoor use and 0.75% loss to air for outdoor use, associated with volatile releases from the articles themselves. An annual rate of release of 9.6 x 10⁻³ % per year to air is proposed, accounting for the finding that for TCPP, 40% of the substance is available for release, which is much higher compared to TDCP (EU, 2006c).

In addition, TCPP can be emitted by sewage treatment plants, as there is a lack of methodology to remove chlorinated PFR from the effluents (Andresen et al., 2004; Meyer and Bester, 2004; Bester, 2005). In addition, because of its relatively high water solubility, leaching of TCPP from landfills is a possibility.

Traffic is assumed to be a source of organophosphorus flame retardants and plasticisers (OPs) in the outdoor environment (Marklund et al., 2005b).

Monitoring data

TCPP was also detected in surface waters in Japan (Fukushima et al., 1992).
TCPP has been analysed in several rivers and sewage treatment plant (STP) effluents in Germany. The concentrations in the River Ruhr were 20-200 ng/L TCPP. The STP effluents had concentrations up to 400 ng/L TCPP. The main sources for the load of organophosphates were sewage treatment plants, but not all contributed proportionally to the number of inhabitants they served (Andresen et al., 2004).

Snow samples collected in northern Sweden at a road intersection and an airport indicated that traffic is a source of organophosphorus flame retardants and plasticisers (OPs) in the outdoor environment. TCPP dominated in the snow samples collected near a road, with levels of 170, 130, and 110 ng/kg at distances of 2, 100, and 250 m away from the road. TCPP was possibly emitted from the interior of cars via their ventilation systems (Marklund et al., 2005b).

TCPP was found in 60–90% of 436 domestic dust samples (with levels ranging from 0.1 to 375 mg/kg). Since TCPP residues in domestic dust are thought to be condensates arising from a primary sources, spot check analysis of various indoor materials was performed. The results showed that insulation and sealant foams contain high levels of TCPP (Ingerowski et al., 1975).

Marklund et al. (2005a) discovered TCPP and other PFR in several indoor environments. The total amounts of PFR in the air samples ranged between 36 and 950 ng/m³; TCPP and TCEP being the most abundant (0.4 to 730 ng/m³). Public buildings tended to have about 3-4 times higher levels of PFR than domestic buildings. Based on estimated amounts of indoor air inhaled and dust ingested, adults and children in the sampled environments would be exposed to up to 5.8 µg/kg/day and 57 µg/kg/day total PFR respectively.

Evaluation of need for screening

As for TCEP, human exposure may be expected. In view of the uncertain toxicological implications of these substances, it is suggested that indoor air sampling be carried out, both active and passive, and also covering the particle bound chemicals (Ingerowski et al., 1975).

For further exposure assessments it is important to consider concentrations in both air and dust, especially since children tend to be relatively more exposed through ingestion of dust than adults.

Exposure by inhalation of indoor air is highest for TCEP and TCPP. It would therefore be relevant to investigate individuals who are exposed to these PFR in their workplace, especially since data regarding their health effects are scarce. Such exposure assessments should include both personal air sampling and analyses of blood and urine from the subjects (Swanson et al., 2004; Marklund et al., 2005a).

Since its potential for human exposure via drinking water, TCPP is recommended to be measured in drinking water sources situated near landfills and PUR applying plants.

To evaluate the environmental contamination with TCPP a similar approach as used for screening for brominated flame retardants (BFR) is suggested: a broad screening of (1) some sewage treatment plants and run-off from landfills in order to detect emissions to water, (2) marine and fresh water sediments from potential source area as Drammenselva estuary (car demolition plants), Ålesund area (upholstered furniture industry) and (3) a combination of moss samples and passive air samples taken close to major cities in order to detect emissions to air.
Analyses

Air samples were collected on solid-phase extraction (SPE) columns, which have been shown to be suitable adsorbents for PFR. After sampling the SPE columns were eluted with DCM. Quantitative analysis can be performed using a GC-NPD or GC/MS systems (Carlsson et al., 1997; Marklund et al., 2005a).

Sludge samples were extracted in a Soxhlet apparatus. The resulting extracts were cleaned up with silica SPE cartridges and applied to gel permeation column (GPC) (Bester, 2005).

Quantitative analysis can be performed using a GC-NPD or GC/MS systems (Carlsson et al., 1997; Marklund et al., 2005a).
3.4 Tris(1,3-Dichloro-2-Propyl)phosphate (TDCP(P))

TDCP is a viscous colourless liquid, used in a range of plastic foams, resins and latexes (WHO, 1998). The annual worldwide demand exceeded 8 000 tonnes in 1997 (Bester, 2005). TDCP is produced by the reaction of phosphorus oxychloride with an organic epoxide in the presence of a catalyst. Both Supestra and Albemarle have production sites for TDCP in Europe (Germany and UK). Total EU production was less than 10 000 tonnes in 2000. By far the most significant applications for TDCP is in the automotive and furniture industries. TDCP operates in the same market place as TCPP. Owing to the higher price of TDCP, TCPP is mostly the preferred flame retardant. Similar to TCPP, TDCP is applied as an additive to the materials, mainly polyurethane.

TDCP appears to be relatively persistent and human health concern arises because of the voluntarily classification of TDCP as category 3 carcinogen by the manufactures (EU, 2006b). The EU risk assessment confirms the classification N R51-53 (toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment) (EU, 2006b).

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>TDCP(P)</td>
<td>Tris(1,3-Dichloro-2-Propyl)phosphate</td>
<td>13674-87-8</td>
</tr>
</tbody>
</table>

Figure 19: Molecular structure of TDCP.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Test Type</th>
<th>Route</th>
<th>Reported Dose</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabbit</td>
<td>LD50</td>
<td>skin</td>
<td>&gt; 23700 mg/kg</td>
<td>United States Environmental Protection Agency, Office of Pesticides and Toxic Substances. Vol. 8EHQ-1280-0401S,</td>
</tr>
<tr>
<td>Rat</td>
<td>LD50</td>
<td>oral</td>
<td>1850 mg/kg</td>
<td>Bromatologia i Chemia Toksykologiczna. Vol. 9, Pg. 141, 1976.</td>
</tr>
<tr>
<td>Fish</td>
<td>LC50</td>
<td>oral</td>
<td>1.1 mg/L</td>
<td>(EU, 2006b)</td>
</tr>
</tbody>
</table>

TDCP is not volatile, but much more water-soluble compared to TCEP and TCPP.
**Toxicological data**

The acute oral LD50 on mice and rats of TDCP ranges between approximately 2000-3000 mg/kg. TDCP is recognised as slightly irritant to rabbit skin. Pregnant rats were given 25-400 mg/kg TDCP/ day on days 7 to 15 of gestation. NOEL and LOEL for maternal toxicity were estimated to 100 mg/kg and 200 mg/kg body weight per day, respectively, and the NOEL and LOEL for fetotoxicity were 200 mg/kg and 400 mg/kg body weight per day, respectively (WHO, 1998). Bacterial tests shows that TDCP has *in vitro* mutagenic potential, primarily after metabolic activation by S9 from PCB activated livers.

In a two year study, groups of 50 male and 50 female Sprague-Dawley rats received approximately 0, 5, 20 and 80 mg TDCP/kg per day by dietary administration for 2 years (WHO, 1998). Reduced body weight and increased male mortality, as well as increased liver, kidney and thyroid weights were observed in the high exposure group. As stated in the WHO, 1998 report it was further concluded that TDCP is carcinogenic at all exposure levels that were tested in both sexes of rats based on the increased occurrence of liver carcinomas. Tumours in other organs were also found.

A similar study was performed by Freudenthal and Henrich (2000), which a chronic toxicity and carcinogenicity bioassay was conducted in Sprague-Dawley rats to determine the toxicological and carcinogenic potential of TDCP after repeated exposure. Four groups of animals, each consisting of 60 male and 60 female rats, received via their diet a daily dose of either 0, 5, 20, or 80 mg TDCP per kg body weight for up to 24 months. Liver, kidneys, testes, and adrenal glands were examined from all animals. Mortality was significantly higher and body weights were significantly lower in the high-dose group when compared to control animals. Haemoglobin, haematocrit, and total erythrocyte values were decreased in the high-dose animals. Microscopic examination revealed a higher incidence of benign neoplasms and non-neoplastic alterations in several organs of the mid and high dose animals. The no-observed-adverse effect level (NOAEL) for chronic toxicity and neoplastic activity was the dietary dose of 5 mg/kg/day (Freudenthal and Henrich, 2000).

In rats, TDCP is absorbed after oral or dermal exposure and rapidly distributed throughout the body with the highest concentration being observed in kidney, liver, and lung (Nomeir et al., 1981; Hughes et al., 2001). It was metabolised and rapidly excreted in urine and faeces and expired as CO2; 80% or more of the TDCP dose was eliminated 24 h after administration and the major urinary metabolite is bis(1,3-dichloro-2-propyl)-phosphate.

The aquatic toxicity of TDCP is higher than for TCPP with lethal concentration from approximately 1 mg/L for rainbow trout and 5 mg/L for *Daphnia magna* (WHO, 1998). The EU risk assessment confirms the classification N R51-53 (toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment). There is no evidence of degradation sufficient to remove the need for the classification (EU, 2006b).

Little data of effects on humans are available. Two cohort studies on workers employed at a TDCP manufacture plants were performed. Some minor respiratory diseases among workers were revealed, but no differences in any if the other clinical parameters investigated such a cancer, heart diseases and mortality (WHO, 1998).

TDCP appears to be relatively persistent and human health concern arise because of the voluntarily classification of TDCP by the manufacturers as category 3 carcinogen. The EU risk assessment confirms the
classification N R51-53 (toxic to aquatic organisms; may cause long-term adverse effects in the aquatic environment) and as Carcinogenic Category 3 R40 (Limited evidence of a carcinogenic effect) (EU, 2006b).

The EU risk assessment 2006, concluded, with some minor exceptions, that at present there is no need for further information and/or testing and no need for risk reduction measures beyond those which are already applied (EU, 2006b).

Degradation in the environment

TDCP is considered as very stable both for oxidation and degradation by chlorine attack (Westerhoff et al., 2005). No degradation occurs in sewage treatment plants (Meyer and Bester, 2004). TDCP is readily metabolised in fish (WHO, 1998). A general lack of ready biodegradability was noted by the EU risk assessment (EU, 2006b).

TDCP is metabolised and rapidly excreted in urine and faeces by rats 24 h after administration and the major urinary metabolite is bis(1,3-dichloro-2-propyl)phosphate (Nomeir et al., 1981; Hughes et al., 2001). A bioconcentration factor of 45 L/kg for fish is suggested for TDCP by the EU risk assessment, implying rapid metabolism and hence no meeting of the bioaccumulation criterion (EU, 2006b).

Use in Norway

No data available.

Emissions

Since TDCP is applied as an additive, it may be subject to volatilisation or leaching from the polymer matrix during lifetime and disposal. A total of 2% release over the lifetime of the article was assumed, by the EU, for most life cycle stages, associated with physical erosion of the polymer.

Identified sources include upholstered furniture, floor coverings, floor polishes and acoustic ceilings. In premises that are subject to strict fire safety standards, such as public buildings, the exposure is higher than in other buildings (Hartmann et al., 2004; Marklund et al., 2005a).

In-service loss rates from furniture and automotive foam were calculated for TDCP by the EU. The loss to air of 0.01% and to wastewater of 0.01% was calculated for indoor service and 0.03% loss to wastewater for outdoor service, associated with volatile releases from the articles themselves. An annual rate of release of 10% per year to air is proposed, accounting for the finding that for TDCP, only 10% of the substance is available for release (EU, 2006b).

Monitoring data

The low volatility and relatively high coefficient suggests that most TDCP found in the atmosphere will adsorb to particulate matter, which will subsequently be immobilised in soil by precipitation (EU, 2006b).

TDCP has been analysed in several rivers and sewage treatment plant (STP) effluents in Germany. The concentrations in the River Ruhr were about 50 ng/L TDCP. The STP effluents exhibit concentrations up to 120 ng/L TDCP. The main sources for the load of organophosphates are sewage treatment plants, but not all contribute proportionally to the number of inhabitants they serve (Andresen et al., 2004). No elimination of TDCP was observed in any of the sampled STPs (Meyer and Bester, 2004).

Marklund et al. (2005a) discovered TDCP and other PFR in several indoor environments. The total amounts of PFR in the air samples ranged between zero and 150 ng/m³ TDCP. Public buildings tended
to have about 3-4 times higher levels of PFR than domestic buildings. Based on estimated amounts of indoor air inhaled and dust ingested, adults and children in the sampled environments would be exposed to up to 5.8 µg/kg/day and 57 µg/kg/day total PFR respectively.

The same author found 230 ng/kg TDCP in snow collected along a road in northern Sweden (Marklund et al., 2005b).

Landfill site were sampled in Japan in 1995 by Yasuhara et al. (1999). Up to 5,500 ng/L TDCP was found in the leachate of the landfill site.

**Evaluation of need for screening**

Air and dust samples from the indoor living environment should be monitored as well as occupational exposure.

Water, soil and sediment are all significant for the distribution of TDCP. Accordingly, waste water and sediments from landfills and PUR production and application industries are recommended for screening (Ålesund; furniture plants).

Since its potential for human exposure via drinking water, TDCP is recommended to be measured in drinking water sources situated near landfills.

**Analyses**

Air samples were collected on solid-phase extraction (SPE) columns (Isolute NH₂, 25 mg, 1 ml, supplied by IST, Mid Glamorgan, UK), which have been shown to be suitable adsorbents for PFR. After sampling the SPE columns were eluted with DCM. Quantitative analysis can be performed using a GC-NPD or GC/MS systems (Carlsson et al., 1997; Marklund et al., 2005a).

Sludge samples were extracted for 6 h with ethyl acetate in a Soxhlet apparatus. The resulting extracts were cleaned up with 1 g silica SPE cartridges and injected to gel permeation column (GPC) (Bester, 2005).

Quantitative analysis can be performed using a GC-NPD or GC/MS systems (Carlsson et al., 1997; Marklund et al., 2005a).
3.5 Tetrakis(2-chloroethyl)-
dichloroisopentylidiphosphate
(V6)

V6 is manufactured by the reaction of
alkylene oxides and phosphorus chlorides
in the presence of catalysts. Albemarle is
the only producer of V6 in Europe (UK),
with a production of less than 5,000 tonnes

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS-Nr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>V6</td>
<td>Tetrakis(2-chloroethyl)dichloroisopentylidiphosphate</td>
<td>38051-10-4</td>
</tr>
</tbody>
</table>

The sole use of V6 is as a flame
retardant. The main downstream use of V6
is in the production of flexible polyurethane foam, used in the automotive and
furniture industries. The flame retardant is
not chemically reacted, but physically
bound within the matrix and therefore has
the potential for migration.

One of the main impurities of V6 is the
PFR TCEP (4.5-7.5%). V6 is a speciality
product for use in the same market as
TCPP and TDPC.

A classification as “not dangerous for
the environment” was agreed in the EU in

![Figure 20: Molecular structure of Tetrakis(2-chloroethyl)dichloroisopentylidiphosphate (V6).](image)

**Toxicological data**

Significantly increased absolute (35%
and 78%) and relative (30% and 73%)
weight was noted in female rats fed with
V6 (150 mg/kg/day) and male rats (600
mg/kg/day) (absolute: 52% and relative:
68%). These findings correlated with
evidence of hepatocellular hypertrophy
among females at 150 mg/kg/day and
findings of slight to marked centrilobular
hypertrophy at 600 mg/kg/day in all males
and females. Based on the data from this
study the NOAEL was calculated as
15 mg/kg/day (CLASSIFICATION AND
LABELLING OF DANGEROUS
SUBSTANCES by the Commission of the
European Communities DG XI, Form of
Dangerous Substances in order to update
September 2004; ECBI/130/04).

V6 is non-irritant to skin and eyes and is
not expected to be a respiratory irritant.

The measured toxicity of V6 to fish and
invertebrates is higher than that of TCEP by
a factor of 2 and 8 respectively. Bearing in
mind, that TCEP is an impurity of V6, a
contribution to the toxicity of V6 is
possible. Data presented in the EU risk
assessment are consistent with the
classification R52-53 for the environment
(harmful to aquatic organisms, may cause
long-term adverse effects in the aquatic
environment). Fish, *Daphnia* and algae
acute *LC₅₀* values all fall in the range 10 to
100 mg/L, and there is no evidence of
significant degradability in standard tests.
Long-term NOEC values for *Daphnia* and
algae are greater than 1 mg/L, however no
value is available for fish. Therefore the
existence of long-term hazard cannot be
ruled out on the basis of the present data.
Based on the data presented in this risk
assessment report, it was proposed not to
classify V6 as having a human health
effect (EU, 2006a).
The low $K_{ow}$ does suggest that V6 will not accumulate in organisms over long periods. Bioaccumulation is not expected (EU, 2006a).

**Degradation in the environment**

V6 is considered as very stable both for oxidation and degradation by chlorine attack (Westerhoff et al., 2005). No degradation in sewage treatment plants occurs (Meyer and Bester, 2004). Biological degradation was detected during a EU risk assessment (37% in 28 days) (EU, 2006a). On the other hand, V6 was characterised in the same report as not significantly biodegradable on the basis of freshwater tests. It is also considered to be persistent in the marine environment (EU, 2006a).

**Use in Norway**

No data available.

**Emissions**

Since V6 is an additive flame retardant it may volatilise or leach from the polymer matrix during the lifetime and disposal of the consumer product. Disposal to landfill is likely the most significant route of disposal of flexible foam.

V6 is characterised as more adsorbing, with a higher molecular weight containing an additional phosphate group and more chlorine, compared to TCPP, hence a lower volatile loss is expected (10%) (EU, 2006a). In service loss rates from furniture and automotive foam were calculated for V6 by the EU. The loss to air of 0.01% and to wastewater of 0.01% was calculated for indoor service over lifetime and 0.03% loss to wastewater per year for outdoor service, associated with volatile releases from the articles themselves. An annual rate of release of $1 \times 10^{-3}$ % per year to air is proposed, accounting for the finding that only 10% of the substance is available for release (EU, 2006a).

Occupational exposure to V6 may occur during the:

- Manufacture of V6
- Manufacture and handling of flexible PUR foam
- Manufacture of furniture
- Manufacture of automotive parts
- Production of re-bonded PUR foam

V6 inhalation exposure varies across the industry sectors. The highest inhalation exposure was estimated to be during the cutting of flexible PUR foam, with the reasonable worst case estimated to be 0.75 mg/m³ and the typical exposures estimated to be 0.05 mg/m³. During the production of V6, the typical inhalation exposure (8 hr TWA) is 30 µg/m³.

The current use pattern provided by industry indicates that most of the V6 produced in the EU in 2000 was used in the production of flexible PUR foam. Most of the V6 used in flexible foam is for the automotive industry, with some used in furniture. Consumers do not come in direct contact with these foams. The foam is only used such that it was enclosed and therefore it was concluded that exposure to consumers is negligible.
Evaluation of need for screening

Waste water and sediments from landfills and PUR production and application industries are recommended for screening.

Since its potential for human exposure via drinking water, V6 is recommended to be measured in drinking water sources situated near landfills.

Analyses

No information available.
References


EU. EU-Risk assessment; Tris(2-chloro-1-methylethyl)phosphate, TCPP. 2006c. EU.


Kemmlein S, Hahn O, Jann O. Emissions of organophosphate and brominated flame


Nomeir AA, Kato S, Matthews HB. The Metabolism and Disposition of Tris(1,3-Dichloro-2-Propyl) Phosphate (Fyrol Fr-2) in the Rat. Toxicology and Applied Pharmacology 1981; 57: 401-413.


3-nitrobenzanthrone (3-NBA) is as a nitro-oxy-PAH, a member of the group of polycyclic aromatic compounds (PAC). Non-substituted polycyclic aromatic hydrocarbons (PAH) are one of the most abundant sub-classes of PAC and since many PAH are carcinogenic they contribute to cancer risk. On the other hand, it is clear that PAH alone cannot explain all of the risk associated with ambient particles and especially nitrated polycyclic aromatic hydrocarbons (nitro-PAH) have been proposed to contribute significantly to the carcinogenicity of airborne particles.

In recent years, other more polar and very potent nitro-compounds have been detected and suggested to partly explain the remaining mutagenic activity. For example, Sera and coworkers (Sera et al., 1994) detected heterocyclic nitro-azaarenes in ambient air and diesel exhaust and Enya et al. (1997) detected 3-nitrobenzanthrone (3-NBA) in ambient air and diesel exhaust.

3-NBA and other nitro- and nitro-oxy-PAH abundantly exist in the particulate matters emitted from diesel and gasoline engines and also on the surface of airborne particulates. They are most likely formed during the combustion of fossil fuels as well as by the photoreaction of parent PAH with nitrogen oxides in ambient air (Enya et al., 1997).

Characteristics of the compound
CAS-nr.: 17117-34-9
Molecular formula: C₁₇H₉NO₃
Melting point: 256-257°C
Log Kᵗₒw: no data

3-NBA seems to be more persistent than non-substituted PAHs (Feilberg et al., 2002).

Figure 21: Chemical structure of 3-nitrobenzanthrone.

Toxicological data
The nitro-PAH 3-nitrobenzanthrone (3-NBA) is primarily known as a highly mutagenic substance through nitro-reduction to N-hydroxy-3-aminobenzanthrone yielding nonacetylated 3-DNA-ABA adducts, or by formation of acetylated 3-NBA adducts (Arlt et al., 2003). 3-NBA is activated by a range of biotransformation enzymes, such as CYP450, sulfotransferases and acetyltransferases (Arlt et al., 2003). DNA-adduct formation by 3-NBA, which is an indication of its mutagenic and carcinogenic
potential, happens both *in vivo* and *in vitro* (WHO, 2003).

Murata et al. (2006) exposed female rats to a single dose of 3-NBA (2 mg/kg) by intraperitoneal injection and observed adduct formation in all the tissues examined, indicating a systematically distribution of the chemical in the body. Similar is reported by Watanabe et al. (2005).

According to WHO-IARC, diesel exhaust is regarded as probably carcinogenic to humans (WHO, 1989). Several nitro- and nitro-oxy-PAHs are shown to be possible carcinogens (WHO 1989; WHO 2003). 3-NBA is found in diesel, and there are studies showing that it is carcinogenic in rats (Arlt, 2005). But even though it is highly mutagenic there are few evidences for it to be a human carcinogen (WHO 2003; Arlt, 2005). WHO, however, emphasizes that this chemical should be attributed to increased attention and that proper data is missing to make certain conclusions about its potential as a carcinogen (WHO, 1998 and 2003).

Very limited information about acute toxic effects of 3-NBA, others than its potential as a mutagen, is available. In an evaluation of 3-NBA Watanabe et al. (2005) exposed mice intraperitoneally to one single dose of 160 mg/kg without reporting any visible adverse effects but DNA-adduct formation.

In a recent report by Nagy et al. (2006), 1 mg 3-NBA was orally administered female rats. In addition to DNA-adduct formation they reported tissue damage of the GI tract, which included hemorrhages, loss of villous surface structure in the small intestine and intestine fragility. The tissue damage appeared not to be permanent, but indicates that 3-NBA may be acutely toxic.

**Degradation in the environment**

Feilberg at the Risø National Laboratory in Denmark studied the photodegradation of 3-NBA. The photostability of 3-NBA is comparable to 1-nitropyrene (1-NP) which means that 3-NBA is more stable than typical PAHs but less than the most persistent nitro-PAHs which can be exposed to atmospheric long-range transport (LRT) (Feilberg et al., 2002).

**Use in Norway**

There is no intentional use of 3-NBA in Norway or any other country. 3-NBA is a by-product of combustion processes, especially diesel fuel. In the laboratory the phototransformation of other PACs leads to 3-NBA. However, the environmental relevance of this source is uncertain (Enya et al., 1997; Feilberg et al., 2002).

**Emissions**

3-NBA was first found in diesel exhaust particles. The concentration in the exhaust particles was directly correlated to engine load (0.6 μg/g at 6 % load and 6.6 μg/g at 80 % load) (Enya et al., 1997). It was also detected in several samples of airborne particles taken at heavy traffic sites in Japan with a concentration range from 5 to 12 pg/m³ (Enya et al., 1997; Tang et al., 2004).

However, at a semi-rural area of Denmark (Risø), 3-NBA and other nitro-PAH were detected in about one-fourth of 31 samples of ambient air. The measured concentrations were in the range of 2-68 pg/m³ (Feilberg et al., 2002).

**Monitoring**

In contrast to PAH and nitro-PAH, 3-NBA is not included in any regular ambient air monitoring program neither in Norway nor in any other country.
Evaluation of need of screening

Arlt concluded that because of its widespread environmental presence, 3-NBA may represent not only an occupational health hazard but also a hazard for larger sections of the general population (Arlt et al., 2003; Arlt, 2005). For an accurate risk assessment more epidemiological studies on 3-NBA-exposed individuals and a broader monitoring of environmental levels of 3-NBA are required.

Measurements of 3-NBA and other relevant nitro- and nitro-oxy-PAH should preferably be performed at sites with a heavy contribution of traffic related pollution and occupational exposure. Since 3-NBA emissions seem to be correlated with engine load an ascending tunnel (like Vålerenga, nordgående) would be suitable for studying a worst-case scenario. A second urban site close to a major residential quarter could give an impression of the general human exposure to 3-NBA. Samples from a gas station could give information about occupational exposure. In addition, the importance of the emission from shipping traffic is not known at the moment and could easily be studied in the locked environment of the fjord area (like Geiranger fjord).

Ideally, the studies mentioned above should be performed with differentiation between the emission from light and heavy diesel engines.

Analyses

A recent review article described the most relevant methods for analysis of nitro-PAH in detail (Zielinska and Samy, 2006): The concentrations of nitro-PAH in typical ambient samples are very low (frequently in the low or sub picograms/m³ range), making their determination a particularly challenging task. Most of the analytical techniques employed for nitro-PAH analysis require a solvent extraction of the organic compounds from the complex environmental matrices and an extensive cleanup of the extracts prior to analysis.

Earlier the isolation of the nitro-PAH fraction was achieved with the use of a combination of normal-phase and reverse-phase liquid chromatography separation. Recently, solid-phase extraction (SPE) using an aminopropyl phase followed by the normal-phase liquid chromatography has been adopted for ambient air and particulate matter related samples. The separation and quantification of nitro-PAH can be performed by either gas or liquid chromatography combined with either mass spectrometry (GC/MS and LC/MS) or various other detection methods:

Gas chromatography coupled with low resolution mass spectrometry (GC/LRMS) (Phousongphouang and Arey, 2003), gas chromatography coupled with high resolution mass spectrometry (GC/HRMS) (Enya et al., 1997), gas chromatography coupled with tandem mass spectrometry (GC/MS/MS) (Feilberg and Nielsen, 2000;Feilberg et al., 2002) and liquid chromatography coupled with a fluorescence detector (HPLC/FD) (Murahashi et al., 2003) or chemi-luminescence detector (HPLC/CD) (Tang et al., 2004).

Most of these methods can be set up to include a wider range of nitro-PAH compounds.
References


Nagy E, Adachi S, Takamura-Enya T, Zeisig M, Moller L. DNA damage and acute toxicity caused by the urban air pollutant 3-nitrobenzantrone in rats: Characterization of DNA adducts in eight different tissues and organs with synthesized standards. Environmental and Molecular Mutagenesis 2006; 47: 541-552.


A large number of organotin compounds exists. Organotin compounds are characterised by the presence of at least one covalent tin-to-carbon bond. The organotin compounds can be divided in four classes, depending on the number of organic groups. The classes are tetraorganotins, triorganotins, diorganotins and monooorganotins. The Sn-C bonds are stable in water, to atmospheric O₂ and temperatures reaching 200 °C. Tin atoms can replace carbon atoms in chemical compounds and a great variety of organotin compounds are known. The compounds and groups of compounds listed in Table 2 are all mentioned in the Norwegian list of Dangerous Substances.

Table 2: Organotin compounds and corresponding CAS no.

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Compound</th>
<th>CAS nr</th>
</tr>
</thead>
<tbody>
<tr>
<td>MBT</td>
<td>Monobutyltin</td>
<td>78763-54-9</td>
</tr>
<tr>
<td>DBT</td>
<td>Dibutyltin</td>
<td>1002-53-5</td>
</tr>
<tr>
<td></td>
<td>Dibutyltin hydrogen borate</td>
<td>75113-37-0</td>
</tr>
<tr>
<td>TBT</td>
<td>Tributyltin</td>
<td>36643-28-4</td>
</tr>
<tr>
<td></td>
<td>Tributyltin benzoate</td>
<td>4342-36-3</td>
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<tr>
<td></td>
<td>Tributyltin chloride</td>
<td>1461-22-9</td>
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<tr>
<td></td>
<td>Tributyltin fluoride</td>
<td>1983-10-4</td>
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<tr>
<td></td>
<td>Tributyltin linoleate</td>
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<td></td>
<td>Tributyltin methacrylate</td>
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<td></td>
<td>Tributyltin naphthenate</td>
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<td></td>
<td>Tributyltin oxide</td>
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<td></td>
<td>Tributyltin sulfide</td>
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<td>Tributyltin adipate</td>
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<td>Tributyltin acetate</td>
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<td>MPT</td>
<td>Monophenyltin</td>
<td>1124-19-2</td>
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<td></td>
<td>Stannane, trichlorophenyl</td>
<td>18952-75-5</td>
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<td>DPT</td>
<td>Diphenyltin</td>
<td>6381-06-2</td>
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<td>TPT</td>
<td>Triphenyltin</td>
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<td></td>
<td>Triphenyltin acetate</td>
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<td>Triphenyltin hydroxide</td>
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<td></td>
<td>Triphenyltin fluoride</td>
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<tr>
<td>Cyhexatin</td>
<td></td>
<td>13121-70-5</td>
</tr>
<tr>
<td>Fenbutatin oxide</td>
<td></td>
<td>13356-08-6</td>
</tr>
<tr>
<td>Tin(II) metan sulphonate</td>
<td></td>
<td>53408-94-9</td>
</tr>
</tbody>
</table>
The production of organotin compounds for commercial use has been going on since the 1940s. In 1992, worldwide total organotin production was more than 50,000 tonnes per year (Mercier et al., 1994), with approximately 25% of the total being triorganotins. Organotins differ in physical, chemical and biological properties and are used in a variety of industrial applications:

**Physical properties**

Table 3: Selected Butyl- and phenyltin compounds and their physical properties

<table>
<thead>
<tr>
<th></th>
<th>Boiling point (°C)</th>
<th>Density (g/cm³)</th>
<th>Solubility (mg/L³)</th>
<th>Vapour Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>MBT (BuSnCl₃)</td>
<td>93</td>
<td>1.69</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td>DBT (Bu₂SnCl₂)</td>
<td>135</td>
<td>No data</td>
<td>4-50 *</td>
<td>0.00016 (kPa at 25°C)</td>
</tr>
<tr>
<td>TBT (Bu₃SnCl)</td>
<td>172</td>
<td>1.21</td>
<td>50*</td>
<td>No data</td>
</tr>
<tr>
<td>MPT (PhSnCl₃)</td>
<td>142</td>
<td>1.84</td>
<td>7.3</td>
<td>0.0233 (Torr, 25°C)</td>
</tr>
<tr>
<td>DPT</td>
<td>No data</td>
<td>No data</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td>TPT (Ph₃SnCl)</td>
<td></td>
<td></td>
<td>40</td>
<td>0.021 mPa</td>
</tr>
</tbody>
</table>

* Solubility in seawater

**Chemical structures**

![Chemical structures of selected organotin compounds](image)

**Toxicological data**

It appears that the tri- and tetra-substituted tin compounds are more toxic than the mono-and di-substituted compounds. The more volatile methyl- and ethyl-substituted tin organic chemicals are more toxic than the tin compounds substituted with longer chain alkyl substituents. Methyl- and ethyl-tin are regarded as very toxic and there are several case reports about lethal outcome after intoxication with these compounds. The methyl- and ethyltins are recognized as primarily neurotoxic, whereas the butyl- and phenyltins are recognized as immunotoxic. It is also recognized that toxicity increases as a function of lipid solubility. WHO made an extensive review of the acute lethal concentrations of several organotin compounds (WHO, 1980).
Monobutyltin

No toxicological data indicating that monobutyltin is toxic to humans is available. Monobutyltin is regarded as relatively non-toxic and no reports indicating significant toxicity on terrestrial and marine environment are found. Acute lethal doses of some mono substituted organic tin compounds are listed in Table 4. Mice treated with a high dose of monobutyltin trichloride (4000 mg/kg) developed hemorrhages in the digestive tract (http://www.atsdr.cdc.gov/index.html).

Table 4: Acute toxicity of mono-substituted organotin compounds. The table is taken from WHO, 1980.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>LD50 mg/kg</th>
<th>Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butylstannoic acid</td>
<td>po</td>
<td>&gt;6000</td>
<td>Mice</td>
</tr>
<tr>
<td>Butyltin trichloride</td>
<td>po</td>
<td>1400</td>
<td>Mice</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2410</td>
<td>Rat</td>
</tr>
<tr>
<td>Octyltin trichloride</td>
<td>po</td>
<td>4600</td>
<td>Mice</td>
</tr>
</tbody>
</table>

Dibutyltin

Few reports are found associating dibutyltin with human health effects with the exception as a skin irritant inducing skin lesions and burns in occupationally exposed humans (WHO, 1990). Animal data shows, however, that dibutyltin is considerably more toxic than the mono-substituted with acute lethal doses around 50 mg/kg (Table 5). Signs of poisoning include liver damage and inflammation in common bile duct. Dibutyltin is primarily regarded as potential immunotoxic. It has a selective effect on the thymus followed by a depletion of lymphocytes indicating suppression of the immune system (http://www.atsdr.cdc.gov/index.html).

The effect on thymus and the lymphoreticular system appears to be most pronounced in rats. Whalen et al. (1999) also showed that dibutyltin inhibits human natural killer cells in vitro. Dioctyltin exert similar effect on thymus. Dibutyltin has strong affinity to dithiol groups (Whalen et al., 1999). Several studies have shown that dibutyltin induce developmental- and reproductive effects, such as increased number of dead fetuses, resorptions and malformations, but these effects are usually combined with maternal toxicity (http://www.atsdr.cdc.gov/index.html). A recent study has indicated that dibutyltin may be a developmental neurotoxicant in vitro and in vivo (Jenkins et al., 2004), but this remains to be elucidated further. US-Agency for Toxic Substances and Deaseses (ATSDR) has evaluated an MRL (exposure levels posing a minimum risk to humans) for dibutyltin dichloride of 0.005 mg/kg/day based on a lowest observable adverse effect level (LOAEL) of 5 mg/kg/day for immunological effects in rats (http://www.atsdr.cdc.gov/index.html).

Table 5: Acute toxicity of di-substituted organotin compounds. The table is an extract from WHO, 1980.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>Sex</th>
<th>LD50 mg/kg</th>
<th>Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dibutyltin dt(2-ethylhexoate)</td>
<td>po</td>
<td>m</td>
<td>200</td>
<td>Rat</td>
</tr>
<tr>
<td>Dibutyltin dt(butyl maleate)</td>
<td>po</td>
<td>m</td>
<td>120</td>
<td>Rat</td>
</tr>
<tr>
<td>Dibutyltin dt(nonyl maleate)</td>
<td>po</td>
<td>m</td>
<td>170</td>
<td>Rat</td>
</tr>
<tr>
<td>Dibutyltin dichloride</td>
<td>po</td>
<td>m</td>
<td>100</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>f</td>
<td></td>
<td>112</td>
<td></td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>f</td>
<td>35</td>
<td>Mice</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td></td>
<td>190</td>
<td>Guinea pig</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>f</td>
<td>150</td>
<td>Rat</td>
</tr>
<tr>
<td>Dibutyltin dilaurate</td>
<td>po</td>
<td></td>
<td>243</td>
<td>Rat</td>
</tr>
<tr>
<td>Dibutyltin oxide</td>
<td>po</td>
<td>m</td>
<td>520</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>ip</td>
<td>m/f</td>
<td>39.9</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>ip</td>
<td>m</td>
<td>24</td>
<td>Mice</td>
</tr>
<tr>
<td>Dibutyltin sulfide</td>
<td>po</td>
<td>f</td>
<td>145</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td></td>
<td>150</td>
<td>Rabbit</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>f</td>
<td>180</td>
<td>Rat</td>
</tr>
<tr>
<td>Dioctyltin acetate</td>
<td>po</td>
<td></td>
<td>2030</td>
<td>Rat</td>
</tr>
<tr>
<td>Dioctyltin dibutylmaleate</td>
<td>po</td>
<td>m</td>
<td>3750</td>
<td>Mice</td>
</tr>
<tr>
<td>Dioctyltin dichloride</td>
<td>po</td>
<td>m</td>
<td>5500</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>8500</td>
<td></td>
</tr>
<tr>
<td>Dioctyltin dilaurate</td>
<td>ip</td>
<td>f</td>
<td>6450</td>
<td>Rat</td>
</tr>
<tr>
<td>Dioctyltin oxide</td>
<td>po</td>
<td>m</td>
<td>800</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>m</td>
<td>2500</td>
<td>Rat</td>
</tr>
</tbody>
</table>
Tributyltin

Tributyltin has been shown to produce irritation of the upper respiratory tract, such as sore throat and burning nose, chest irritation and tightness after inhalation exposure. TBT is also a skin and eye, irritant similar to DBT (WHO, 1990). It is also reported nausea and vomiting after exposure by inhalation to tributyltin in paint for mildew control (http://www.atsdr.cdc.gov/index.html).

Animal studies have shown that tributyltin is less toxic than dibutyltin with acute lethal oral doses between 100 and 200 mg/kg (see Table 6). Signs of poisoning include liver damage and inflammation in common bile duct. It is suggested that the hepatotoxic effects is due to formation of dibutyltin. Sub-lethal doses of TBT are shown to induce enlargement of adrenal glands. Lower doses in sub-chronic exposure studies have shown a reversible reduction in serum thyroxin (T4) and TSH levels indicating effects on the thyroid gland (Adeeko et al., 2003; http://www.atsdr.cdc.gov/index.html).

In the study by Adeeko et al. (2003), it was reported increases in post-implantation losses and decreased litter size in dams exposed to the highest doses (20 mg/kg day during gestation). As for DBT, TBT is primarily regarded as an immunotoxin as shown in several animal studies and in vitro studies. It has a selective effect on the thymus, inducing thymic atrophy followed by a depletion of lymphocytes, and inhibits activity of natural killer cells indicating suppression of the immune system (http://www.atsdr.cdc.gov/index.html; Luebke et al., 2006). US-ATSDR has evaluated an MRL (exposure levels posing a minimum risk to humans) for TBT-oxide of 0.0003mg/kg/day based on a LOAEL of 0.025 mg/kg/day for immunological effects in rats (http://www.atsdr.cdc.gov/index.html).

TBT is most known for it high toxicity towards some aquatic organisms, in particular to marine molluscs as an endocrine disruptor (WHO, 1990). For example, the NOEL for the development of imposex in female dogwhelks is below 1.5 ng TBT/L. The 96h LC50 to fish range between 1.5µg/L and 36µg/L (WHO, 1990).

TBT is used as a bactericide and algicide of which the algicidal concentrations range from less than 1.5µg/L to more than 1000µg/L depending on the species (WHO, 1990).

Table 6: Acute toxicity of tri-substituted organotin compounds. The table is an extract from WHO, 1980.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>Sex</th>
<th>LD50</th>
<th>Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triethyltin</td>
<td>po</td>
<td>f</td>
<td>4</td>
<td>Rat</td>
</tr>
<tr>
<td>acetate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triethyltin</td>
<td>ip</td>
<td>m</td>
<td>5.7</td>
<td>Rat</td>
</tr>
<tr>
<td>sulfate</td>
<td>ip</td>
<td></td>
<td>5.3</td>
<td>Guinea pig</td>
</tr>
<tr>
<td>Triethyltin</td>
<td>ip</td>
<td>f</td>
<td>5</td>
<td>Rat</td>
</tr>
<tr>
<td>chloride</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tributyltin</td>
<td>po</td>
<td></td>
<td>46</td>
<td>Mice</td>
</tr>
<tr>
<td>acetate</td>
<td>po</td>
<td></td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>Tributyltin</td>
<td>po</td>
<td>m</td>
<td>133</td>
<td>Rat</td>
</tr>
<tr>
<td>benzoate</td>
<td></td>
<td></td>
<td>108</td>
<td>Mice</td>
</tr>
<tr>
<td>Tributyltin</td>
<td>po</td>
<td></td>
<td>132</td>
<td>Rat</td>
</tr>
<tr>
<td>chloride</td>
<td></td>
<td></td>
<td>117</td>
<td>Rat</td>
</tr>
<tr>
<td>Tributyltin</td>
<td>po</td>
<td></td>
<td>129</td>
<td>Mice</td>
</tr>
<tr>
<td>benzoate</td>
<td></td>
<td></td>
<td>180</td>
<td>Mice</td>
</tr>
<tr>
<td>Tributyltin</td>
<td>po</td>
<td></td>
<td>195</td>
<td>Rat</td>
</tr>
<tr>
<td>oleate</td>
<td></td>
<td></td>
<td>137</td>
<td>Rat</td>
</tr>
<tr>
<td>Tributyltin</td>
<td>po</td>
<td></td>
<td>1000</td>
<td>Rat</td>
</tr>
<tr>
<td>salicylate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trihexyltin</td>
<td>po</td>
<td></td>
<td>1000</td>
<td>Rat</td>
</tr>
<tr>
<td>acetate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trioctyltin</td>
<td>po</td>
<td></td>
<td>80</td>
<td>Rat</td>
</tr>
<tr>
<td>chloride</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triphenyltin</td>
<td>po</td>
<td></td>
<td>81</td>
<td>Mice</td>
</tr>
<tr>
<td>acetate</td>
<td>po</td>
<td></td>
<td>136</td>
<td>Rat</td>
</tr>
<tr>
<td>ip</td>
<td>m</td>
<td></td>
<td>7.9</td>
<td>Mice</td>
</tr>
<tr>
<td>dermal</td>
<td></td>
<td></td>
<td>450</td>
<td>Rat</td>
</tr>
<tr>
<td>ip</td>
<td>f</td>
<td></td>
<td>8.5</td>
<td>Rat</td>
</tr>
<tr>
<td>po</td>
<td>m</td>
<td></td>
<td>21</td>
<td>Guinea pig</td>
</tr>
<tr>
<td>ip</td>
<td>m</td>
<td></td>
<td>3.7</td>
<td>Guinea pig</td>
</tr>
<tr>
<td>Triphenyltin</td>
<td>po</td>
<td>m</td>
<td>135</td>
<td>Mice</td>
</tr>
<tr>
<td>chloride</td>
<td></td>
<td></td>
<td>30-50</td>
<td>Rabbit</td>
</tr>
<tr>
<td>hydroxide</td>
<td>po</td>
<td>m</td>
<td>240</td>
<td>Rat</td>
</tr>
<tr>
<td>po</td>
<td>m</td>
<td></td>
<td>27.1</td>
<td>Guinea pig</td>
</tr>
<tr>
<td>po</td>
<td>f</td>
<td></td>
<td>31.1</td>
<td>Guinea pig</td>
</tr>
<tr>
<td>po</td>
<td>m</td>
<td></td>
<td>171</td>
<td>Rat</td>
</tr>
<tr>
<td>po</td>
<td>f</td>
<td></td>
<td>268</td>
<td>Rat</td>
</tr>
</tbody>
</table>
Phenyltins

The phenyltin compounds include mono-, di-, tri-, and tetraphenyltin. Most knowledge is available on triphenyltin. It is reason to believe that toxicity increase as a function of the number of phenyl substituents. Only few cases on human intoxication are reported. Inhalation of fungicide containing TPT induced dizziness, nausea, photophobia and temporary loss of consciousness. The patients completely recovered within 10 days after hospitalization (WHO, 1999). Two other case studies revealed neurological effects, such as disturbance of consciousness, spontaneous involuntary movement of hands and disorientation to people, time and places. The patients recovered within a year (http://www.atsdr.cdc.gov/index.html).

Table 7 Acute toxicity of tetra-substituted organotin compounds after oral and intraperitoneal administration. The table is an extract from WHO, 1980.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>LD50 mg/kg</th>
<th>Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tetraethyltin</td>
<td>po</td>
<td>40</td>
<td>Mice</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>15</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>40</td>
<td>Mice</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>9</td>
<td>Rat</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>40</td>
<td>Guinea pig</td>
</tr>
<tr>
<td></td>
<td>po</td>
<td>7</td>
<td>Rabbit</td>
</tr>
</tbody>
</table>

Oral lethal TPT concentration is similar to TBT with an estimated LD50 of 160 mg/kg in rats and mice (WHO, 1999). Rabbit and guinea pig are more sensitive with an estimated LD50 of 10-50 mg/kg (WHO, 1994). Sub-chronic investigations with rats, mice, and dogs showed a decrease in immunoglobulin levels, body weight gain, and white blood cells and an increase in liver weights and death. NOAELs for immunological parameters, which appear most sensitive in animal studies, from several of these dietary studies has been estimated as 3.4-4.1 mg/kg body weight per day in mice (3-month exposure), 0.30-0.35 mg/kg body weight per day in rats (13-week exposure), and 0.21 mg/kg body weight per day in dogs (52-week exposure) (WHO, 1999). TPT is in animal studies shown to cause reproductive effects, similar to TBT, at relatively low doses administered during pregnancy (~1mg/kg/day). However, maternal toxicity was observed in these studies and it is unclear if these effects were due to maternal toxicity (WHO, 1999) (http://www.atsdr.cdc.gov/index.html). As for the butyltins TPT is immunotoxic in animals studies, showing immunosuppressive properties such as decrease in spleen and thymus weights, blood cells, neutrophils, and lymphocytes resulting in altered humoral and cellular immunity (WHO, 1999). However, the effects are shown less pronounced than observed with TBT.

TPT is highly toxic towards aquatic organisms as an endocrine disruptor (WHO, 1999). The most sensitive effect appears to be imposex in gastropods and NOEC is assumed to be less than 1 ng/L. The 96h LC50 to fish (Fathead minnow) and copepods is 7.1 µg/L, and 8 µg/L respectively, and LC50s in a 48-h exposure for Daphnia magna were 10-200 µg/L (WHO, 1999). The NOEC for reproduction in Daphnia magna in a 21-day exposure is reported to 0.1 µg/L. The toxicity towards alga depends on species and occurs from approximately 1 µg/L (WHO, 1999). TBT and TPT are responsible for imposex in snails, neo- and mesogastropods, a phenomenon where females develop male sexual caracteristica (Horiguchi et al., 1997).

Degradation

Degradation of organotin compounds occurs by stepwise removal of organic groups from the tin atom. The toxicity decreases with increasing dealkylation. The removal of organic groups can be caused by both abiotic and biotic processes, such as UV irradiation, chemical
cleavage and biological cleavage of the Sn-C bond. Photolysis by sunlight appears to be the fastest degradation route in upper water levels, but not important in water at greater depths, sediments or soils (Hoch, 2001). Some bacteria and microalgae species are found to be capable of degrading TBT in different environmental compartments (Reader and Pelletier, 1992; Tsang et al., 1999). Up to date, few microorganisms with this ability have been identified and little is known about the environmental conditions needed for such decay processes. However, toxic concentration of organotin, temperature, light, pH, salinity and nutrition are some factors that have an impact on biological activity (Hoch, 2001; Dubey and Roy, 2003).

Use in Norway

TBT and TPT as chemicals have not been produced in Norway, but are used for the fabrication of other products. A search in Vetrinærkatalogen resulted in no hits for organotin compounds, indicating that veterinary medicaments used in Norway do not contain organotin.

Antifouling agents, containing TBT and TPT, are not longer permitted in Norway for ships longer than 25 m. The estimated amount of antifouling containing TBT was 9000 kg in Norway in 2003 (SFT, TA-2127/2005).

Emissions

In Norway, boats and shipyards are the only known sources of TBT emissions. SFT estimated the emission of TBT to water as 8000 kg in 2003 (SFT, TA-2127/2005).

German industry reports through the European Pollutant Emission Register (EPER), the emission of 267 kg/yr organotin to water (http://www.eper.de).

Emission data of organotin from diffuse sources like recreational boats are not given.

Monitoring

Organotin compounds are amongst the most widely used organometallic compounds. Due to the widespread use considerable amounts of these compounds have entered the different ecosystems. To date, most attention has been given to TBT and its degradation products in water and sediments due to TBTs toxic effect on aquatic life at low concentrations. In 2006, a vast amount of publications concerning TBT and degradation products in different environmental compartments and organisms are available. Three reviews covering occurrence in environmental samples, biological chemistry and potential effects where used as a starting point for the present literature survey (Hoch, 2001; Buck-Koehntop et al., 2006; Florea and Busselberg, 2006), in addition to “Organometallic Compounds in the Environment” (Hoch, 2001; Craig, 2003). The information for TPT, DPT and MPT is limited.

Organotin in aquatic systems

Studies of TBT and its degradation products have to a grate extent been concentrated to areas with heavy ship traffic such as harbours, marinas, shipyards, and coastal areas due to the direct emission of TBT from antifouling paints. Sewage sludge, municipal and industrial wastewater leaking of landfills does make a significant contribution as input to aquatic systems. TBT is rapidly adsorbed onto suspended particulate material or taken up by algae (Luan et al., 2006) and zooplankton (Hu et al., 2006; Michaud and Pelletier, 2006). Hence it is removed from the water column over time to be found in sediments and biota. These processes are faster than aqueous decay. When new input of TBT to water decreases, the concentration in the water column will be reduced.
quite rapidly. Some studies claim that the ongoing restrictions concerning use of organotins in ship industry in many countries do show a positive effect on marine environment (Maquire, 1984; Jorundsdottir et al., 2005). Even so, organotin compounds are still released into the aquatic systems from various sources and will represent an environmental risk for some time to come. A selection of quantitative results is given in Table 8.

Table 8: Concentration of different organotins in water.

<table>
<thead>
<tr>
<th>Sample type/Location</th>
<th>Unit</th>
<th>MBT</th>
<th>DBT</th>
<th>TBT</th>
<th>TPT</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>River water, Portugal</td>
<td>ng Sn /L</td>
<td>&lt;3-26</td>
<td>&lt;3-30</td>
<td>&lt;3-29</td>
<td></td>
<td>(Diez et al., 2005)</td>
</tr>
<tr>
<td>River water, Spain</td>
<td>ng Sn /L</td>
<td>6.9-41</td>
<td>5.5-68</td>
<td>9.3-488</td>
<td></td>
<td>(Gomez-Ariza et al., 2006)</td>
</tr>
<tr>
<td>Sea water, Netherland</td>
<td>ng Sn /L</td>
<td>3-310</td>
<td>0.1-810</td>
<td>0.1-3620</td>
<td></td>
<td>(Ritsema and Laane, 1991)</td>
</tr>
<tr>
<td>Surface water, India</td>
<td>ng Sn /g dw</td>
<td>6-33</td>
<td>10-89</td>
<td></td>
<td></td>
<td>(Bhosle et al., 2004)</td>
</tr>
<tr>
<td>Municipal wastewater, Switzerland</td>
<td>ng Sn /g dw</td>
<td>245</td>
<td>523</td>
<td>157</td>
<td></td>
<td>(Fent and Muller, 1991)</td>
</tr>
<tr>
<td>Sea water, China</td>
<td>ng/L</td>
<td></td>
<td>9.8</td>
<td>&lt; 6.8</td>
<td></td>
<td>(Gao et al., 2004)</td>
</tr>
</tbody>
</table>

**Organotin in sediments**

Organotins are easily adsorbed to total suspended matter and are deposited in the sediments, which may act as a sink for TBT and its degradation products. Degradation of TBT is relatively slow in sediments, with half-lives reported in the region of years (de Mora and Pelletier, 1997) compared with days or weeks in water column (Maquire, 1984). After the restriction of the use of TBT containing antifouling in Canada, TBT was found in sediments much more frequently than in the water column (Maquire, 1984). The authors concluded that the regulation had been generally effective in reducing TBT contamination in water, but not in sediments. Organotin in sediments is available to filter- and sediment-feeding organisms and may enter the food web through this pathway.

Table 9: Concentration of different butyltins in sediments.

<table>
<thead>
<tr>
<th>Sample type/Location</th>
<th>Unit</th>
<th>MBT</th>
<th>DBT</th>
<th>TBT</th>
<th>Sum BT</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sediments</td>
<td>ng Sn/g dw</td>
<td>35-440</td>
<td>67-2607</td>
<td>98-4702</td>
<td></td>
<td>(Diez et al., 2005)</td>
</tr>
<tr>
<td>Sewage sludge, Switzerland</td>
<td>ng/g dw</td>
<td>245</td>
<td>523</td>
<td>157</td>
<td></td>
<td>(Fent and Muller, 1991)</td>
</tr>
<tr>
<td>Marine sediments, Norway</td>
<td>ng/g dw</td>
<td>3-94</td>
<td>2-255</td>
<td>1-987</td>
<td>&lt;0.0660</td>
<td>(SFT, 2005)</td>
</tr>
<tr>
<td>Marine sediment, Oman</td>
<td>ng Sn/g</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0660</td>
<td>(de Mora et al., 2003)</td>
</tr>
<tr>
<td>Marine sediment, Denmark</td>
<td>ng Sn/g</td>
<td></td>
<td></td>
<td></td>
<td>1-19</td>
<td>(Strand et al., 2003)</td>
</tr>
</tbody>
</table>

Table 10: Concentration of different phenyltins in sediments.

<table>
<thead>
<tr>
<th>Sample type/Location</th>
<th>Unit</th>
<th>MPT</th>
<th>DPT</th>
<th>TPT</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marine sediments, Norway</td>
<td>ng /g dw</td>
<td>1-47</td>
<td>&lt;10</td>
<td>&lt;23</td>
<td>(SFT, 2005)</td>
</tr>
</tbody>
</table>
Organotins are lipophilic compounds and have easy access to different parts of trophic chains. Organotin is taken up by animals by dietary uptake and/or uptake directly from solution. TBT and its degradation products are found in a variety of living organisms. Data of TPT and its degradation products are limited.

### Table 11: Concentration of butyltins in organisms.

<table>
<thead>
<tr>
<th>Sample type/Location</th>
<th>Unit</th>
<th>MBT</th>
<th>DBT</th>
<th>TBT</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zooplankton, Canada</td>
<td>&lt;0.05-312</td>
<td>32-453</td>
<td>&lt;0.05-68</td>
<td>(Michaud and Pelletier, 2006)</td>
<td></td>
</tr>
<tr>
<td>Sea Snails, Japan</td>
<td>ng/g ww</td>
<td>11-501</td>
<td>8-443</td>
<td>7-797</td>
<td>(Horiguchi et al., 1997)</td>
</tr>
<tr>
<td>Fish, Korea</td>
<td>ng/g dw</td>
<td>51-2860</td>
<td>10-290</td>
<td>50-3900</td>
<td>(Shim et al., 2005)</td>
</tr>
<tr>
<td>Mussels, UK</td>
<td>ng/g dw</td>
<td>38-734</td>
<td>46-1439</td>
<td>101-2348</td>
<td>(SFT, 2005)</td>
</tr>
<tr>
<td>Mussels, Norway</td>
<td>ng/g dw</td>
<td>&lt;0.05</td>
<td>32-453</td>
<td>&lt;0.05-68</td>
<td>(Michaud and Pelletier, 2006)</td>
</tr>
</tbody>
</table>

### Table 12: Concentration of phenyltins in organisms.

<table>
<thead>
<tr>
<th>Sample type/Location</th>
<th>Unit</th>
<th>MPT</th>
<th>DPT</th>
<th>TPT</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sea Snails, Japan</td>
<td>ng/g ww</td>
<td>n.d.</td>
<td>n.d.</td>
<td>5-2714</td>
<td>(Horiguchi et al., 1997)</td>
</tr>
<tr>
<td>Mussels, UK</td>
<td>ng/g dw</td>
<td>&lt; 20</td>
<td>&lt; 10</td>
<td>8-1560</td>
<td>(Shim et al., 2005)</td>
</tr>
<tr>
<td>Fish, Korea</td>
<td>ng/g dw</td>
<td>&lt;LOD-308</td>
<td>&lt;LOD-256</td>
<td>6.3-38.6</td>
<td>(SFT, 2005)</td>
</tr>
<tr>
<td>Mussels, Korea</td>
<td>ng Sn/g dw</td>
<td>&lt;3-1820</td>
<td>&lt;3-1820</td>
<td>6.3-38.6</td>
<td>(SFT, 2005)</td>
</tr>
<tr>
<td>Mussels, Norway</td>
<td>ng/g dw</td>
<td>&lt; 0.76</td>
<td>&lt; 0.76</td>
<td>6.3-38.6</td>
<td>(SFT, 2005)</td>
</tr>
</tbody>
</table>

Organotin in atmosphere

The data on organotin species in the atmosphere is limited, and show some discrepancy in the conclusions. Detectable amounts of organotin species in the atmosphere were found to be negligible (Blunden and Chapman, 1982).

However, volatile organotin species such as methylated forms of butyltin derivatives were detected in three European estuaries. Significant exports of volatile tin species to adjacent coastal waters were found. Evaluation of seasonal fluxes to the atmosphere, led the authors to the conclusion that volatilisation is a major sink (Tessier et al., 2002). Laboratory studies of emission of volatile butyltin species from TBT contaminated seawater and sediments are reported (Mester and Sturgeon, 2002; Saint-Louis and Pelletier, 2004).

Long-Range Transport

Most organotin species are not volatile and not considered available for LRT. However, biomethylation has been found to produce volatile butyltin species (Tessier et al., 2002).

The following national monitoring programs collect data on organotin:

Miljøgifter langs norskekysten

Data from Norwegian Joint assessment and monitoring program (JAMP) show that TBT was present in Norwegian marine waters in 2004, mostly close to harbours, but also in samples from stations remote from known sources. Matrix studied were dogwhelk (Nucella Lapillus) and blue mussels (Mytilus edulis). The concentration found in dogwhelk was reported to be <0.26 mg/kg dw, while the concentration in blue mussels varied between 0.006-2.8 mg/kg dw. No
significant trends were found (SFT, 944/2005). Previously published reports under JMP/JAMP can be downloaded from http://www.sft.no/program___37037.aspx.

Statlig program for forurensingsovervåking: Miljøgifter i havner

An environmental survey was executed in nine harbours and three river systems in Nordland, Norway. Concentrations of TBT varied from low- to highly contaminated according to SFTs classification system (DNV, 0507-2003).

Sediments in harbours located in Telemark, Vestfold, Akershus and Østfold were analysed for TBT in 1999. The concentrations of TBT determined were high throughout all areas investigated, locations with severe TBT pollution were revealed (SFT, 849-2002).

Sediments and mussels from several harbours at eight locations in Agder were analysed for TBT in the time period 1997-1998. The TBT concentration in sediments varied from 0.015-2.562 mg/kg dw. TBT measurements of sediments showed values that correspond to level IV and V in the SFT classification system in 13 of 14 locations. TBT concentration in mussels from Risør was found to be 1.635 mg/kg dw (NIVA, 4232-2000).

Sediments and mussels in harbours located in Harstad, Tromsø, Hammerfest and Honningsvåg were analysed for TBT and TPT, TPT and their degradation products, respectively in 1997-98. In Harstad the TBT concentration in sediments varied between 0.001-35.0 mg/kg. Generally, sediment concentrations of TBT from the harbours investigated represented level IV-V according to SFTs classification system. TPT was detected in the sediment samples showing the highest TBT values as well. TBT and TBT concentrations in mussels varied between of 0.006-14.9 and 0.007-2.5 mg/ kg dw respectively (SFT, 786/2000).

TBT concentrations in mussels from 39 Norwegian harbours were determined. The concentrations found were varying, but often high (SFT, 610/1995).

Other monitoring reports

Organotin concentration in cod from inner Oslo Fjord was found to be lower than previously reported. The average concentration of TBT and TPT were 4.5 and 3.6 µg/kg wet weight, respectively. TPT was detected in the same concentration level as TBT, which should be of concern, taken into account what is known about national distribution of TPT. The average concentrations of degradation products from both compounds are reported to be lower than detection limit (NIVA, 5242-2006).

OCEANOR in collaboration with Eurofins executed a survey of a landfill in Trondheim harbour in 2003. Concentrations of TBT in mussels in the region of 0.137-0.251 mg TBT/kg were reported (OCEANOR, 2003; http://www.trondheim.havn.no/pilotprosjektet/C75040_3791_R1.pdf).

The Swedish Status and Trends Monitoring Program (SSTMP) was launched in 2003. Under this program marine sediments from 16 locations distributed within the Swedish continental shelf were studied. TBT and its degradation products were detected in all samples and varied in the region of 1-110 µg/kg dw (Cato and Kjellin, 2005).

Danish, Norwegian and Swedish data on measured TBT concentrations and effects in marine gastropods are gathered in a comprehensive assessment of TBT levels in coastal and open waters of Skagerak and Kattegat. The data show that TBT poses a threat to marine organisms inhabiting the Skagerak and Kattegat. No areas were unaffected. (Forum Skagerak, 2006; http://www.forumskagerak.com).
Evaluation of need for monitoring

Despite the legislative restrictions concerning the use of toxic organotins, TBT and TPT are still found in high concentrations in the marine environment. The slow degradation of organotins in historically contaminated sediments poses a risk of contamination of water and biosphere due to remobilization or desorption processes. Harbours are areas where high organotin concentrations still are expected, due to leakage from antifouling paints from ships with length less than 25 m in addition to historical contamination. High concentrations of TBT in sediments have earlier been found in most Norwegian harbours (SFT, 610-1995, SFT, 786/2000, OCEANOR, 2003). Examples of potential screening locations are the harbours of the cities Narvik, Trondheim, Kragerø and Oslo.

Additionally, the marine environment close to shipyards and mechanical workshops doing sandblasting of boats and offshore installations are areas with high potential for organotin emission. Vest Sandblåsing, Hitra, handles both ships and offshore installations and is a suitable location for measurements. Matrix of choice will be sediments and sludge.

Both possible historical and current emissions of organotin from industry producing/using PVC may be discovered in industrial and municipal wastewater. Hydro Polymers, Heroya Industrial Park, Porsgrunn has produced PVC since 1951 and is per se the only producer in Norway. Matrix of choice will be sludge. Norsk Titanduk A/S uses PVC in their production. Matrix of choice will be sludge.

Leaking of organotins from PVC in landfills may also contribute to contaminating aquatic systems. Lindum Ressurs og Gjennvinning, Drammen, handles both municipal and hazardous waste and will be a suitable location for measurements. Matrix of choice will be sludge and sediments.

Analysis

Depending on the choice of sample preparation method, butyltins and phenyltins may be determined in the same run. Several analytical techniques have been used for determination of organotin. Most of them are based on gas chromatography (GC) in combination with mass spectrometry (MS), atomic absorption spectrometry (AAS), flame photometry (FPD) and inductively coupled plasma atomic emission spectrometry (ICP-AES). The use of liquid chromatography in combination with ICP-MS is also reported. A few examples of methods and techniques that are applied for determinations of organotins in different matrixes are given.

a) Determination of butyltins in sediments: Extraction, derivatisation, cleaned on silica gel, GC-MS (Michaud and Pelletier, 2006)
b) Determination of butyltins in biological samples: Digestion, derivatisation, cleaned on silica gel, GC-MS (Michaud and Pelletier, 2006)
c) Determination of butyl- and phenyltins in biota: Extraction, drying, derivatisation, GC-FPD (Harino et al., 1992; Harino et al., 1998)
d) Determination of butyl- and phenyltins in sediments: Extraction, HPLC- ICPMS, separation on C18 (White et al., 1998)
References


A literature survey on selected chemical substances – TA-2238/2007


Maquire RJ. Transformation of the Tributyltin Species in Toronto Harbour Sediment. Abstracts of Papers of the American Chemical Society 1984; 188: 31-ENVR.


Shim WJ, Yim UH, Kim NS, Hong SH, Oh JR, Jeon JK, Okamura H. Accumulation of butyl- and phenyltin compounds in starfish and bivalves from the coastal environment of Korea. Environmental Pollution 2005; 133: 489-499.


6 Noble metals

6.1 Platinum

Platinum is one of the rare elements and the average concentration in the earth crust is estimated to be approximately 1-5 µg/kg. Platinum can be found in its metallic form or as several mineral forms. Platinum shows considerably greater affinity for sulphur than oxygen, and for this reason is found mainly as sulphides. Platinum does not corrode in air at any temperature and does not dissolve in 1 M [H+] , which places it among the noble metals.

<table>
<thead>
<tr>
<th>Formula</th>
<th>Compound</th>
<th>CAS-Nr.</th>
<th>EC-No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt</td>
<td>Platinum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PtO</td>
<td>Platinum(II)oxide</td>
<td></td>
<td>n.a.</td>
</tr>
<tr>
<td>PtO2</td>
<td>Platinum(IV)oxide</td>
<td>1314-15-4</td>
<td></td>
</tr>
<tr>
<td>PtCl2</td>
<td>Platinum(II)chloride</td>
<td>10025-65-7</td>
<td></td>
</tr>
<tr>
<td>PtCl4</td>
<td>Platinum(IV)chloride</td>
<td>13454-96-1</td>
<td></td>
</tr>
<tr>
<td>(NH4)2PtCl6</td>
<td>Ammonium Hexachloroplatinat(IV)</td>
<td>16919-58-7</td>
<td>240-973-0</td>
</tr>
<tr>
<td>(NH4)2PtCl4</td>
<td>Ammonium Tetrachloroplatinat(II)</td>
<td>13820-41-2</td>
<td>231-786-5</td>
</tr>
<tr>
<td>K2PtCl6</td>
<td>Potassium Hexachloroplatinat(IV)</td>
<td>16921-30-5</td>
<td>240-979-3</td>
</tr>
<tr>
<td>K2PtCl4</td>
<td>Potassium Tetrachloroplatinat(II)</td>
<td>10025-99-7</td>
<td>233-050-9</td>
</tr>
<tr>
<td>Na2PtCl6</td>
<td>Sodium Hexachloroplatinat(IV)</td>
<td>16923-58-3</td>
<td>240-983-5</td>
</tr>
<tr>
<td>Na2PtCl4</td>
<td>Sodium Tetrachloroplatinat(II)</td>
<td>10026-00-3</td>
<td>233-051-4</td>
</tr>
<tr>
<td>Other Tetrachloroplatinites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other Hexachloroplatinites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gasses (Petroleum), Platformer Stabilizer Off, Light Ends Fraction</td>
<td>68919-07-3</td>
<td>272-880-6</td>
<td></td>
</tr>
</tbody>
</table>

However, platinum is a complexing agent and is affected by halogens, cyanides, sulphur, and hydroxides. Digestion of platinum is done by the use of aqua regia or HCl/Cl2. The complex compounds dominate the chemistry of platinum compounds in aqueous solution. Many of the salts, particularly those with halogen- or nitrogen-donor ligands, are water-soluble. Platinum, has a pronounced tendency to react with carbon compounds, especially alkenes and alkynes, forming Pt(II) coordination complexes (WHO, 1991).
Platinum may exist as several different species, some of which are extensively used and developed as anticancer drugs (Pasetto et al., 2006). Some platinum drugs exert considerable toxicity, especially on the peripheral nervous system and on the kidney (Safirstein et al., 1986; Hartmann et al., 1999; Screnci and McKeage, 1999; Markman, 2003). The Pt-drugs are mutagenic and suspected carcinogens and might be of concern in relation to occupational exposure on hospitals (Ravindra et al., 2004). It is beyond the scope of this overview to go into detail on the toxicity of these drugs, since they primarily are used as therapeutic agents and therefore designed to be bioactive. The emission of Pt-drugs from hospitals are considered as minor (Kummerer et al., 1999), but should be considered in future investigation due to their toxicity.

The toxicity of Pt compounds depends considerably on their water solubility (WHO, 1991). In its metallic state Platinum can be regarded as non-toxic, although fine dust particles have shown to cause some irritation on the gastrointestinal epithelium in a rat model, and one case which reported contact dermatitis from a platinum ring. Table 13 shows some acute toxicity data on rats of different Pt-compounds. In general the toxicity decrease in the following order cis-[PtCl2(NH3)2] > PtCl4 > Pt(SO4)2·4H2O > PtCl2 > PtO2. Cisplatin (cis-[PtCl2(NH3)2]) is an anticancer drug. Signs of poisoning observed for (NH4)2[PtCl4], include hypokinesia, piloerection, diarrhea, convulsions, laboured respiration, and cyanosis (WHO, 1991). Hexachloroplatinic acid, H2[PtCl6], is highly nephrotoxic in rats. After an intraperitoneal LD50 injection of 40-50 mg/kg, rats died of renal failure, hypocalcaemia, and hyperkalaemia. The necrotizing renal tubular lesions involved the entire renal cortex (WHO, 1991).

Table 13: Acute toxicity of platinum and platinum compounds. The table is taken from WHO, 1991.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>Sex</th>
<th>LD50</th>
</tr>
</thead>
<tbody>
<tr>
<td>PtO2</td>
<td>po</td>
<td>m</td>
<td>&gt;8000</td>
</tr>
<tr>
<td>PtCl4</td>
<td>po</td>
<td>m</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>PtCl2</td>
<td>po</td>
<td>m</td>
<td>3423b</td>
</tr>
<tr>
<td>PtCl4</td>
<td>ip</td>
<td>m</td>
<td>670</td>
</tr>
<tr>
<td>PtCl2</td>
<td>po</td>
<td>m</td>
<td>240</td>
</tr>
<tr>
<td>PtCl4</td>
<td>po</td>
<td>m/f</td>
<td>276b</td>
</tr>
<tr>
<td>PtCl2</td>
<td>ip</td>
<td>m</td>
<td>38</td>
</tr>
<tr>
<td>PtCl4</td>
<td>iv</td>
<td>m</td>
<td>26.2</td>
</tr>
<tr>
<td>PtCl2</td>
<td>iv</td>
<td>m</td>
<td>41.4</td>
</tr>
<tr>
<td>Pt(SO4)2·4 H2O</td>
<td>po</td>
<td>m</td>
<td>1010</td>
</tr>
<tr>
<td>Pt(SO4)2·4 H2O</td>
<td>ip</td>
<td>m</td>
<td>310f</td>
</tr>
<tr>
<td>PtCl2·4 H2O</td>
<td>ip</td>
<td>m</td>
<td>138-184f</td>
</tr>
<tr>
<td>(NH4)2[PtCl6]</td>
<td>po</td>
<td>m/f</td>
<td>195b</td>
</tr>
<tr>
<td>(NH4)2[PtCl6]</td>
<td>po</td>
<td>m/f</td>
<td>~200</td>
</tr>
<tr>
<td>(NH4)2[PtCl6]</td>
<td>po</td>
<td>m</td>
<td>212</td>
</tr>
<tr>
<td>(NH4)2[PtCl6]</td>
<td>po</td>
<td>f</td>
<td>125</td>
</tr>
<tr>
<td>H2[PtCl6]</td>
<td>ip</td>
<td>m</td>
<td>40-50</td>
</tr>
<tr>
<td>Na2[PtCl6]</td>
<td>po</td>
<td>m/f</td>
<td>25-50</td>
</tr>
<tr>
<td>Na2[Pt(OH)6]</td>
<td>po</td>
<td>m/f</td>
<td>500-200</td>
</tr>
<tr>
<td>K2[PtCl4]</td>
<td>po</td>
<td>m/f</td>
<td>50-200</td>
</tr>
<tr>
<td>K2[Pt(CN)4]</td>
<td>po</td>
<td>m/f</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>[Pt(NH3)2]Cl2</td>
<td>po</td>
<td>m/f</td>
<td>&gt;15000</td>
</tr>
<tr>
<td>[Pt(NO2)2(NH3)2]</td>
<td>po</td>
<td>m</td>
<td>~5000</td>
</tr>
<tr>
<td>[Pt(NO2)2(NH3)2]</td>
<td>po</td>
<td>f</td>
<td>&gt;5110</td>
</tr>
<tr>
<td>[Pt(C5H7O2)2]</td>
<td>po</td>
<td>m/f</td>
<td>&gt;500</td>
</tr>
<tr>
<td>cis-[PtCl2(NH3)2]</td>
<td>po</td>
<td>m/f</td>
<td>~20</td>
</tr>
<tr>
<td>cis-[PtCl2(NH3)2]</td>
<td>ip</td>
<td>m</td>
<td>12</td>
</tr>
<tr>
<td>cis-[PtCl2(NH3)2]</td>
<td>ip</td>
<td>m</td>
<td>7.7</td>
</tr>
<tr>
<td>cis-[PtCl2(NH3)2]</td>
<td>iv</td>
<td>m</td>
<td>7.4</td>
</tr>
<tr>
<td>trans-[PtCl2(NH3)2]</td>
<td>po</td>
<td>m/f</td>
<td>&gt;5110</td>
</tr>
</tbody>
</table>

a m = male; f = female
b From the original values given as mg A/kg (= mg atom/kg)
c Results from two different laboratories

Human health effects of Pt are primarily confined to occupational exposure as in for example platinum metal refineries and catalyst manufacture plants (WHO, 1991).
The halogenated Pt-salts are primarily known as powerful sensitisers inducing allergic responses such as respiratory sensitisation and asthmatic reactions (WHO, 1991; Ravindra et al., 2004). The compounds mainly responsible for platinum sensitisation are the halogenated Pt-salts (Linnett and Hughes, 1999), such as hexachloroplatinic acid ($H_2[PtCl_6]$), ammonium hexachloroplatinate ($\text{(NH}_4\text{)}_2[PtCl_6]$), potassium tetrachloroplatinate, ($\text{K}_2[PtCl_4]$), potassium hexachloroplatinate, ($\text{K}_2[PtCl_6]$) and sodium tetrachloroplatinate, ($\text{Na}_2[PtCl_4]$). The allergic response generally increases with increasing number of chlorine atoms. In USA, American Conference of Governmental Industrial Hygienists (ACGIH) recommends a time-weighted Threshold Limit Value (TWA-TLV) for daily occupational exposure to soluble platinum salts at 2 µg Pt/m³ (WHO, 1991). In addition ACGIH recommends a TLV of 1 mg/m³ for platinum metal.

According to the WHO report, platinum compounds at concentrations in the mg/L or mg/kg range affect aquatic and terrestrial plants, and several studies have shown that Pt can bioaccumulate and is bioavailable (WHO, 1991; Ravindra et al., 2004; Zimmermann et al., 2005). Very few toxicological studies have been performed on aquatic animals, but the effect of Pt depends on its chemical state. A LC50 value of 520 µg $H_2[PtCl_6]/L$ was calculated on a chronic 3-week exposure study on Daphnia magna. Biochemical responses were observed at concentrations less than 50 µg/L (WHO, 1991). Ferreira and Wolke (1979) investigated exposure of tetra-chloroplatinatic acid ($\text{PtCl}_2\text{HCl}-\text{6H}_2\text{O}/\text{H}_2[PtCl_4]$) on the coho salmon Oncorhynchus kisutch. In a static bioassay they reported, 24-, 48-, and 96-h LC50 values of 15.5, 5.2, and 2.5 mg Pt/L, respectively. General swimming activity and opercular movement was affected at 0.3 mg/L. Lesions in the gills and the olfactory organ were also observed at 0.3 mg/L or more. Concentrations of 0.03 and 0.1 mg/L had no effect.

**Degradation**

No data available.

**Use in Norway**

Platinum has a variety of applications:
- catalytic converters, sensors and spark plugs
- catalyst in chemical processing
- substitute for gold in jewellery
- high temperatures- and no corrosive wires and contacts
- catalyst in cracking process of crude oil
- in dental/medical equipments and reconstructives
- in cytostatica

**Emissions**

Data on emissions of platinum to the environment from industrial sources are not available. During the use of platinum containing catalysts, some platinum may escape into the environment, depending on the type of catalyst. Of the stationary catalysts used in industry, only those used for ammonia oxidation emit significant amounts of platinum (WHO, 1991).

Vehicle traffic is the main source of contamination with platinum-group elements (PGE) to the urban environment. The emission of fine PGE-containing particles and their occurrence in urban areas suggest the possibility for long-range transport. A considerable increase in Pt concentrations in snow cores from remote areas as Greenland, has been reported (Barbante et al., 2001). An assessment of PEG deposition in the northern hemisphere indicates that relatively large fractions of PGE emitted from catalysts are transported at both regional and global scales (Rauch et al., 2005).
Monitoring data

Pt is not included in the national annual programmes for monitoring air and precipitation in Norway, Sweden or Iceland.

Automobile catalysts were introduced in Germany and UK in 1984 and 1993, respectively. Since then, investigations of PGEs (Pt, Pd and Rh) in different environmental samples such as air, road dust, soil, plants and water have been undertaken and several studies from different countries are published. The data shows some discrepancies that may be due to, e.g., traffic density, weather conditions, sampling methods and period of use of catalytic converters, affecting the content of PGEs. However, as a general trend, elevated concentrations of PGEs are reported in airborne particles. Pt seems to be associated with a wide range of particle diameters (Alt et al., 1993; Gomez et al., 2002), however, in particles < 10 µm diameter, the highest concentrations are associated with the fraction < 0.39 µm. Elevated Pt-content in vegetation growing near main roads has been reported. Different types of plants have been cultivated on authentic soil-material from a German highway to study bioavailability. Pt was detected in all types of grown plants, and the transfer coefficients shown to be comparable to the values for Cu (Schafer et al., 1998).

A selection of reported data for determination of Pt in environmental samples is presented below.

Air:

The average Pt content in airborne particles sampled in the city centre of Dortmund, was in the range of 0.02-5.1 pg/m³ (Alt et al., 1993), while it was in the range 7.3–13.1 pg/m³ in downtown Madrid, Göteborg and Rome, and 4.1 pg/m³ in Munich (Gomez et al., 2002). The Pt content (in pg/m³) in airborne particulate matter samples in Buenos Aires was found to vary from 2.3 to 47.7, with a mean value of 12.9 ± 7 (Bocca et al., 2006).

Road and tunnel dust:

Average Pt content in dust from two tunnels in Frankfurt, Germany was in the range of 165-198 ng/g (Helmers et al., 1998). Dust from a tunnel in Graz, Austria, held a Pt-content of 55 ng/g in 1994 and 81 ng/g in 1998 (Schramel et al., 2000; Zischka et al., 2002). Three different studies of Pt contents in road dust were undertaken in UK in the period of 1995-1999. The Pt concentration was in the range of 7-335 ng/g (Farago et al., 1996; Higney et al., 2002). In Białystok, Poland, average Pt content in tunnel dust was 23.3 ng/g in the particle fraction < 75 µm, while road dust varied from 34-110 ng/g (Lesniewska et al., 2004).

Soil:

Surface soil sampled along different roads with various traffic densities in Athens, Greece, showed Pt concentrations in the range of 2-141 µg/g (Riga-Karandinos et al., 2006). Surface soil sampled in distances of 0.6-3 m from a highway in Mainz, Germany, showed Pt concentrations decreasing from 87 to 2.5 ng/g (Muller and Heumann, 2000). The Pt-concentrations in soils adjacent to main roads in Sao Paulo were in the range 0.3-17 ng/g (Morcelli et al., 2005).

Road run-off:

Pt content in surface sediments of an infiltration basin and wetlands receiving run-off from roads in Perth, Australia were 9.0–104 ng/g, with the highest concentrations typically found in the topographic low point of the basins (Whiteley and Murray, 2005).
Biota:

Roadside wild carrot (*Daucus carota*) sampled at four heavy traffic locations in USA, showed an average Pt content of 14.6 μg/g (Gagnon et al., 2006). Average concentration of platinum content in grass samples (*Poa trivialis*) collected at location with high traffic density in Białystok, Poland, was 9 ng/g (Lesniewska et al., 2004). Pine needles were collected in Palermo, Italy, and Pt-concentrations varied between 1 and 102 ng/g (Dongarra et al., 2003).

Evaluation of need for monitoring

The emission of platinum has increased markedly during the last two decades due to introduction of catalyst in vehicles. The bioavailability and high tolerance of plants to platinum indicate a serious risk for platinum entering the food webs, although the levels found to date are not considered to be any health risk per se (Gomez et al., 2002; Gagnon et al., 2006). With an annual screening of urban air and road dust it is possible to measure changes from today’s concentration.

Run-off from car demolition plants may also contain platinum compounds. In this case, choices of matrices would be surface water, effluents from the plants and surface water sediments from the surroundings. Possible emissions of platinum containing compounds from medical and dental applications may be discovered in municipal wastewater. Matrices of choice would be wastewater and sludge.

Analysis

There are several techniques for determination of Pt in environmental samples. Neutron activation analysis (NAA) is highly sensitive and accurate, but less available for most laboratories. Both high- and low-resolution inductively coupled plasma mass spectrometry (ICP-MS, ICP-HRMS) may be used. However, hafnium causes interference on all Pt isotopes. Using ICP-MS, a mathematical correction for the contribution from HfO⁺ has to be included. By the use of ICP-HRMS in high resolution mode (Δm/m ~10000) the Pt peak may be resolved from the peak caused by HfO⁺. Using plasma mass spectrometry, Pt may be analysed together with most other heavy metals.
6.2 Silver

Silver is a white, ductile metal that belongs to the rare elements and the estimated average concentration in the earth's crust is 100 µg/kg. Silver can occur in the pure metallic form, but mostly as sulphide in the mineral argentite, Ag₂S. The electrical and thermal conductivity of silver are higher than those of other metals. Important alloys are formed with copper and mercury. Metallic silver is insoluble in water, but some silver salts, such as AgNO₃, are soluble. Silver forms complexes with chloride, ammonia, thiosulphate, cyanide and dissolved organic matter. Silver occurs in oxidation state +1 in almost all silver compounds.

<table>
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<tr>
<th>Abbreviation</th>
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<td>Ag</td>
<td>Silver</td>
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<td>Silver chloride</td>
<td>7783-90-6</td>
</tr>
<tr>
<td>AgBr</td>
<td>Silver bromide</td>
<td>7785-23-1</td>
</tr>
<tr>
<td>Ag(NO₃)</td>
<td>Silver nitrate</td>
<td>7761-88-8</td>
</tr>
</tbody>
</table>

**Characteristic data**

- Atomic no: 47
- Atomic weight (Da): 107.870
- Density (g/cm³): 10.490
- Melting point (°C): 961.8
- Water solubility: insoluble (Ag); 1.93 mg/L (AgCl); 0.14 mg/L (AgBr); 2.2 x 10⁶ mg/L (Ag(NO₃))

**Toxicological data**

- LC₅₀ (96-h trout): 6.5-13 µg/L
- LC₅₀ (96-h flatworm): 30 µg/L
- LC₅₀ (marine bacteria): 3000 µg/L
- LC₅₀ (96-h Marine Scallop): 100 µg/L
  (Ratte, 1999; Ward and Kramer, 2002; Bianchini et al., 2005).

Metallic silver and insoluble silver compounds appear to pose minimal risk to human health (WHO, 1977; ATSDR, 1990; Drake and Hazelwood, 2005) Silver in any form is not thought to be toxic to the immune, cardiovascular, nervous or reproductive systems, and there is no scientific evidence of silver to be carcinogenic (Drake and Hazelwood, 2005). Perhaps the most prominent effect of silver is discoloration of skins and the eyes. Prolonged ingestion, inhalation or dermal absorption of silver and its compounds may cause development of a characteristic, irreversible, bluish-grey discoloration of the skin and organs (argyria) and/or eyes (argyrosis). The discoloration is most prominent in areas exposed to sunlight (WHO, 1977; Gulbranson et al., 2000). The pigmentation is not toxic per se, but is considered as an undesirable health effect.

Water soluble silver compounds may in addition to agyria and argyrosis cause other health effects at high doses and chronic occupational exposure, such as irritation of the eyes, skin, respiratory and intestinal tract, kidney and lung damage (WHO, 1977; Klaassen, 1996). International expert groups have evaluated safety limits of occupational exposure to both soluble and insoluble silver. The most recent recommendations differ between soluble and insoluble silver. A threshold limit value of 0.01 mg/m³ and 0.1 mg/m³ are suggested for soluble silver and insoluble silver compounds respectively (Drake and Hazelwood, 2005).

Water soluble silver compounds, such as AgNO₃, are well known to have antibacterial properties, and silver in combination with sulfadiazine is extensively used as an anti-bacterial agent for the treatment of burns. Anti-bacterial activities include reactions with thiol-groups of proteins, binding to DNA and cell wall, and electron transport (Furr et al., 1994; Liau et al., 1997; Russell, 1997). Novel silver based antibacterial agents are developed (Dias et al., 2006). Silver ions may bind to DNA and it has been suggested that some silver
compounds are mutagenic, but as of yet no firm evidence of this has been reported.

Silver in its ionic form is highly toxic to aquatic animals and plants (WHO, 2002). Mortality of juvenile Rainbow trout has been reported at concentrations less than 5 µg/L in acute/sub acute experiments. The main mechanism of acute silver toxicity in freshwater fish is by inhibiting Na/K-ATPase activity and thereby blocking Na+ and Cl− uptake in the gills, which will disrupt the osmoregulation and ionic regulation (WHO, 2002). At concentrations of 1-5 µg/L it is observed mortality of several keystone species, such as amphipods and daphnids (WHO, 2002).

Chronic lowest- and no-observed-effect concentrations (LOECs and NOECs) for fish and invertebrates indicate effects at concentrations higher than 0.1 µg free silver/L. Ionic silver, however, is rapidly complexed to suspended materials, which will considerably reduce its bioavailability and toxicity. Silver is also less toxic in seawater, which may be attributed to high concentrations of salts and less concentration of freely dissociated silver ions. The most toxic silver ion is silver nitrate (AgNO₃), which tend to be dissociated in water, whereas silver compounds such as silver thiosulfate, silver chloride, and silver sulphide are shown less toxic (WHO, 2002).

Some acute toxicity data on mammals is available (WHO, 1977; WHO, 2002). Intravenous administration of 50 mg/kg bw is lethal to dogs. In drinking water, 1590 mg/L silver nitrate for 37 weeks is lethal to rats. Signs of poisoning observed in animals receiving high doses of silver included liver- and kidney damage, reduced haemoglobin levels and histopathological changes in the brain (WHO, 1977; WHO, 2002). An LD₅₀ value of 50 mg/kg bw silver nitrate was observed during a 14 day period in mice after oral administration. Intraperitoneal administration of 13.9 mg/kg bw is lethal to mice, and 20 mg/kg bw is lethal to rabbits (Ratte, 1999; Ward and Kramer, 2002; Bianchini et al., 2005).

Data on the toxicity of silver to marine organisms are limited. Silver speciation and acute toxicity in marine organisms are studied and other mechanism than uptake of free Ag+-ions via the gills are discussed (Ward and Kramer, 2002; Bianchini et al., 2005).

Freshwater fish and amphibians are the most sensitive vertebrates to dissolved silver. Toxicity of silver in water depends on the concentration of free Ag+-ions. Water characteristics such as pH, hardness, salinity, presence of complexing agents and dissolved organic matter are important parameters that has an impact on the concentration of free Ag+-ions, and thus the toxicity of silver (Ratte, 1999; Morgan et al., 2004a; Morgan et al., 2004b; Morgan et al., 2005a; Morgan et al., 2005b). Silver in its ionic form (Ag+) is highly toxic to freshwater rainbow trout (Ratte, 1999; Morgan et al., 2004a; Morgan et al., 2004b; Morgan et al., 2005a; Morgan et al., 2005b).

Additional toxicity values for aquatic organisms are listed by Ratte (1999). At concentrations normally encountered in the environment, food-chain biomagnification of silver in aquatic systems is unlikely, and there is per se no evidence of substantial biomagnification of silver in aquatic organisms (Ratte, 1999). Elevated silver concentrations in biota occur in the vicinities of sewage outfalls, electroplating plants, mine waste sites, and silver iodide-seeded areas (http://www.inchem.org).

Degradation in the environment

The global biogeochemical movements of silver are characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources.
Most of the silver lost to the environment will remain in soil, sediments, or wastewater sludge at the emission site. Silver is immobilised by precipitation to insoluble salts, complexation or adsorption to clays, organic matter or manganese and iron oxides (Ratte, 1999). Ag released into the atmosphere is associated to fine particles that are subjected to long-range transport (Lin et al., 2005; Adachi, 2006).

**Use in Norway**

Silver has a variety of applications:

- Electrical and electronic products
- Photography
- Jewellery and tableware
- Dental alloys for fittings and fillings
- Antibacterial/antibiotic treatment of serious burns
- Antibacterial agent in form of nano particles
- Seeding of clouds to produce rain
- High capacity Ag-Zn –and Ag-Cd batteries

In 2005, the world total silver demand was 24.5 Mkg, and the main silver applications were:

- Industrial applications: 11.6 Mkg
- Photography: 4.7 Mkg
- Jewellery and silverware: 7.1 Mkg
- Coins and medals: 1.1 Mkg

(http://www.silverinstitute.org/publications/wss06summary.pdf)

The use of silver treads, silver ions or silver nano-particles as antibacterial agents is growing. Silver seems to conquer the position earlier held by Triclosan as antibacterial agent (KemI, 2005). Nano silver is used in a variety of products such as washing machines, toothpaste, shampoo, body care products, textiles, disinfecting sprays, air condition systems etc.

**Emissions**

No total emission data for Norway is available.

Calculated annual transport of silver from the Kongsberg silver mine; Christian 7. stoll (horizontal pit) and Underberg stoll are 0.11 and 0.01 kg/year, respectively. Silver concentration measured in Kobberbergselva in 2002 was < 0.05 µ/L (http://www.miljostatus.no/templates/Pagewide_____4092.aspx).

In 2004, the estimated release to the environment in the USA via emissions, discharges, and waste disposal from sites listed in the Toxic Release Inventory were 254 260 kg for silver and 413 735 kg for silver compounds. (TRI, 2004; http://www.epa.gov/tri/).

At present the amount of silver released from washing machines appears unclear. One supplier claims that the emissions from their machines are in the range of 0.05-0.19 g Ag+/year. Swedish Water and Wastewater Association (SWWA) says this at least is a 2 to 4 fold enhancement of domestic silver emission. SWWA expresses concern for the impact an enhancement of domestic silver emission will have on the cleaning capacity in municipal wastewater plants and for the use of wastewater sludge within agriculture. Possible development of antibiotic resistant bacteria due to silver expose is also discussed.

**Monitoring data**

Today there is no ongoing annual monitoring of Ag or Pt in air or precipitation in Norway, Sweden or Iceland.

Atmospheric depositions of metals due to long-range transported pollution are monitored every fifth year since 1977, through a moss survey. Median concentrations of Ag reported were 0.07 ng/g in
1977, 0.03 ng/g in 1995 and 0.021 ng/g in 2000 (SFT, 2001; SFT rapport 838/01).

Measurements of silver in rivers, lakes, and estuaries using clean techniques show levels of about 0.01 µg/L for pristine, unpolluted areas and 0.01–0.1 µg/L in urban and industrialized areas (Ratte, 1999).

Trace metal concentrations at four sites that represent different degrees of anthropogenic, particularly vehicular traffic influence were determined. In addition, bioavailability was studied. The highest Ag concentration (0.11 ng/m³) was found in particle fraction 1.5-3.0 µm. Water-soluble mass fraction of Ag was found to be low (Birmili et al., 2006).

Reference and recent acid-leachable concentrations of some seldomly monitored trace elements (SMTE; Ag, Be, Ga, In, Sb and Tl) in sediments from four boreal oligotrophic lakes in a south to north transect in Sweden were determined by Grahn et al. (Grahn et al., 2006a; Grahn et al., 2006b). Concentration of Ag was 0.16-0.66 mg/kg dw. Increased concentrations of Ag were found in recent sediments, which infer an elevated loading of Ag. (Grahn et al., 2006a; Grahn et al., 2006b).

Evidence of food chain biomagnification of Ag in fish liver was observed when the Ag contents of abiotic and biotic compartments of different lakes of Nahuel Huapi National Park, Patagonia, Argentina were analysed (Guevara et al., 2005). The highest Ag concentrations in fish liver were 10 µg/g dw in brown trout and 29 µg/g dw in rainbow trout.

**Evaluation of need for screening**

Background silver concentrations found in Norway (SFT, 2001) are per se relatively low. An intermittent monitoring of metals in moss has been performed since 1977 and will reveal possible concentration changes. The moss survey represents mainly deposition from the air.

Emissions of silver containing compounds from medical and dental applications and processes of development of photographic films will be reflected in municipal wastewater. This could be monitored by the analysis of wastewater and sludge. Monitoring Ag in wastewater and sludge from industries producing formaldehyde and polyester or industries using silver as a catalyst for other applications seems particularly advisable.

**Analysis**

Total concentration of silver can be determined together with most other metals. There are several techniques suitable for silver determination depending on concentration level. The most commonly used techniques are:

- ICP-MS / ICP-HR-MS
- GF AAS
- ICP-AES
- F-AAS
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Morgan TP, Guadagno CM, Grosell M, Wood CM. Effects of water hardness on the physiological responses to chronic waterborne silver exposure in early life stages of rainbow trout (Oncorhynchus mykiss). Aquatic Toxicology 2005a; 74: 333-350.

Morgan TP, Guadagno CM, Grosell M, Wood CM. Effects of water hardness on toxicological responses to chronic waterborne silver exposure in early life stages of rainbow trout (Oncorhynchus mykiss). Environmental Toxicology and Chemistry 2005b; 24: 1642-1647.


Ward TJ, Kramer JR. Silver speciation during chronic toxicity tests with the mysid, Americanysis bahia. Comparative Biochemistry and Physiology C-Toxicology & Pharmacology 2002; 133: 75-86.


A literature survey on selected chemical compounds

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<td>Ingunn Skaufel Simensen</td>
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<td>A literature survey on selected chemical substances</td>
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<td>The Norwegian Pollution Control Authority (SFT) commissioned a literature survey of 14 compound groups, overviewing the available literature on polyfluorinated compounds (f.ex. PFOS), phosphor containing flame retardants, 3-nitrobenzanthrone, tin-organic compounds and the noble metals platinum and silver until December 2006. The survey provides the foundation on which decisions for the future needs for further screening will be made. Suggestions for geographical sampling locations and important sample compartments were also part of the study.</td>
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