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DE SÉCURITÉ SANITAIRE
DES ALIMENTS

Maisons-Alfort, 13 March 2009

OPINION

THE DIRECTOR GENERAL

of the French Food Safety Agency
on potential human health risks related to the residual presence of perfluorooctanoic acid (PFOA) in non-stick coatings for cookware

Context of the request

On 13 December 2007, the French Food Safety Agency (AFSSA) was requested by the Caen branch of the consumer advocacy association *UFC Que Choisir* to issue an opinion on potential risks to human health due to residues of perfluorooctanoic acid (PFOA) in non-stick coatings for cookware.

Method of expertise

The collective expert assessment was conducted by the scientific panel on "Food Contact Mmaterials" based on past and current evaluations from national (COT), European (EFSA) and international (OECD) authorities:

- in 2005, the European Food Safety Authority (EFSA) classified PFOA in list 3 of the Scientific Committee on Food (SCF)[1] based on potential exposure data (expected uses of the substance, determination of residual content in food contact materials and migration calculation using a worst-case scenario), physico-chemical properties and toxicity data;

- in January 2005, the OECD published the results of a survey on production and use of PFOA, related substances and products/mixtures containing this substance (OECD, 2005);

- a risk assessment of PFOA and its salts is currently being undertaken by the U.S. Environmental Protection Agency (US EPA, 2005);

- in February 2006, Norway (CEE, 2006) submitted a proposal to classify PFOA and its salts and esters to the European Commission's Committee on Classification and Labelling. In October 2006, the T, R61-20/22-36-40-48/22-48/23[2] classification was adopted by this same committee;

1 no TDI could be established but use under specific conditions, i.e. in articles for repeated use, may be acceptable given its non-genotoxicity and the very low exposure levels.

2 T: toxic
R61: May cause harm to the unborn child
R20/22: Harmful by inhalation and if swallowed.
R36: Irritating to eyes
R40: Limited evidence of a carcinogenic effect.
R48/22: Harmful: danger of serious damage to health by prolonged exposure if swallowed.
R48/23: Toxic: danger of serious damage to health by prolonged exposure through inhalation.

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- in October 2006, the British Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT, 2006) published a Tolerable Daily Intake (TDI) of 3 µg/kg per day for PFOA and its salts based on toxic effects on the liver, kidneys, blood and immune system;

- a High Production Volume (HPV) file has been submitted to the OECD (OECD, 2006 and 2007; US EPA, 2006) and is in the process of being finalised;

- in February 2008, EFSA conducted a risk assessment of PFOA and its salts and established a Tolerable Daily Intake of 1.5 µg/kg per day based on hepatotoxic effects.

1. Physico-chemical properties and uses

1.1. General information

The IUPAC chemical name for PFOA is perfluorooctanoic acid. Its structure ($C_8HF_{15}O_2$, CAS No. 335-67-1) is shown in Figure 1 below. The most commonly used synonyms are: pentadecafluorooctanoic acid, pentadecafluoro-n-octanoic acid, perfluorocaprylic acid and PFOA (perfluorooctanoic acid). **APFO, which corresponds to the ammonium salt of PFOA (see Figure 2), is the most widely used form (CAS No. 3825-26-1). These two substances are metabolised equivalently (EFSA, 2008).**

Figure 1 (PFOA)

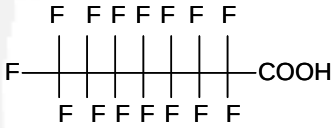
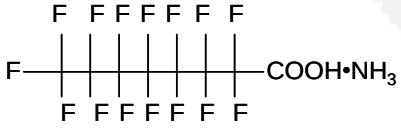


Figure 2 (APFO)



The octanol/water partition coefficient (log Kow) cannot be determined because PFOA, rather than being truly water soluble, forms microdispersion micelles (US EPA, 2005). The water solubility of PFOA is 9.5 g/l (OECD, 2006). It should also be noted that when mixed with water and hydrocarbons, PFOA forms three immiscible phases indicating that it is both hydrophobic and oleophobic (COT, 2006).

PFOA's primary physico-chemical characteristics are as follows (OECD, 2006):

Molecular weight: 414.09 $g.mol^{-1}$

Melting point (°C): 54.3°C

Boiling point (°C): 188°C (at 1013 hPa)

Density: 1.8 g/cm^3 at + 20°C

Vapour pressure: 4.2 Pa at 25°C

1.2. Types of food contact materials

APFO is primarily used as an emulsifier/dispersing agent during the polymerisation process of fluoropolymers [polytetrafluoroethylene (PTFE) polymer, and copolymers of tetrafluoroethylene with hexafluoropropene and/or perfluoropropylperfluorovinyl ether]. These fluoropolymers are used to produce articles for repeated use in contact with numerous types of food products (coatings for cooking utensils and particularly non-stick cookware, parts for food processing equipment, tubes, etc.). In these applications, the maximum addition level is 0.5% (EFSA, 2005).

Non-stick coating production involves high-temperature processes, typically between 350°C and 450 °C (Powley, 2005).

The main non-stick cookware with PTFE coating includes frying pans, saucepans and cake pans.

Contact foods are all types of foods that require cooking.

Contact conditions range from a few minutes (for searing meat) to 1 to 2 hours (for simmering).

Heating temperatures are no greater than 180°C (for stovetop cooking) to 250°C (for baking).

2.Hazard identification and characterisation of PFOA and its salts for human health

2.1. Absorption, Distribution, Metabolism and Excretion (ADME)

Available ADME studies indicate that PFOA is rapidly absorbed after oral administration. PFOA is not metabolized; the liver, blood and kidneys are the major sites of distribution in rats. Its half-life varies from a few hours to a few days in rats and monkeys to one year or more in humans. PFOA can cross the blood-placenta barrier and is mainly distributed in the foetal liver.

The pharmacokinetics of PFOA in non-human primates were analysed during a standard elimination study after (i) intravenous administration in 3 male and 3 female *Cynomolgus* monkeys and (ii) during a 6-month study in male *Cynomolgus* monkeys. These studies confirmed urinary elimination as the main mode of excretion. After intravenous administration of 10 mg/kg in males and females, the average half-life was 20.9 days for males and 32.6 days for females. In the 6-month study, the monkeys received 3, 10 and 30 mg/kg per day of APFO. A stable serum concentration was achieved after 4 to 6 weeks, with levels lower than expected according to the elimination rate, and were not proportional to the dose. Elimination half-lives were around 20 days in males.

In adult rats, PFOA is absorbed orally. Less absorption occurs with dermal exposure. The pharmacokinetic parameters in serum and distribution of PFOA have been examined in the tissues of adult rats further to gavage, intravenous (i.v.) and intraperitoneal (i.p.) administrations. PFOA is primarily distributed in the liver, serum and kidneys, and in other organs to a lesser extent. There is no preferential accumulation in the lipid fraction or fatty tissues (being both hydrophobic and lipophobic, PFOA probably binds with the proteins found in plasma). The distribution of PFOA is mainly extracellular. PFOA is not metabolised and it undergoes enterohepatic circulation.

Gender differences are observed in the elimination of PFOA in adult rats after gavage, i.v. and i.p. administration. Urine is the major route of PFOA excretion in female rats, while urine and faeces are the two routes of excretion in males. In female rats, after oral administration, estimated serum half-lives depend on the dose and range from 2.8 to 16 hours, whereas in male rats, estimated serum half-lives do not depend on the dose and range from 138 to 202 hours. The average half-life of PFOA in male rats is 70 times longer than in females (5.7 days versus 1.9 hours). In female rats, PFOA elimination appears to be biphasic (a rapid phase and a slow phase). The rapid excretion of PFOA in female rats is most likely due to active tubular secretion (organic anion carriers); this tubular renal secretion is probably controlled by hormones. Hormonal changes during gestation do not appear to change the rate of elimination in rats.

A few studies investigated the kinetics of PFOA during the development of Sprague-Dawley rats. PFOA easily crosses the blood-placenta barrier and can be found in the mother's milk. Differences in PFOA elimination by gender occur during development. At the age of 4-5 weeks, elimination mirrors that of adults and the gender-related difference becomes easily apparent.

Post-weaning distribution studies in rats have shown that PFOA is primarily distributed in serum, liver and kidneys.

In humans, a half-life study was carried out among 27 occupationally exposed persons. During this study, serum samples were collected every 6 months over a 5-year period. Two interim reports describing the results are available. The first interim report suggested a median serum half-life of 344 days, with extreme values of 109 to 1,308 days (the two most extreme values of 654 and 1,308 days were observed in two women).

2.2. Acute toxicity

PFOA is moderately toxic by ingestion and inhalation and is less harmful via skin contact.

In acute oral toxicity studies in rats, LD50 values are greater than 500 mg/kg for male Sprague-Dawley rats, range between 250 and 500 mg/kg for female Sprague-Dawley rats, and arelower than 1,000 mg/kg for male and female Wistar rats (Glaza 1997).
No deaths were observed after rats were exposed to 18.6 mg/l of APFO via inhalation for 1 hour.
In rabbits, the LD50 through dermal exposure is greater than 2,000 mg/kg (Glaza, 1995).

2.3. Sub-chronic and chronic toxicity

Available studies on toxicity through repeated administration show that the liver is the target organ of APFO toxicity in both rats and monkeys.

In rats and mice, repeated toxicity studies showed that the liver is the target organ. Because of elimination differences related to gender, effects appear at lower doses in male rats than in adult females.
Dietary administration of PFOA to rats for 90 days caused a significant increase of the liver weight at doses of 1,000 ppm (76.5 mg/kg per day) in females and at doses of 100 ppm (5 mg/kg per day) in males. Moreover, hepatic cell necrosis was observed in males at 30 ppm (1.7 mg/kg per day), and hypertrophy at 100 ppm (5.6 mg/kg per day). Based on hepatic effects, the No Observed Adverse Effect Level (NOAEL) was 0.6 mg/kg per day for males and 22 mg/kg per day for females (Goldenthal, 1978).

A dietary sub-chronic toxicity study (90 days) in male rats (daily doses equal to 0, 0.06, 0.64, 1.94 and 6.4 mg/kg per day) showed a reduced body weight gain at the highest dose. Doses of 0.64 mg/kg per day and higher caused a significant increase in relative liver weight and an increased hepatic palmitoyl-CoA oxidase activity (a marker of peroxisome proliferation). Histopathological changes included hypertrophy and hepato-cellular necrosis (Perkins *et al*., 2004).

During a 13-week study in rhesus monkeys, exposure to doses of 30 mg/kg per day and above resulted in animal deaths. Clinical signs of toxicity were noted starting at 3 mg/kg per day. During a 6-month study in *Cynomolgus* monkeys, increased liver weight was noted at 3 mg/kg per day, but there was no proof that PPARα activity increased. The Lowest Observed Adverse Effect Level (LOAEL) in this study was 3 mg/kg per day. The No Observed Adverse Effect Level (NOAEL) could not be determined (Butenhoff *et al*., 2002).

2.4. Effects on development and reproduction

APFO's toxicity on development has been investigated, for various routes of exposure, in rats, rabbits and mice.

In rats, no effects on development have been observed at the maximum dose of 150 mg/kg.

In rabbits, the only effect that APFO has induced on development is an increase in the frequency of skeleton variations in offspring. The NOAEL for development is 1.5 mg/kg.

In mice, the toxicity induced by exposure to APFO during gestation includes full litter resorption and dose-dependent neonatal mortality, reduced postnatal survival, delay in eye opening, growth deficits and alterations to gender-specific pubertal maturity. The NOAEL for development is less than 1 mg/kg. Exposure through lactation is not a major factor in the occurrence of these events. Early mortality induced by PFOA during gestation is independent of the induction of peroxisome proliferator-activated receptor alpha (PPARα). Post-natal mortality induced by PFOA is dependent on PPARα expression.
A two-generation reproductive study in rats at doses up to 30 mg/kg per day showed an effect on the body weight and sexual maturation of pups from the first generation (F1) and no effects on pups from the second generation (F2) until weaning.

2.4.1. Effects on development

In rabbits, the study on prenatal development toxicity showed a significant rise in skeleton variations after oral exposure to 5 mg/kg per day of APFO with an NOAEL of 1.5 mg/kg per day. No maternal toxicity was observed at the maximum tested dose of 50 mg/kg per day.

In rats, toxicity of PFOA on prenatal development has been investigated orally and by pulmonary administration. Orally, the LOAEL and NOAEL for maternal toxicity were respectively 150 and 5 mg/kg per day and no effects on development were observed at the maximum dose of 150 mg/kg per day (Staples and Burgess, 1984).
By inhalation, the LOAEL and NOAEL were respectively 10 and 1 mg/m^3 for maternal toxicity and 10 and 25 mg/m^3 for effects on development.

In mice, toxicity of PFOA on development following oral administration showed maternal toxicity: reduced weight gain (leading to a benchmark dose with a 5% response level, BMD5, of 6.76 mg/kg per day and a lower benchmark dose[3], BMDL5, of 6.76 mg/kg per day) and increased liver weight (with respective BMD5 and BMDL5 values of 0.20 and 0.17 mg/kg per day). Toxicity for development was also highlighted. The estimated BMD5 and BMDL5 values on the incidence of full litter resorption and neonatal mortality (determined by survival upon weaning) observed at the dose of 5 mg/kg per day were 2.84 and 1.09 mg/kg respectively. Significant alterations of postnatal growth and development were observed at 1 and 3 mg/kg per day, with respective estimated BMD5 and BMDL5 levels of 1.07 and 0.86 mg/kg, for neonate weight loss at weaning, and 2.64 and 2.10 mg/kg per day for delayed eye opening. The estimated BMD5 and BMDL5 levels for reduced phalanx ossification were less than 1 mg/kg. The estimated BMD5 and BMDL5 values for reduced foetal weight at term were estimated to be 10.3 and 4.3 mg/kg respectively.

The potential involvement of PPARα activation as mode of toxicity during development has been investigated in a dose-response study with wild-type (129S1/SvlmJ) and knockout (PPARα null) mice (Stanley, 2007; Andersen *et al*., 2008).

The results of this study show that PFOA: (1) does not affect weight gain in wild and mutant mice; (2) does not affect either the number of implanted embryos, or the number and weight of pups at birth, in wild and mutant mice; (3) increases full and early litter resorption in wild and mutant mice; (4) reduces neonatal survival, delays eye opening and decreases weight gain after birth in wild-type mice but not in PPARα null mice.

2.4.2. Effects on reproduction

Rats were exposed to 0, 1, 3, 10 and 30 mg/kg per day of PFOA during a two-generation reproductive study (Butenhoff *et al*. 2004). In this study, a decrease in the average body weight

3 Lower benchmark dose, corresponding to the lower limit of the BMD confidence interval

of first-generation (F1) pups was observed during lactation (results of both sexes combined) at 30 mg/kg per day. In F1 male pups in the treated groups at 10 and 30 mg/kg per day, weight gain significantly decreased over the period of 8-50 days after weaning, and weight gain significantly slowed down at 10 mg/kg per day starting on post-weaning day 36, and at 30 mg/kg per day starting on post-weaning day 8. Weight gain significantly decreased in F1 females in the treated group at 30 mg/kg per day during the first 15 days after weaning, and their body weight decreased after the 8th day. Fertility parameters did not change in F1 generation animals. A significant rise in mortality, primarily during the first days after weaning, and a significant delay in the sexual maturation of F1 males and females were noted at 30 mg/kg per day. No effects were observed in 2nd generation pups. However, it should be noted that the 2nd generation pups were killed at weaning, and therefore, potential post-weaning effects could not be observed.

2.5. Genotoxicity

Based on *in vitro* and *in vivo* studies, PFOA and its salts do not appear to be genotoxic.

APFO does not induce gene mutations in various strains of *Salmonella typhimurium,* Escherichia *coli,* in CHO K-1 cells and did not cause chromosomal aberrations in cultured human lymphocytes, in the presence and absence of metabolic activation (Lawlor, 1995 and 1996).
However, APFO induces chromosomal aberrations and polyploidy in cultured CHO cells. However, these results are unclear (confirmed **with** but **not without** metabolic activation) and are probably related to a cytotoxic effect. In addition, APFO is inactive in the mouse bone marrow micronucleus test (Murli, 1996) and in the cell transformation test on mouse embryonic fibroblasts in C3H 10T½ (Garry and Nelson, 1981).

2.6. Carcinogenicity

Carcinogenic potential of APFO was investigated in two chronic dietary studies in Sprague-Dawley rats. Under the test conditions, APFO appears to be carcinogenic, causing liver tumours, Leydig cell tumours (LCTs) and pancreatic acinar cell tumours (PACTs) in males. The increased frequency of mammary fibroadenomas in female rats is questionable because incidences were comparable to the controls. Extrapolation to humans remains debatable due to the mechanism of action.

In the first study, APFO was administered in the diets of Sprague-Dawley rats at concentrations of 0.30 ppm (average daily doses of 1.3 and 1.6 mg/kg per day in males and females respectively) and 300 ppm (14.2 and 16.1 mg/kg per day in males and females respectively) for 104 weeks. Non-neoplasic hepatic toxicity was dose-dependent and included megalocytosis, cystoid degeneration and portal mononuclear infiltration. In females, a non-significant increase in mammary fibroadenomas was noted. APFO also induced a dose-dependent but non-significant increase in Leydig cell adenomas (Sibinski, 1987)

In a second study, a high dietary dose of APFO (300 ppm for 24 months equivalent to 13.6 mg/kg per day) increased the incidence of hepatocellular adenomas, Leydig cell adenomas and pancreatic acinar cell hypoplasia in male rats (Biegel *et al*., 2001). The incidence of pancreatic tumours in rats was low and occurred only at high doses.

From these two carcinogenicity studies, EFSA therefore concluded that PFOA induces hepatocellular adenomas, Leydig cell adenomas and pancreatic acinar cell hyperplasia (EFSA, 2008).

For several years, the scientific community has agreed that the mechanism of action for liver tumours mediated by peroxysome proliferation is species specific and that this proliferation does not occur in humans.

Recent investigations have provided proof that substances responsible for peroxysome

proliferation act via PPARα receptor activation and that in humans, expression of the gene responsible for this activation is very low.

As a result, it is considered that the hepatic carcinogenic effects observed in rodents related to peroxysome proliferation cannot be extrapolated to humans due to significant inter-species differences that are both quantitative (PPARα's level of hepatic expression) and qualitative (panel of genes controlled by PPARα) (INRS, 2003; Lake, 1995; Lee *et al*., 1995; Green, 1995; Varanasi *et al*., 1996; Holden and Tugwood, 1999).

As is the case for liver tumours, Leydig cell tumours and pancreatic acinar cell tumours are secondary to hepatic PPARα activation; this would imply that like liver tumours, these tumours would most likely not be induced in humans.

However, as pointed out by EFSA, not all of the liver toxicity could be ascribed to PPRα activity. Consequently, the possibility that these tumours may occur in humans cannot be completely discounted (EFSA 2008).

2.7. Epidemiological studies

Epidemiological and medical surveys have been conducted among production workers occupationally exposed to PFOA in the United States. The majority of these studies have been surveys on representative samples and have mainly targeted men (Gilliland and Mandel, 1993; Alexander, 2001a and b). A retrospective mortality study demonstrated a statistically significant relationship between death from prostate cancer and the duration the person was employed at the PFOA production site (Gilliland and Mandel, 1993). However, when this study was updated and more specific exposure measurements were used over a longer time period, this significant relationship with prostate cancer was no longer observed. The other mortality studies did not contain adequate exposure data that could be linked to the occurrence of health effects. A study that examined the hormone levels of workers showed increased estradiol levels in workers with the highest PFOA serum concentrations; however, these results should have been indexed on body mass. Higher cholesterol and triglyceride levels in workers were also associated with PFOA exposure, which contrasts with the hypolipemic effects observed during experimental studies in rats. Furthermore, a statistically significant positive association was reported in workers between PFOA exposure and T3 (triiodothyronine) levels exclusively (Olsen and Zobel, 2007).

2.8. Toxicological Reference Value

Scientific publications on PFOA all indicate that hepatotoxicity in rodents may occur at lower doses than non-hepatic effects. The lowest NOAEL was 0.06 mg/kg per day for increased liver weight observed at 0.64 mg/kg per day in a sub-chronic toxicity study in rats. The BMDL10, defined as the lower limit of the 95% confidence interval of the dose for a 10% increase in liver weight versus control animals, was 0.4, 0.3 and 0.44 mg/kg per day in weeks 4, 7 and 13 respectively. A BMDL10 of 0.74 mg/kg per day was estimated for hepatocytic megalocytosis in the male rats in the two-year carcinogenicity study (Sibinski ,1987). A LOAEL of 1 mg/kg per day was identified for increased liver weight, and focal to multifocal hepatic necrosis in the P and F1 generational male rats in the two-generation reproductive study (Butenhoff *et al*., 2005), and the BMDL10 values were 0.31 mg/kg per day in both generations. Increased maternal liver weight was also reported in the reproductive mouse study, and the BMD10 and BMDL10 values were 0.52 and 0.46 mg/kg per day, respectively.

Evaluation by the "Committee on Toxicity of chemicals in food, consumer products and the environment" (COT, 2006)

Based on these studies, the BMDL10 of 0.3 mg/kg per day was selected by the COT (2006) to establish a Tolerable Daily Intake (TDI). An uncertainty factor of 100 was then applied to take into account inter- and intra-species variations. As a result, the TDI recommended by the COT for PFOA is 3 µg/kg b.w.

Evaluation by the European Food Safety Authority (EFSA, 2008)

EFSA, which based its evaluation on the same data as the COT, also concluded that the BMDL10 of 0.3 mg/kg b.w. was a solid starting point for the establishment of a TDI. This value of 0.3 was divided by a safety factor of 200 (10 for inter-species differences; 10 for intra-species differences and 2 to compensate for uncertainties concerning elimination rates) leading to a **TDI of 1.5 µg/kg b.w.**

3. Exposure data:

3.1. Exposure via the environment

Perfluorinated derivatives are used for multiple purposes. PFOA/APFO is primarily used in the production of fluoropolymers and fluoroelastomers. Entry into the environment occurs during production and use of PFOA/APFO. Other sources of release into the environment are related to PFOA residues contained in fluoropolymer and fluoroelastomer products, products and fire-retardant foams containing perfluorocarboxylates, perfluorooctylsulfonyl (PFOS) based products and fluorotelomer based products. An indirect source of PFOA in the environment is the degradation (biotic and abiotic) of some fluorotelomer-based products.

High PFOA levels have been observed near industrialised and urbanised areas. PFOA concentrations up to 67 µg/l and 3,200 µg/l have been reported in sewage effluent and landfill effluent. In natural water samples (rivers, lakes, rain water), the maximum analysed concentration of PFOA was 11.3 µg/l. High PFOA concentrations have also been found in coastal waters near industrialised and urbanised sites, with a maximum concentration of 15.3 µg/l.

3.2. Exposure via foodstuffs

Based on low levels of PFOA essentially in fish and drinking water and on consumption data from four European countries (Italy, Netherlands, Sweden and England), EFSA (2008) estimated that average dietary exposure to PFOA was approximately 2 ng/kg b.w. per day and that maximum exposure was equal to 6 ng/kg b.w. per day.

3.3. Exposure via food contact materials

The amount of perfluorinated derivatives used to produce materials and packaging intended to come in contact with food are relatively low.

The ammonium salt of perfluorooctanoic acid (APFO) is used as a polymerisation aid in the production of fluoropolymer-based materials (PTFE), and particularly non-stick coating for cookware.

Few data are available in the literature about migration of perfluorinated compounds from contact materials to food, mainly because of analytical difficulties (EFSA, 2008).

In EFSA's 2005 opinion, maximum possible migration was estimated based on the determination of residual PFOA and APFO in a homogeneous fluoropolymeric sample containing the substance and obtained by extrusion and sintering at high temperature (to obtain an agglomerate of the treated material to give it sufficient cohesion and rigidity). Although the analytical method was not clearly described, residual PFOA and APFO were never detected in the sample. However, in order to be as safe as possible, a worst-case migration was estimated as **17 µg/kg food**, based on the method's detection limit of 0.022 mg/kg polymer and the hypothesis of a 0.6 cm thick coating with a density of 2.1 g/cm^3 (EFSA, 2005).

The studies of Begley *et al*. (2005) defined a maximum migration of **0.09 µg/kg food** determined based on residual PFOA levels ranging from 4 to 75 µg/kg coating in the hypothesis of a 100% transfer. To that end, the following postulates were considered: a 28 cm diameter frying pan, with a uniform 75 µm coating having an assumed density of 2.2g/cm^3, in contact with 1,200 g food.

However, it was shown that when PTFE film was heated at 175°C cooking temperatures for 2 hours, only 17% of the total PFOA contained in the film migrated to the simulant. Migration under the abovementioned conditions was therefore **0.15 µg/kg food**. The work of E. Sinclair (ES&T 2007) came to the same conclusion.

To complete these results and identify whether inappropriate use through overheating could lead to additional migration, frying pans were subject to a temperature gradient with a 1.5 minute period at a temperature that increased from 270°C to 320°C. PFOA was not detected in the "overheated" coatings (Begley *et al*., 2005). These results were confirmed by Charles *et al*. (2005). The absence of PFOA in coating following treatment at these high temperatures may suggest the total migration of PFOA in food after volatilisation from the coating , corresponding to a maximum transferable amount of 0.9 µg PFOA to 1 kg food.

To conclude, two maximum theoretical migration values are available: 17 µg/kg (EFSA, 2005) and 0.09 µg/kg (Begley *et al*., 2005).
Under realistic conditions, the migration was 0.15 µg/kg food (Begley *et al*., 2005).

In its 2008 opinion, EFSA considered that data are insufficient to establish the contribution of food contact materials to PFOA total dietary exposure (EFSA, 2008).

However, considering that every day, a 60 kg adult eats one kilogram of food cooked with cookware covered with PTFE coating and based on a maximum theoretical migration of 17 µg/kg food (EFSA, 2005), exposure via contact materials would be 0.3 µg/kg b.w. per day.
A more realistic estimate, based on an observed migration of 0.15 µg/kg food (Begley *et al*., 2005), would give an exposure via contact materials of 0.0025 µg/kg b.w. per day.

These exposure levels via food contact materials are much lower than the TDI of 1.5 µg/kg b.w. per day established by EFSA.

These estimates are based on worst case scenarios since they consider that a daily portion of 1 kg of food in contact with cookware (which is an extremely high assumption for cooking). Moreover, these scenarios do not consider the fact that the amount of PFOA decreases over time as the cookware is used.

4. Conclusions:

PFOA is a perfluoroalkylated compound that belongs to the class of persistent organic pollutants.

Considering (1) that PFOA and its salts are not genotoxic, (2) that the carcinogenesis mechanism in rodents cannot be extrapolated to humans, (3) that the TDI established by EFSA (2008) for PFOA is 1.5 ug/kg per day, (4) that realistic exposure to PFOA via food contact materials would be 0.0025 µg/kg per day and maximum theoretical exposure would be 0.3 µg/kg b.w. per day and (5) that EFSA estimated maximum dietary exposure to be 6 ng/kg b.w. per day (EFSA, 2008), the consumer health risk related to residues of PFOA in non-stick coating for cookware is considered to be negligible.

Keywords

PFOA, non-stick coating, food contact materials, perfluorinated compounds

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