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Polychlorinated dibenzodioxins, dibenzofurans and PCBs - a toxicological approach

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Abstract

Dioxins and PCBs are extremely toxic environmental contaminants in very low concentrations and can get into food from air, soil or aquatic sediments. The reduction of residues of such persistent environmental contaminants, generically called dioxin-like compounds, both for the protection of the environment and for consumers, is now becoming of particular importance globally.

As several cases have been mentioned in recent years in which dioxins have been accidentally identified in the food chain, in this paper we propose a review of the main sources, pathogenic effects and symptoms induced by the human consumer, the maximum limits and possibilities to reduce them in food.

Keywords: polychlorinated dibenzodioxins, polychlorinated dibenzofurans, PCBs

1.Introduction

Dioxins and PCBs are persistent environmental contaminants, the result of by-products in many industries, as well as uncontrolled combustion (volcanoes, fossil fuels). They have been identified in almost all components of the environment, where, due to their chemical structure and resistance to biodegradation, they end up accumulating in the food chain, while retaining their toxic effects [10, 12, 14, 17].

The level of bioaccumulation in living organisms depends on the time of exposure, the dose ingested and the place occupied on the phylogenetic scale.

Human exposure to dioxin-like compounds is mainly due to the consumption of foods, especially of animal origin, and can lead to a variety of conditions, such as various types of cancer, reproductive, immune and hormonal disorders, and chloracne [11, 17, 19, 25, 31].

Vegetable products are not such an important source for humans, but rather for animals.

Some dioxin contamination events have been mentioned in the late years. In 1999 high levels of dioxins were reported in Belgium, in chicken meat and eggs and in 2008, in Ireland, were found up to 200 times the safe limit of dioxins in pork meat. There were also some feed incidents, such as with the dried citrus pulp pellets imported from Brazil for the feed of cows in Germany and Netherlands, which cause increase in milk levels.

After the Belgium incident, the EU authorities have implemented a number of measures to reduce the levels of dioxin-like contaminants in the food chain [10, 15].

2. Polychlorinated dibenzodioxins and dibenzofurans

Polychlorinated dibenzodioxins (PCDD) and polychlorinated dibenzofurans (PCDF), generically referred to as "dioxins", are two categories of tricyclic aromatic ethers that have up to 8 chlorine atoms attached to carbon atoms 1-4 and 6-9. There are a total of 75 possible PCDD congeners and 135 PCDF congeners.

Depending on the order of the chlorine atoms in the molecule, the toxicity of these substances is different.

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These compounds are not intentionally produced, but result as by-products in the development of chemical processes based on trichlorophenol (pesticide industry, steel, paints, paper bleaches). In the atmosphere, the main source is combustion processes, including the incineration of waste (municipal solid waste, sewage sludge, medical waste), the burning of various fuels (wood, coal, petroleum-derived fuels), uncontrolled combustion sources (burning of steps, volcanic eruptions) [4, 10,12, 14, 17].

Favorable conditions for the formation of dioxins are high temperature, the presence of UV rays and the presence of radicals in the reaction of the chemical process.

The most studied and toxic form of dioxin is 2,3,7,8-tetrachlor-dibenzo-p-dioxin (2,3,7,8 TCDD) (Figure 1):

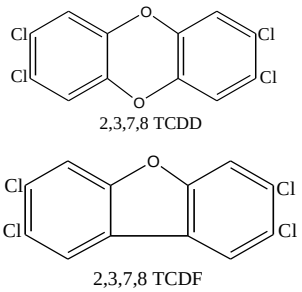

Figure 1. Chemical structure of 2,3,7,8 TCDD and 2,3,7,8 TCDF [10, 21]

Dioxins evaporate slowly, have poor water solubility, but dissolve easily in fatty substances and in organic matter with fatty properties. Dioxins do not react easily with other chemicals. Due to their chemical, physical and biological stability, dioxins persist in the environment for a long time.

The geographical distribution of dioxins depends on their source and mode of transport. The dioxin reservoirs previously formed by the release from the combustion processes, the deposition on the ground by volatilization and the transport of the material particles on which they were adsorbed and by the soil erosion processes re-release the sequestered dioxins. Atmospheric transport is the main mechanism of dioxin dispersion in the atmosphere.

Dioxins are removed from the atmosphere either by wet deposition (precipitation, fog) or by drying (sedimentation of material particles on which dioxins are adsorbed). Highly chlorinated compounds are removed from the atmosphere by deposition on the surface of soil, vegetation or water, and less chlorinated congeners are removed by atmospheric turbulence and diffusion [25, 26].

On the ground, dioxins are adsorbed on its particles, which can only be removed by the processes of erosion or washing by water. When deposited on the surface of water, dioxins adhere to water particles and deposit in sediment.

In air, water and soil, dioxins are resistant to degradation and hydrolysis, with only slight and slow degradation by photolysis and photooxidation.

Human exposure to dioxins is mainly achieved by transfer along the path: atmospheric emissions → air → deposition → terrestrial / aquatic food chain → human ration. The EPA estimates that the main contribution to dioxin intake is from the consumption of meat (especially beef), fish and shellfish, milk and dairy products [5, 9, 19, 24, 27].

Toxicity. Toxicity equivalents and toxicity equivalence factors

The biological activity of dioxins is variable, and because humans are exposed to a mixture of dioxins, the toxicity of an exposure depends on the composition of the mixture. The biological activity of different congener dioxins is compared with the activity of 2,3,7,8 TCDD, which is the most biologically active. The toxic potency of a mixture of dioxins is thus expressed in TCDD, or TEQ (Toxicity Equivalents) toxicity equivalents. For example, exposure to a mixture of dioxins with a potency of 2 ng TEQ / kg means that the total mixture is expected to have an exposure strength equal to 2 ng TCDD / kg. The TEQ value for a mixture is calculated by multiplying the mass or concentration of each dioxin by a Toxicity Equivalence Factor (TEF) and summing all the dioxins in the mixture. TEF is calculated by various biochemical and toxicological methods as a way to express the toxicity of dioxins in connection with TCDD.

The toxicity of dioxins is extreme, being ranked among the top 10 most toxic substances for humans. The LD50 for guinea pigs is 0.6 mg / kg body weight and for rats it is 22 mg / kg body weight [10, 22].

For humans, the minimum risk levels (MRLs) for TCDD are 200 pg / kg / day, 20 pg / kg / day, and 1 pg / kg / day. Acute MRL is based on

immunological effects in mice, intermediate MRL on immunological effects in guinea pigs, and chronic MRL on developmental effects in rhesus monkeys. WHO recommends a tolerable daily dose of 1-4 pg TEQ / kg / day and a tolerable weekly dose of 14 pg / kg. The lethal dose is 1 /g / kg body weight. It is estimated that in European countries the average intake of dioxins per adult ration is between 0.4 -1.5 pg TEQ / kg / day [10, 22].

Toxicokinetics

Because fats are absorbed efficiently in the gastrointestinal tract, dioxins administered in a lipid matrix are expected to be completely absorbed into the bloodstream. Experiments on rats showed approximately 90% absorption of 2,3,7,8 TCDF after oral administration of a single dose in a 1: 1 mixture of ethanol: vegetable oil and 70-85% absorption of 2,3,4,7,8 PeCDF. From the ration, which contains a much more complex matrix, 50-60% of a dose of TCDD was absorbed.

After absorption from the gastrointestinal tract, TCDD enters the lymph in the form of chylomicrons. Once in the blood, they are quickly removed, appearing mainly in the liver and adipose tissue (74 – 81% of th0e administered dose). After removal of the chylomicrons, the remaining dioxins bind to the serum proteins. In the liver, binding proteins play an important role in the intake of dioxins in the blood, even of poorly chlorinated congeners. When rodents are exposed to increasing doses of TCDD, preferential accumulation occurs in the liver microsomes, so that, unlike adipose tissue, a higher concentration is reached here several times. This fact has also been found in humans.

The elimination of unmetabolized dioxins by faeces is an important route of elimination in humans, with the contribution of fecal elimination to total elimination being between 14 (1,2,3,4,6,7,8 HpCDD) to 90% (OCDD) (Rohde et al. , 1999). The half-life of dioxins is between 6 and 11 years [10, 19, 21].

The mode of action of dioxins

Some mammalian cells, such as liver cells, contain a large molecule called the aryl-hydrocarbon (Ah) receptor, which can be imagined as a pocket. Some foreign compounds, such as dioxins, fit well in this pocket. Once in the pocket, the dioxins activate the Ah receptor, and the couple formed reaches the nucleus of the cell, which contains the body's genes. In the nucleus, the couple can either activate or suppress the genes that control the normal life cell cycle. For example, some cells may start to grow preferentially and others may not die, as is normal. It is important to note that gene activation or suppression is not the same as DNA damage. Cancer is a complex multistage process. If the Ah-dioxin receptor couple is present at high enough levels for a sufficiently long time in the cell nucleus, it can promote the development of cancerous cells [8, 10, 17].

Pathogenic effects and symptoms

Due to the usual and daily exposure to low doses of dioxins through food, breastfeeding (newborns), the epidemiological data on health effects are quite few. Regarding the carcinogenic effects of dioxins by general exposure, none of the studies conducted by specialized agencies (AEA Technology, 1999; ATSDR, 1998; EPA, 2000; Fiedler et al., 2000, IARC, 1997, Scientific Committee on Food, 2000, 2001) did not conclude that toxins were carcinogenic in this way [8, 25].

In contrast, adverse effects on childbirth weight, neurological development, neurobehavioral development, thyroid hormone status and the immune system in children have been reported. Prenatal exposure to dioxins has also been found to lead to cognitive and motor impairments, which may persist when the family environment is not optimal [11].

By being exposed to high doses of dioxins, international bodies have determined that they are human carcinogens. Liver cancers are more common, but they may have multiple locations.

Other effects found are:

- chloracnea and other dermal effects;
- changes or possible changes in glucose metabolism and the risk of diabetes;
- impaired thyroid function;
- deficiencies in growth and development, neurological and neurobehavioral development;
- elevated gamma-glutamyl transferase levels;
- altered concentrations of reproductive hormones in men;
- impaired liver function and liver effects;
- possible alterations in serum lipids and cholesterol levels;
- possible alterations in the concentration of alanine aminotransferase and aspartate aminotransferase;
- alterations of the immune system;

- possible eye damage;
- increased mortality from cardiovascular disease [4 ,9, 10, 22, 26, 31].

3. PCBs

PCBs (biphenyl-polychlorinated) are a class of chemical compounds in which chlorine atoms replace some or all of the hydrogen atoms in the biphenyl molecule. Their basic structure is shown in Figure 2:

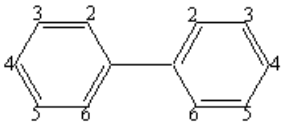

Figure 2. Basic structure of the PCB [1, 2, 29]

PCBs have been produced and marketed under different names: Arochlor, Pyranol, Pyrochlor (USA), Phenochlor, Pyralene (France), Clopehn, Elaol (Germany), Kanachlor, Santotherm (Japan), Fenchlor, Apirolio (Italy) and Sovol (USSR).

An important property of PCBs is that they are inert. PCBs are resistant to both acids and bases and are thermally stable, making them useful for many applications, including as dielectric fluids in transformers, heat transfer fluids, and lubricants.

At high temperatures, PCBs are combustible, and combustion products can be even more dangerous than the original products. Combustion products also include hydrochloric acid and PCDFs and PCDDs. PCDFs also result during PCB processing [1, 2].

In general, PCBs are insoluble in water, with the highest solubility being orthochlorinated congeners (5 mg / l). Solubility decreases rapidly in congeners with the ortho-free position, especially in those with the para-occupied position. PCBs are soluble in non-polar organic solvents and biological lipids [2].

Distribution in the environment

Evaporation can be an important means of transport in the environment for PCBs dissolved in surface water. The different volatility and solubility of congeners are expected to be the cause of their redistribution in surface water and sediment. In water, sediment adsorption and suspended particles are also a major partitioning product of PCBs between water and the solid phase. Subsequent adsorption and sedimentation can immobilize PCBs for a long time in aquatic systems, forming reservoirs from which PCBs are slowly released for a long time.

In the atmosphere, PCBs are removed by wet deposition (precipitation, fog) and dry (sedimentation of material particles on which they are adsorbed). By depositing PCBs in surface water, their level becomes 500 times higher in the upper layers than in the lower ones, so that the fish that feed at these levels bioconcentrate the PCBs more than those that feed at the lower levels. In water, PCBs bioconcentrate in the aquatic food chain, with fish containing the largest amounts.

In air, the vapor phase reactions of PCBs with hydroxyl radicals (formed by photochemical reactions from sunlight) are the dominant transformation processes. In water, as in soil, the biodegradation of PCBs is slow, both in aero and anaerobic conditions, depending on the proportion and location of chlorination, concentration, type of existing microbial population, available nutrients, temperature [1, 7, 13, 29].

Exposure

The concentration of PCBs in the air in urban areas is around 5 ng / m^3 (Eisenrich et al., 1992) and 0.2-0.95 ng / m^3 in rural areas (Hoff et al., 1992). In marine waters, the concentration of PCBs was between 0.01-0.7 ng / m^3. In drinking water the concentration is below 100 ng / l, and at a consumption of 2 l / day there is an exposure of less than 200 ng PCB per day through water.

The main route of exposure of the population to PCBs is through the consumption of contaminated food, especially meat, fish and chicken products, but also through air and drinking water [1, 7, 12, 20].

Toxicokinetics

Data on the toxicokinetics of PCBs in humans are limited to information from cases of accidental ingestion of food contaminated with PCBs and cases of occupational exposure by inhalation or dermal route. The administered PCBs can be absorbed by oral or dermal inhalation.

In the gastrointestinal tract, PCBs pass passively into lipophilic cell membranes and blood vessels and are absorbed by the lymphatic system via lipids. The major carriers of plasma PCBs are lipoprotein fractions.

Due to their lipophilic nature, especially highly chlorinated congeners, PCBs tend to accumulate in

lipid-rich tissues. The highest concentrations of PCBs are usually found in the liver, adipose tissue, brain and skin.

PCBs are converted to hydroxy and methyl sulfonate metabolites, which are not excreted but can be retained and stored in specific tissues and fluids in the body.

PCBs are metabolized by the monooxygenic-nasal microsomal system, catalyzed by cytochrome P450 into polar metabolites that may be conjugated to glutathione and glucuronic acid.

The apparent half-life of PCBs in the blood was found to be shorter in children than in their mothers after exposure by ration to Kanechlor; this can be explained by the growth of children (the arrangement of PCBs in the growth tissues, which leads to an accelerated decrease in the concentration of PCBs in the blood due to dilution rather than elimination) (Yakushiji et al., 1984). Prolonged enzymatic induction in occupational exposures may lead to shorter persistence of PCBs in professionally exposed individuals than in the general population.

The main routes of elimination of PCBs are through feces, urine and breast milk [6, 10].

Action mode

PCBs have a wide variety of mechanisms of action, which depend on how they replace chlorine in the molecule. The most notable difference in mode of action is due to the presence or absence of chlorine molecules at positions 2, 2´ and 6, 6´. Those PCBs that do not contain chlorine in the ortho position and have two adjacent pairs of chlorine in the meta and para positions have high binding affinity to Ah receptors, analogous to dioxins and dibenzofurans. Congenital PCBs that have 2 or more chlorine atoms in the ortho positions do not exhibit dioxin-like toxicity due to the lack of significant binding to Ah receptors.

Multi-ortho-substituted PCBs have other important mechanisms of action, including effects on neurological development, dopamine levels, and tumor promotion. It should be noted, however, that in general, the specific effects of multi-ortho-substituted PCBs are observed at higher doses than similar dioxin-induced PCBs associated with some non- or mono-ortho-substituted PCBs, which are strong agonists Ah receptors.

PCBs and endocrine disruptors bind to endocrine receptors leading to endocrine disruption and negative reproductive effects. Studies by Seegal et al. (1991) showed that mono- and di-ortho-substituted PCBs have a more pronounced effect on neurological development, toxicity probably mediated by a mechanism independent of Ah receptors.

PCBs also induce hepatocellular carcinomas, but the mechanism of action is unknown [1, 6, 19, 20, 21].

Pathogenic effects and symptoms

The link between PCB exposure and human health reflects the great diversity of human exposure to various congeners and contaminants present in PCB-contaminated products and by-products resulting from their combustion. Evidence suggests that exposure to PCBs is associated with an increased risk of cancer in the digestive tract, especially the liver and malignant melanomas. However, limited exposure data and, in some cases, the presence of other risk factors prevent a clear correlation between exposure and response. Exposure to PCBs is also associated with reproductive impairment, such as reduced growth rate, delayed development, and neurological effects (although some neurological impairments manifested at an early age may disappear during childhood) [21, 29].

Immunological changes, manifested as increased rates of infection and changes in circulating lymphocyte populations. Dermatological changes are also found, including chloracne and changes in the pigmentation of the skin, nails and gums, and deformities of the nails after exposure to highly chlorinated congeners.

The presence of dioxins in food

Due to the high cost of analyzing dioxins and similar compounds, there is relatively little information on their presence in food, but in recent years, analytical capacity has largely increased to complement the additional testing.

The most tested chemical methods for dioxin analyses are the multidimensional gas-chromatography and MS-techniques. In recent years, other methods were tested, such as ELISA technology and PCR technology, method by which dioxins bind to the Ah receptor in a specific manner and subsequently to a specific piece of DNA în so-called AhPCR kit [3, 18].

FDA analyzes show that they can be found in all foods, but only in products of animal origin, ie red

meat, fish and dairy products reach concentrations higher than 0.1 ppt [23, 24, 27, 28]. In general, high concentrations of dioxins and related compounds are associated with high amounts of fats, especially animal fats. The foods with the highest content of such compounds are fatty beef, ham, fatty cheeses, fatty fish (eg salmon) and butter.

Fruits, vegetables and cereals have significantly lower dioxin levels, but are consumed in large quantities, and these foods also contribute to total dioxin exposure (Douglass and Murphy, 2002, cited by the Committee on the Implications of Dioxin in the Food Supply Food and Nutrition Board, 4).

The content of dioxins and similar compounds in various types of food is presented in Table 1:

Table 1.Concentration of dioxins and similar compounds per gram in different types of food (according to the Committee on the Implications of Dioxin in the Food Supply Food and Nutrition Board, 4)

Food category	Dioxin content and similar compounds, TEQ (ppt)
Meat, fish, poultry, eggs	
Beef, pork, lamb	0,005 – 0,046
Processed meats	0,01 – 0,21
Fish, shells	0,01 – 0,33
Bird	0,004 – 0,06
Eggs	0,01 – 0,05
Dairy products	
Cheese	0,002 – 0,24
Cream / ice cream	0,0001 – 0,06
Milk	0,0006 – 0,01
Fats, oils, nuts	
Butter	0,22
Vegetable oils	0,002 – 0,06
NUTS	0,003 – 0,006
Bread and cereals	
Bread	0,001 – 0,05
Salty biscuits	0,001 – 0,02
Pasta	0,0001 – 0,02
Breakfast cereals	0,0007 – 0,01
Fruits vegetables	
Fruits	0,0007 – 0,01
Vegetables	0,0001 – 0,05

Influence of processing on the content of dioxin and similar compounds

Information on the penetration or generation of dioxins and similar compounds during food processing and packaging is limited. However, the analysis of current practices and processing may be useful in estimating the potential sources of these compounds entering food through these pathways.

There are many ways in which food can be processed, some of which can influence the dioxin content of food and some of which have no influence [4].

The processes that are unlikely to affect the content of dioxins and similar compounds are:

- grinding, mixing, modeling at room temperature;
- other processing techniques such as sorting, cutting and removing debris / cracks;
- flame peeling, used in onion processing, can generate dioxins and similar compounds due to high temperature processing, but can be removed later by washing;
- scalding, pasteurization, heat sterilization, baking and frying;
- freezing of fruits, vegetables and meat products and cooling and refrigerated storage of fresh or processed products;
- frying meat, fruits or vegetables, unless dioxins are introduced into the food through fat and oils contaminated during frying.

Processes that can influence the content of dioxins and similar compounds in food are:

- heat processing of meat that has been shown to reduce dioxin levels by fat loss (however, because high temperatures and other favorable conditions can produce dioxins, further studies are needed to determine whether high temperature processing during baking, extrusion, expansion and short-term pasteurization at high

temperature have an impact on dioxin levels in finished products);

- extraction and drying during food processing, in particular the extraction of fats and moisture. For example, a food whose moisture has been reduced will have a higher dioxin content than its mass, even if the amount of dioxins is the same as in the original product (eg dehydrated peas); the extraction of oils from foodstuffs may concentrate the dioxins in the original product into the oil intended for consumption (for example, the production of fish oil); certain forms of solvent extraction may concentrate existing dioxins or introduce them into solvent residues;
- the separation of raw milk during which the existing dioxins will be reduced in skimmed milk (aqueous phase) and will increase in cream (lipid phase);
- the sorting (including the removal of fat) of the meat, analogous to the extraction, reduces the dioxin content of the final product;
- filtration process may involve the use of dioxin-containing filtering agents that may become a source of food contamination; for example, the identification of 1,2,7,8 TCDF and 2,3,7,8 TCDD in coffee filter paper in Japan.

Processes such as irradiation, ozonation, UV light treatment, sunlight and chlorination have not yet been examined for their impact on dioxin levels in food.

While the focus on human exposure to dioxins has focused on the ingestion of animal products as the most likely source of human exposure to dioxins, processing and packaging may play an insignificant role in this regard [4].

Maximum permitted limits in food and feed

The maximum limits for dioxins and PCBs in food are given in Table 2.

The maximum limits for dioxins and PCBs in feed are given in Table 3.

Table 2. Maximum limits for dioxins and PCBs in foodstuffs [30]

Food category	**Maximum limits**	
	Sum of dioxins (WHO-PCDD/F-TEQ)	**Sum of dioxins and dioxins similar to PCBs (WHO-PCDD/F-PCB-EQ)**
1. Meat and meat products, excluding edible offal of the following animals: - cattle and sheep - bird - pig	 3 pg/g fat 2 pg/g fat 1 pg/g g fat	 4,5 pg/g fat 4 pg/g fat 1,5 pg/g fat
2. Liver of the animals referred to in point 1 and liver products	6 pg/g fat	12 pg/g fat
3. Fish and fish products, except eel. The maximum limits apply to crustaceans, except for brown crab meat and lobster head and thorax and other large crustaceans.	4 pg/g wet weight	8 pg/g wet weight
4. Eel meat and derived products	4 pg/g wet weight	12 pg/g wet weight
5. Raw milk and dairy products, including butterfat	3 pg/g fat	6 pg/g fat
6. Chicken eggs and egg products	3 pg/g fat	6 pg/g fat
7. The fat of the following animals: - cattle and sheep - bird - pig	 3 pg/g fat 2 pg/g fat 1 pg/g fat	 4,5 pg/g fat 4 pg/g fat 1,5 pg/g fat
8. Mixed animal fats	2 pg/g fat	3 pg/g fat
9. Vegetable oils and fats	0,75 pg/g fat	1,5 pg/g fat
10. Oils from marine life (fish oil, fish liver oil and other organisms intended for human consumption)	2 pg/g fat	10 pg/g fat

Table 3. Maximum values for feed products (status as of 28th November 2019) [16, 32]

Products intended for animal feed	**Dioxins (sum of PCDD/Fs): Maximum content in WHO-PCDD/F-TEQ 1**	**Sum of dioxins and dioxin-like PCBs (sum of PCDD/Fs and DL-PCBs): Maximum content in WHO-PCDD/F-PCB-TEQ 1**	**Non-dioxin-like PCBs: Maximum content 2**
1	**2**	**3**	**4**
relative to a feed with a moisture content of 12 %			
Feed materials of plant origin with the exception of:	0,75 ng/kg	1,25 ng/kg	10 µg/kg
- vegetable oils and their by-products	0,75 ng/kg	1,5 ng/kg	10 µg/kg
Feed materials of mineral origin	0,75 ng/kg	1,0 ng/kg	10 µg/kg
Feed materials of animal origin:			

Table 3. Maximum values for feed products (status as of 28th November 2019) [16, 32] (*continued*)

1	2	3	4
- Animal fat, including milk fat and egg fat	1,50 ng/kg	2,0 ng/kg	10 µg/kg
- Other land animal products including milk and milk products and eggs and egg products	0,75 ng/kg	1,25 ng/kg	10 µg/kg
- Fish oil	5,0 ng/kg	20,0 ng/kg	175 µg/kg
- Fish, other aquatic animals, and products derived thereof with the exception of fish oil, hydrolysed fish protein containing more than 20 % fat and crustacea meal	1,25 ng/kg 3	4,0 ng/kg 3	30 µg/kg 3
- Hydrolysed fish protein containing more than 20 % fat	1,75 ng/kg	9,0 ng/kg	50 µg/kg
- Crustacea meal	1,75 ng/kg	4,0 ng/kg	30 µg/kg
Feed additives belonging to the functional groups of binders and anti-caking	0,75 ng/kg	1,5 ng/kg	10 µg/kg
Feed additives belonging to the functional group of compounds of trace elements.	1,0 ng/kg	1,5 ng/kg	10 µg/kg
Premixtures	1,0 ng/kg	1,5 ng/kg	10 µg/kg
Compound feed with the exception of:	0,75 ng/kg	1,5 ng/kg	10 µg/kg
- compound feed for pet animals and fish	1,75 ng/kg	5,5 ng/kg	40 µg/kg
- compound feed for fur animals	---	---	---

4.Conclusions

Monitoring dioxin-like compound levels has become a very important tool at European level to ensure food security for citizens. Given the particularly serious effects on human health, reducing the chances of contamination of the food chain with such compounds must be a priority for food producers as well as for potentially polluting industries, so that such contamination is kept to a minimum.

Compliance with Ethics Requirements. Authors declare that they respect the journal's ethics requirements. Authors declare that they have no conflict of interest and all procedures involving human or animal subjects (if exist) respect the specific regulation and standards.

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