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**FOODWEB**

FINNISH ENVIRONMENT INSTITUTE

# Summary of the selected contaminants

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## FOODWEB-project

The Baltic environment, food and health: from habits to awareness

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S Y K E

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## Dioxins (PCDD/F) and dioxin like PCBs (PCDD/F, DL-PCBs)

### Chemical properties

Dioxins are unintentionally formed by-products of anthropogenic activities. PCDD/F are formed for example in manufacturing of certain chemicals, incineration of municipal waste and the earlier bleaching of wood pulp and energy production through combustion. Dioxins are a class of structurally and chemically related to polyhalogenated aromatic hydrocarbons which mainly includes polychlorinated dibenzo-p-dioxins (PCDD), dibenzofurans (PCDF) and the “dioxin-like” biphenyls (dl-PCBs). There are 210 theoretically possible congeners in the group, including 75 polychlorinated dibenzo-p-dioxin (PCDD) and 135 polychlorinated dibenzofuran (PCDF) congeners.

Dioxins are very stable against chemical and microbiological degradation and therefore persistent in the environment. Only 7 of the 75 possible PCDD congeners, and 10 of the 135 possible PCDF congeners, those with chlorine substitution in the 2,3,7,8 positions, have dioxin-like toxicity. Likewise there are 209 possible PCB congeners, only 12 of which have dioxin-like toxicity.

### Occurrence in food items

About 95% of human exposure occurs through consumption of food of animal origin, with meat, dairy products and fish being the main sources. This is due to the fact that PCDD/Fs and dl-PCBs accumulate and biomagnify in animal and fish fat. PCDD/F are also found from eggs, vegetable oils, vegetable fats, animal fats.

### Environmental and health effects

PCDD/F and dl-PCBs are found to be teratogenic, mutagenic and carcinogenic for mammals and fishes. They can damage the immune, endocrine and nervous system of animals and also affect to re-productiveness negatively. PCDD/F and dl-PCBs are also potential carcinogenic and mutagens. They can damage immune system and liver and also change the balance of hormones.

## Recommendations

Scientific Committee on Food (SCF) in Europe established a tolerable weekly intake value (TWI) for dioxins and dioxin-like PCBs of 14 pg/kg toxic equivalent (WHO-TEQ) body weight. This TWI concurs with the lower end of the range TDI of 1-4 pg/kg WHO-TEQ body weight, established by the World Health Organisation (WHO).

## Non dioxin like PCBs (NDL-PCBs)

### Chemical properties

Polychlorinated biphenyls (PCBs) are chlorinated aromatic hydrocarbons, which were previously intentionally manufactured and due to their physical and chemical properties (such as non-flammability, chemical stability, high boiling point, low heat conductivity and high dielectric constants), were widely used in a number of industrial and commercial applications like in electric and hydraulic devices and lubricants e.g. coolants in transformer and condensation oil mixtures.

There are 209 theoretically possible congeners of PCBs that can be divided into different groups according to their biochemical and toxicological properties. Non-ortho and mono-ortho substituted PCBs show toxicological properties that are similar to dioxins. They are therefore often termed "dioxin-like PCBs". Most other PCBs do not show dioxin-like toxicity and are therefore termed "non-dioxin-like PCBs".

### Occurrence in food items

NDL-PCBs will accumulate in meat, liver and particularly fat tissues of farm animals. In addition, NDL-PCB will be transferred into milk and eggs. More than 90% of the NDL-PCB exposure in the general population is via food. NDL-PCBs are also found from fish and edible offal like liver.

### Environmental and health effects

Long-term effects of PCBs from human and laboratory mammal studies include increased risk of cancer, infections (damages the immune system), reduced cognitive function accompanied by adverse behavioral effects (hypothyroidism, infertility, ischemic heart disease, hypertension, diabetes, liver disease, asthma and arthritis), as well as giving birth to infants of lower than normal birth weight. There are also indications that PCB is involved in reproductive disorders in the marine top predators seal and bald eagle.

### Recommendations

TDI for PCB<sub>7</sub> according to AFFSSA is 0.01 µg/kg/bw/day.

## FURANS

### Chemical properties

Furans are formed while thermal food processing or domestic cooking.

### Occurrence in food items

Furans are present in a variety of heat-treated commercial foods for adults and infants. The highest furan concentrations were found in coffee brews and jarred baby food containing vegetables.

Furans can also be detected from other prepared (canned) food, ready-to-eat soups, (fried) chicken, soya, rapeseed oil, cereal-based products, bread and fruit juice.

### Environmental and health effects

Furans can cause air, soil and water pollution and it has been classified as possibly carcinogenic for humans. Furans can cause damages to the immune, nervous, and endocrine systems and reduction in reproduction potential. It also damages the fetus and infant.

### Recommendations

TDI -value for furans according to US EPA is 1 µg/kg/bw/d.

## POLYAROMATIC HYDROCARBONS (PAHs)

### Chemical properties

Polycyclic aromatic hydrocarbons (PAHs) constitute a large class of organic compounds that are composed of two or more fused aromatic rings. They solely consist of carbon and hydrogen and do not contain hetero atoms. They are primarily formed by incomplete combustion or pyrolysis of organic matter during various industrial processes. PAHs generally occur in complex mixtures which may consist of hundreds of compounds.

### Occurrence in food items

Humans are exposed to PAHs by various pathways. While for non-smokers the major route of exposure is consumption of food, for smokers the contribution from smoking may be significant. PAHs can contaminate foods during industrial smoking, heating and drying processes that allow combustion products to come into direct contact with food. Contamination of cereals and vegetable oils (including seed oils and olive residue oils) with PAHs usually occurs during technological processes like direct fire drying, where combustion products may come into contact with the grain, oil seeds or the oil. PAHs are also formed as a result of certain home food preparation methods, such as grilling, roasting and smoking. PAHs can be detected for example from smoked fish, smoked meat, fried meat, dried fruit, oils, vegetables, cereals, sweets, coffee.

### Environmental and health effects

PAHs have moderate to low acute toxicity. PAHs are carcinogenic and cause respiratory and cardiovascular diseases. In most studies, the site of tumour development was related to the route of administration, e.g. gastric tumours after oral administration, skin tumours after dermal application. PAHs are poorly absorbed and they are extensively metabolised in mammals and do not bioaccumulate. Different metabolic pathways can lead to highly reactive intermediates involved in the mutagenic/carcinogenic process of PAHs.

### Recommendations

Not available TDI.



# ACRYLAMIDE

## Chemical properties

Acrylamide ( $\text{CH}_2=\text{CHCONH}_2$ ) may be formed in foods, typically carbohydrate-rich and protein-low plant commodities, during cooking or other thermal processing such as frying, baking or roasting at temperatures of 120 °C or higher.

## Occurrence in food items

Acrylamide forms typically from carbohydrate-rich and protein low plant commodities during thermal food processing at too high temperature like in frying, baking and roasting. Acrylamide is detected in potato chips, French fries, plums, black olives, coffee, pastry, cookies, bread, breakfast cereals, jarred baby foods.

## Environmental and health effects

Acrylamide is carcinogenic and it has also neurotoxic and genotoxic effects.

## Recommendations

The oral tolerable daily intake (TDI) for acrylamide according to US EPA is 0.2 µg/kg/bw/d.

## ORGANO TINS; TRIBUTYL- AND TRIPHENYL TIN (TBT / TPhT)

### Chemical properties

Organotins are used (especially tri-substituted compounds) in antifouling paints for boats and cooling towers, preservatives for wood, cotton, textiles, paper and stain for buildings, slimicides in industrial process, molluscicides to prevent schistosomiasis and in the case of triphenyltin (TPhT), as a fungicide in agriculture.

### Occurrence in food items

The most important source of organotin exposure, to the general population, is from food and in particular fish and other seafood.

### Environmental and health effects

TBT and TPhT are highly toxic to aquatic organisms and furthermore, tend to bioaccumulate through the food chain (in particular in fish and seafood). TBT and TPhT cause masculinization in female snails ("imposex") and in fish at low concentrations (1 ng/L in water), suggesting that these compounds are endocrine disruptors. Organism's capability to metabolize organotins, can vary quite much. Molluscs for example can accumulate loads of these compounds due to their weak xenobiotic metabolism whereas fishes can degrade TBT quite efficiently. It has been noted that TPhT metabolism in fishes is much weaker than their TBT metabolism.

### Recommendations

TDI as a group for TBT, DBT, TPhT and DOT 0.25 µg/kg/bw/d has been established.

## KADMIUM (Cd)

### Chemical properties

Cadmium occurs naturally in the environment in its inorganic form as a result of volcanic emissions and weathering of rocks. It is found mainly associated with zinc (Zn) and to a lesser extent with lead (Pb) and copper (Cu). It is thus an inescapable by-product of the metallurgy of these elements. Cadmium is used in many technological applications and is released into the environment by smelting of other metals, burning of fossil fuels, incineration of waste materials and by the use of phosphate and sewage sludge fertilizers.

### Occurrence in food items

The consumption of food is the main source (accounts for approximately 90 %) of Cd exposure for the non-smoking general population. Smokers have on average twice the body burden of a non-smoker. The major part of the dietary cadmium exposure were cereals and cereal products, vegetables, nuts and pulses, starchy roots or potatoes, meat and meat products, fish, other seafood, fungi,

### Environmental and health effects

Especially endangered species to cadmium exposure are herbivore cattle (cows) and earthworms. In water column Cd accumulates in mollusks, lobsters and fish. Freshwater organisms are found to be more sensitive than marine organisms. Cadmium absorbed into the body is eliminated very slowly, with a biological half-life estimated to be 10-30 years. Cadmium exposure has been associated with nephrotoxicity, osteoporosis, neurotoxicity, carcinogenicity and genotoxicity, teratogenicity, and endocrine and reproductive effects. Newer data on human exposure to cadmium have been statistically associated with increased risk of cancer such as in the lung, endometrium, bladder, and breast.

### Recommendations

TWI of cadmium is 2.5 µg/kg/bw and TDI 0.4 µg/kg/bw/d.

## MERCURY (Hg)

### Chemical properties

Mercury is emitted into the environment from a number of natural (volcanic activity) as well as anthropogenic sources. The most important sources of direct emissions to water are dental amalgam use, chlorine-alkali industry and power plants, ferrous and non-ferrous industries, waste disposal, batteries, measuring and controlling (eg, thermometers) equipments, lamps, electronics, laboratory chemicals and pharmaceuticals, gold and silver recovery, paper or film coating photographic applications, burning of fossil fuels in power plants, crematoria, zinc (Zn) and copper (Cu) production. Atmospheric emissions and deposition is of high importance when it comes to sources of mercury and forms presently the greatest risk for environments even far from emission sources due to long range transport. Methyl-Hg is formed naturally mainly in the freshwater and marine environments.

### Occurrence in food items

Mercury is widely distributed within food but methyl mercury, it's most toxic form, is found at significant levels only in fish and seafood products. Exposure to mercury from food sources other than fish and seafood products is not relevant in the present context because they are containing inorganic mercury and would not contribute to the exposure to methyl mercury. Mercury can accumulate in organisms mainly by food chain, but also by respiration.

### Environmental and health effects

Inorganic mercury in food is considerably less toxic than methyl mercury. Methyl mercury is highly toxic particularly to the nervous system, and the developing brain is thought to be the most sensitive target organ for methyl mercury toxicity. It can also damage kidneys, causes depression, irritability, memory disturbance and spasms.

### Recommendations

Provisional Tolerable Weekly Intake (PTWI) for methyl mercury is 1.3 µg/kg/bw.

## LEAD (Pb)

### Chemical properties

Lead occurs naturally in the environment, but its industrial use (e.g. mining, smelting, processing, use in plumbing solders and alloys, pigments, batteries, ceramics, electricity and heat production etc.) has resulted in increased levels in soil, water and air. Emissions from waste incinerators were important contributors to lead in the environment, as was leaded fuel in the past years.

### Occurrence in food items

Human exposure to lead can occur via food, water, soil, dust and air. In the environment, inorganic lead predominates over organic lead. Cereal products, followed by potatoes, cereal grains (except rice), cereal-based mixed dishes and leafy vegetables and tap water were the most important contributors to lead exposure in the general European population

### Environmental and health effects

Lead is absorbed more in children than in adults and it accumulates in soft tissues including liver and kidneys. It can also accumulate to bone tissue, with increasing age. Lead affects virtually every system in the body, including the blood, the cardiovascular, renal, endocrine, gastrointestinal, immune and reproductive systems. Nevertheless, the most critical target for lead appears to be the central nervous system, particularly the developing brain, Lead is classified as a carcinogen

### Recommendations

The CONTAM Panel concluded that the current PTWI of 25 µg/kg/bw is no longer appropriate as there is no evidence for a threshold for critical lead-induced effects (EFSA Journal 2010).

## ARSENIC (As)

### Chemical properties

Arsenic is found in many metal rich geological materials and it is obtained as a by-product of the production of e.g. copper (Cu), lead (Pb), cobalt (Co) and gold (Au). Anthropogenic sources of arsenic releases to the environment include both: industrial emissions, mainly non-ferrous mining and smelting and metal using industry, the production of energy from fossil fuels and fertilizers.

### Occurrence in food items

The dominating pathway for arsenic to the terrestrial leafy vegetables is by direct atmospheric deposition, while arsenic in the root crops is a result of both soil uptake and atmospheric deposition. Food products of terrestrial origin generally contain low concentrations of total arsenic but have often a higher proportion of inorganic arsenic. The food subclasses of cereal grains and cereal based products, followed by food for special dietary uses (seaweed), bottled water, coffee and beer, rice grains and rice based products, fish, vegetables (root vegetables) and mushrooms were identified as largely contributing to the inorganic arsenic daily exposure in the general European population. Arsenic has also been detected from meat, chicken, juice concentrates and dairy products. Reported data on total arsenic content in food commodities indicate that fish and fish products, seafood, including seaweed are the major worldwide food sources of total arsenic.

### Environmental and health effects

Arsenic has different inorganic and organic forms, of which the inorganic ones are more toxic. Inorganic arsenic has been classified as carcinogenic to humans. The main adverse effects reported to be associated with long term ingestion of inorganic arsenic in humans are skin lesions, cancer, developmental toxicity, neurotoxicity, cardiovascular diseases, abnormal glucose metabolism, and diabetes. Neurotoxicity is mainly reported with acute exposure from deliberate poisoning or suicide, or at high concentrations in drinking water. Evidence of cardiovascular disease (Blackfoot disease, peripheral vascular disease, coronary heart disease, myocardial infarction and stroke) and diabetes in areas with relatively low levels of inorganic arsenic exposure is inconclusive.

### Recommendations

Tolerable daily intake, an oral TDI, is set for 1 µg/kg/bw/d.

## Polybrominated diphenylethers (PBDEs)

### Chemical properties

PBDEs are a class of brominated hydrocarbons with a basic structure consisting of two phenyl rings linked by an oxygen atom. There are 209 possible compounds, commonly referred to as PBDE congeners, which differ in the number and position of the bromine atoms in the two phenyl rings. Three commercial technical mixtures of PBDEs, pentaBDE (including congeners -28, -47, -99, -154), octaBDE (including congeners -183, -203) and decaBDE (including congener -209), have been marketed under different trade names. PBDEs are used as additives in flame retardants which are generally used at concentrations between 5 and 30 % by weight in plastics, textiles, electronic castings and circuitry. PBDEs are ubiquitously present in the environment and likewise in biota and in food and feed.

### Occurrence in food items

The dominant food category, containing PBDEs, is “fish and other seafood (including amphibians, reptiles, snails and insects)”, followed by “meat and meat products (including edible offal)” and “animal and vegetable fats and oils”, “milk and dairy products” and “eggs and egg products”. The levels of BDE-209 were the highest in almost all food categories except for “fish and other seafood”. In the “food for infants and small children”, BDE-47 was the congener with the highest levels.

### Environmental and health effects

PBDEs can accumulate in blood, breast milk and adipose tissue. PBDEs can cause liver and nerve damages and they disturb also the hormonal balance.

### Recommendations

In Europe, due to the limitations and uncertainties in the current data base on PBDEs, the derivation of a health based guidance value (TDI) was not appropriate.

## Perfluorinated compounds (PFC): Perfluorooctane sulfonate (PFOS) and Perfluorooctanoic acid (PFOA)

### Chemical properties

Perfluoroalkylated substances (PFAS) is the collective name for a vast group of fluorinated compounds, including oligomers and polymers, which consist of neutral and anionic surface active compounds with high thermal, chemical and biological inertness. Perfluorinated organic compounds have been widely used in industrial and consumer applications including stain- and water-resistant coatings for fabrics and carpets, oil-resistant coatings for paper products approved for food contact, fire-fighting foams, mining and oil well surfactants, floor polishes, and insecticide formulations.

### Occurrence in food items

Human exposure to PFAS compounds, including PFOS and PFOA, is likely to occur via a number of different routes like ingestion of non-food materials, dermal contact and inhalation. PFOS has been shown to bio accumulate in fish and other seafood, fowl, meat, fruits, vegetables, eggs, milk and dairy products. Other possible sources could be related to food via packaging material or cookware or more direct exposure from the techno sphere (e.g. household dust).

### Environmental and health effects

PFC compounds are found to be carcinogenic and they can damage immune and hormonal systems. They also increase the risk of anorexia, blood cancer, high cholesterol level, hyperactivity, liver damages.

### Recommendations

The CONTAM Panel has established a TDI for PFOS of 0.15 µg/kg/bw/d and for PFOA of 1.5 µg/ kg/bw/d. PFOS is listed in Stockholm Convention.



## MYCOTOXINS

### Chemical properties

Mycotoxins are secondary metabolic products synthesized by fungi. There are various kinds of mycotoxin groups like aflatoxins, ochratoxins and fusarium toxins. Aflatoxins can occur in food and feed as a result of fungal contamination by moulds, primarily by *Aspergillus flavus* and *A. parasiticus*. Ochratoxin A (OTA) is a mycotoxin produced by several fungal species in the *Penicillium* and *Aspergillus* genera. A variety of *Fusarium* fungi, which are common soil fungi, are important plant pathogens growing on the crop in the field, producing a number of different mycotoxins of the class of trichothecenes (T-2 toxin, HT-2 toxin and deoxynivalenol (DON)).

### Occurrence in food items

Aflatoxins can be formed under warm and humid conditions. They are most likely to contaminate tree nuts (almonds, hazelnuts, pistachios, Brazil nuts, cashew nuts, walnuts, pecan nuts), ground nuts (peanuts), figs and other dried fruits, spices, crude vegetable oils, cocoa beans and maize. Ochratoxin contamination of food commodities includes cereals and cereal products, pulses, coffee, beer, grape juice, dry vine fruits and wine as well as cacao products, nuts and spices. Fusarium toxins can be found in various cereal crops (wheat, maize, barley, oats, and rye) and processed grains (malt, beer and bread). Ripening of crop, conserving semidry cereals, conserving other food products and food storage can affect to the formation of mycotoxins.

### Environmental and health effects

Aflatoxin B1 is genotoxic, carcinogenic and increasing evidence demonstrates that it also has the potential to affect the immune system, nutrition and growth. OTAs are found to be carcinogenic, nephrotoxic, teratogenic, immunotoxic and possibly neurotoxic and genotoxic. Fusarium toxins (DON, T-2 toxin and HT-2) toxins are toxic to all animal species as well as to humans. They are found to affect to the apoptosis of proliferating cells including bone marrow cells (inhibition of haematopoiesis) and cells of the immune system (lymphoid depletion) account for the systemic toxicity following dietary exposure. Acute/subacute toxicity of DON is characterised by vomiting (vomiting is seen in pigs, whereas delayed gastric emptying has been observed in rats and mice), feed refusal, weight loss and diarrhoea.

## Recommendations

For OTA exposures the range of tolerable daily intake (TDI) would be below 5 ng/kg/bw/d.

For fusarium toxins (DON) a provisional maximum tolerable daily intake (PMTDI) of 1 µg/kg/bw/d on the basis of the no observed-effect level (NOEL) of 100 µg/kg bw per day.

TDI for the sum of T-2 and HT-2 toxins is set to be 0.1 µg/kg/bw/d.

## GLYKOALKALOIDS

### Chemical properties

Glycoalkaloids are a group of nitrogen-containing compounds that are naturally produced in various cultivated and ornamental plant species of the Solanaceae family. This large family of plants includes commonly consumed vegetables such as potatoes, tomatoes, eggplants, and peppers. Examples of non-food plants in the Solanaceae family include tobacco, petunia, and climbing or bittersweet nightshade. Glycoalkaloids are self synthesise by plants for protection against diseases and insects.

### Pathway to food items

$\alpha$ -Chaconine and  $\alpha$ -solanine occur naturally in potatoes (*Solanum tuberosum*) and other members of the Solanaceae family. Solanine is also present in apples, bell peppers, cherries, sugar beets, and tomatoes. Potatoes that have been exposed to light in the field or during storage may become green, due to an accumulation of chlorophyll. This greening may affect only the surface (peel) or it may extend into the flesh of the potato. Exposure to light is only one of the stress factors affecting potatoes. Alpha-solanine and alpha-chaconine, gives the potatoes a bitter taste. These natural toxicants (stress metabolites) have insecticidal and fungicidal properties.

### Environmental and health effects

Glycoalkaloids are toxic to humans if consumed in high concentrations. They can cause different symptoms in digestive and nervous system: ulcer of gastrointestinal tract, damages of mucosa, bleedings, stomach ache, constipation, diarrhoea, apathy, drowsiness, ataxia, muscle weakness, cramps, unwanted urination, paralysis, coma, respiratory arrest, nausea, vomiting, abdominal cramps, burning sensation in the throat, headache, dizziness, fever, sensory impairment, hallucinations and hypothermia. It is believed that eating the high content of potatoes during pregnancy affects fetal development.

### Recommendations

There is no TDI-value at the moment.

## NITRATE / NITRITE

### Chemical properties

Nitrate is a naturally occurring compound that is part of the nitrogen cycle, as well as an approved food additive.

### Pathway to food items

Nitrate is formed naturally in living and decaying plants and animals, including humans. Human exposure to nitrate is mainly through the consumption of vegetables, and to a lesser extent water and other foods. Nitrate is also formed endogenously. In contrast exposure to its metabolite nitrite is mainly from endogenous nitrate conversion. Nitrate is also used in agriculture as a fertilizer to replace the traditional use of livestock manure and in food processing as an approved food additive.

Some vegetables, particularly leafy vegetables, have been shown to have relatively high levels of nitrate, but implications for food safety are unclear (oak leaf lettuce, rucola etc). It is worth mentioning that also some leafy herbs like mint, oregano and thyme contained high nitrate levels, but would on the other hand be consumed only in low amounts. Other food items containing nitrates are tomato, potato and eggplant, meat, cheese, celery, lettuce, spinach, beet, radish, rucola, Chinese cabbage, dill, kohlrabi, parsley, leek, cabbage, rutabaga, carrot, cauliflower, cucumber, pumpkin, tomato, potato, mushrooms, onion, garlic, berries, fruits, cereals and pulses.

### Environmental and health effects

Nitrate per se is relatively nontoxic, but its metabolites, nitrite, nitric oxide and N-nitroso compounds, make nitrate of regulatory importance because of their potentially adverse health implications. On the other hand recent research shows that its conversion to nitrite plays an important antimicrobial role in the stomach. The toxicity of nitrate is known to be low. The bonding ability of oxygen decreases due to modification of met haemoglobin.

### Recommendations

An Acceptable Daily Intake (ADI) for nitrate of 3.7 mg/kg/bw/d. ADI for nitrite is set for max 0.07 mg/kg/bw/d. (The EFSA Journal 2008). TDI to nitrate according to US EPA is 1.6 mg/kg/bw/d y and for nitrite 0.1 mg/kg/bw/d.