Federal Institute for Risk Assessment

Per- and Polyfluorinated Alkyl Substances (PFAS): Status Quo of consumer health assessment on PFAS

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Welcome

Welcome to the BfR-Symposium on Per- and Polyfluorinated Alkyl Substances (PFAS): Status Quo of Consumer Health Assessment on PFAS.

Per- and polyfluorinated alkyl substances (PFAS) are industrial chemicals with unique technical properties. They are employed in numerous industrial and consumer products. Because of their widespread use, they have become ubiquitous in the environment.

In its Opinion on PFAS in foodstuffs from 2008, the BfR stressed the multitude of uncertainties regarding toxicological effects and exposure from food and other consumer products. Since then, the BfR has devoted several research projects to answering questions on carry-over, toxicokinetics, toxicology and migration of PFAS. The results of these investigations will be presented in the symposium and be complemented by the current findings from other experts.

The purpose of this symposium is to formulate the status quo in such a way that it becomes applicable to future risk assessments. Furthermore, we will identify open questions and new research objectives.

On the behalf of the Federal Institute of Risk Assessment we wish you all a scientifically and socially rewarding experience, as well as a very enjoyable stay.

Table of Contents

Scientific Program

Thursday, 6 March 2014

10:30–10:45 a.m.
Welcome and Opening Remarks
Professor Dr. Dr. Andreas Hensel,
Federal Institute for Risk Assessment (BfR)

10:45–11:15 a.m.
PFAS, variety, applications and use
Dorte Herzke, Norwegian Institute for Air Research

I. Toxicology
Chair: Dr. Ulrike Bernauer, BfR

11:15–11:45 a.m.
Toxicological background
PD Dr. Detlef Wölfle, BfR

11:45–12:15 a.m.
Molecular effects of PFOA on the transcriptome of human primary hepatocytes
Dr. Thorsten Buhrke, BfR

12:15–12:45 a.m.
Status quo of epidemiological research on perfluorinated compounds (PFC)
Dr. Michael Schümann, Department of Health and Consumer Protection, City of Hamburg

12:45–2:00 p.m. Lunch

2:00–2:30 p.m.
Regulatory assessment under REACH
PD Dr. Thomas Schulz, BfR

2:30–3:00 p.m. **Discussion**

II. Toxicokinetics and Carry over
Chair: Professor Dr. Hans Schenkel, Landesanstalt für Landwirtschaftliche Chemie

3:00–3:30 p.m.
Carryover from soil to plants in a long-term lysimeter experiment and a nutrition solution experiment
Dr. Thorsten Stahl, Hessian State Laboratory

3:30–4:00 p.m.
Occurence of PFAS in food of plant origin
Dr. Josef Müller, Fraunhofer IME

4:00–4:30 p.m. Coffee Break

4:30–5:00 p.m.
Metabolism and carry over of PFAS in ruminants
Janine Kowalczyk, BfR

5:00–5:30 p.m.
Modelling of toxicokinetics in pigs and laying hens
Dr. Jorge Numata, BfR

5:30–6:00 p.m. **Discussion**

End of the first day

Friday, 7 March 2014

09:00–09:10 a.m.
Welcome and summary of the first day
Dr. Ulrike Pabel, BfR

III. Exposure of consumer
Chair: Professor Dr. Peter Fürst, Chemical and Veterinary Analytical Institute Muensterland-Emscher-Lippe

09:10–09:40 a.m.
PFAS in the environment
Dr. Annegret Biegel-Engler,
Federal Environment Agency

09:40–10:10 a.m.
Human biomonitoring of PFCs – internal levels and total exposure
Prof. Dr. Hermann Fromme, Bavarian Health and Food Safety Authority

10:10–10:40 a.m.
Migration of poly- and perfluorinated compounds from food contact materials into food
Dr. Jutta Tentschert, BfR

10:40–11:10 a.m.
Dietary exposure to selected perfluoroalkyl acids (PFAAs) in four European regions
Stefanie Klenow,
PD Dr. Gerhard Heinemeyer, BfR

11:10–11:45 a.m. Coffee Break

11:45–12:15 a.m.
Human exposure to perfluoroalkyl substances
Robin Vestergren, Norwegian Institute for Air Research

12:15–12:45 a.m. **Discussion**

IV. Discussion and conclusion
Chair: PD Dr. Helmut Schafft, BfR

12:45–1:45 p.m.
Plenary discussion

1:45–2:00 p.m.
Closing Remarks
Professor Dr. Dr. Andreas Hensel, BfR

Abstracts of oral presentation

Session I - Toxicology

PFAS, variety, applications and use

(PFAS, deren Vielfalt, Vorkommen und Verwendung)

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[2.]SWEREA AB; Stockholm, Sweden

Perfluoroalkyl and polyfluoroalkyl substances (PFAS) are used in numerous industrial and consumer products because of their special chemical properties, for instance the ability to repel both water and oil. A broad variety of PFAS have been introduced into the Norwegian market through industrial use (e.g. via fire fighting foams and paints) as well as in treated customer products such as textiles and coated paper. Our present knowledge of the exact chemical PFAS compositions in preparations using perfluorinated compounds is limited. This lack of knowledge means that it is difficult to provide an accurate assessment of human exposure to these compounds or to the amount of waste that may contain treated products. It is a growing concern that these potentially harmful compounds can now be found throughout the global environment.

Recently a number of studies investigated samples of industrial and consumer products in Norway, Germany and other countries. Only few of the analysed industrial and consumer products contained none of the polyfluorinated substances that were analysed but this does not exclude the occurrence of unknown PFAS. Notable was that perfluorooctanesulfonate (PFOS), which has been strictly regulated in Norway since 2007, was found in amounts close to or exceeding the EU regulatory level in a number of products, mostly within the leather or carpet product groups. High amounts of fluorotelomer alcohols (FTOHs) were found in waterproofing agents, carpets and textiles. The almost ubiquitous presence of PFAS in a broad range of consumer products can give rise to a constant diffuse human exposure that might eventually result in harm to humans.

Toxicological Background

(Toxikologische Grundlagen)

Detlef Wölfle

Department Safety of Consumer Products, Federal Institute for Risk Assessment

PFAS are widely used in many industrial and consumer products and are found ubiquitously in the environment, food and tissues of wildlife and humans. Due to the persistence of some PFAS the substances have raised safety concern and therefore intense research is on-going in humans, experimental animals and cell cultures. PFAS, particularly perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts, were evaluated by several national and international agencies, e.g. EFSA (2008). Liver was identified as the main target organ (hypertrophy, biochemical effects, adenomas) and developmental and reproductive effects were also observed in experimental animals at relatively low doses of both PFOS and PFOA. Due to the absence of a genotoxic potential tumor induction observed in rats treated with PFOS (liver, thyroid, mammary gland (?)) or PFOA (liver, acinar pancreas, testicular Leydic cells) is considered to be mediated via non-genotoxic mechanisms. In addition, changes in thyroid hormones and high densitiy lipoprotein levels were observed in Cynomolgus monkeys. Based on a NOAEL (0.03 mg/kg bw/day) from a subchronic monkey study EFSA has established a tolerable daily intake (TDI) for PFOS of 150 ng/kg bw (EFSA, 2008). For PFOA the lowest BMDL10 value (0.3 mg/kg bw/day) derived for liver effects in mouse and rat studies was used as a point of departure for setting a TDI of 1.5 µg/kg bw (EFSA, 2008). Recent studies indicate that the mammary gland (growth and differentiation) is a sensitive target tissue of prenatal exposure to PFOA at low doses in mice (0.01 mg/kg bw on gestation days 10-17). Low dose effects of PFOA were also reported to result in behavioural effects (prenatal exposure to 0.3 mg/kg bw/day) and in immune effects (changes in mature splenic lymphocyte populations at 0.49 mg/kg bw/day) in mice.

Pharmacokinetic data on both substances PFOS and PFOA indicate slow elimination with long serum half-lives in humans (of approx. 5.4 and 3.8 years, respectively). Much shorter serum half-lives were reported for PFOS in male and female monkeys (131 and 110 days) and rats (43 and 60 days) and for PFOA in male and female monkeys (33 and 21 days) and rats (6-8 days and 3-16 hours respectively). Similarly for perfluorohexanesulfonate (PFHxS) a long serum elimination half-life of 7.3 years was estimated in humans compared to shorter half-lives in male and female monkeys (141 and 87 days) and rats (30 and 1.5 days). PFAS with shorter chain length, e.g. perfluorobutane sulfonate (PFBS), are reported to be eliminated faster in rats.

While PFOS and PFHxS reduce cholesterol and triglyceride levels in mice, PFBS does not induce these effects. Similarly perfluorohexanoic acid (PFHxA) has a shorter serum half-life in rats (3-6 hours) compared to PFOA and induces liver effects (increase in weight, peroxisome proliferation) at doses which are 2 orders of magnitude higher than those for PFOA. While the shorter chain PFAS have a lower toxicity, longer chain PFAS, e.g. perfluorononanoic acid (PFNA) are persistent contaminants with a comparable developmental toxicity in rats to that of PFOA. Interestingly, PFOA can also be formed in the mammalian metabolism (rat) from precursors such as polyfluoroalkyl phosphate surfactants (PAPS) and the fluorotelomer alcohol 8:2 FTOH. Similarly 6:2 FTOH can be metabolised in vitro (rat, mouse, human hepatocytes) to PFHxA and perfluoropentanoic acid.

Liver toxicity and developmental effects of PFAS are known to be associated with the activation of the peroxisome proliferator-activated receptor alpha (PPARα). However, species differences in the PPARα suggest that humans are less responsive to the hepatotoxic effects of

PFAS than rodents. The activation of the PPARα has been shown to be dependent on the chain length of PFAS with the highest activity around C8, but transcriptional changes may involve other nuclear receptors (e.g. the constitutive activated receptor, CAR). In addition recent studies indicate that some PFAS induce oestrogen receptor transactivity and antagonise the androgen receptor activity in vitro.

Molecular effects of PFOA on the transcriptome of human primary hepatocytes

(Molekulare Wirkmechanismen)

Thorsten Buhrke, Eileen Scharmach, Sophie Pevny, Manuela Rößler, Katja Bitter, Alfonso Lampen

Department Food Safety, Federal Institute for Risk Assessment

The toxicological data of perfluorooctanoic acid (PFOA) give cause for concern as the substance was shown to damage the liver of rodents and to impair embryo development. On the molecular level, the hepatotoxic effects were attributed to the PFOA-mediated activation of peroxisome proliferator-activated receptor alpha (PPARα), a ligand-dependent transcription factor that is involved e. g. in the regulation of lipid metabolism in the liver. PPARα-dependent effects are less pronounced in humans than in rodents due to its lower expression level in the human liver. Therefore, the hazard potential of PFOA for humans is controversially discussed.

To analyse the effects of PFOA in human hepatocytes, a microarray analysis was conducted to screen for PFOA-mediated alterations in the transcriptome of human primary hepatocytes by using the GeneChip HG-U133 plus 2.0 (Affymetrix). A subsequent network analysis revealed that PFOA had an impact on several signalling pathways in addition to the well-known activation of PPARα. PFOA was shown (i) to induce activation of the AP-1 transcription factor via upregulation of the proto-oncogenes c-Jun and c-Fos (ii) to downregulate the estrogen receptor ERα (iii) to activate the pregnane X receptor (PXR) and the constitutive androstan receptor (CAR), and (iv) to inhibit the function of the hepatocyte nuclear factor 4α (HNF4α) which is an essential factor for liver development and embryogenesis. The hypotheses derived from the microarray data were confirmed by using the HepG2 cell line as a model for human proliferating hepatocytes. With respect to the observed activation of the AP-1 transcription factor it was shown that PFOA stimulates cellular proliferation via upregulation of the expression of various cyclines which have a central function in the regulation of cell cycle control. Moreover, PFOA-mediated inhibition of HNF4α activity was confirmed by various molecular assays. In sum, the results of the study indicate that PFOA affects additional, PPARα-independent pathways in human hepatocytes. The observed deregulation of these pathways may be relevant for human health. Thus, the elucidation of novel modes of action of PFOA contributes to the ongoing risk assessment of the substance.

Status quo of epidemiological research on perfluorinated compounds (PFC)

(Stand der epidemiologischen Forschungen)

Michael Schümann

Department of Health and Consumer Protection, City of Hamburg[1]

The production of PFCs has become large-scale since the 1950s for a variety of applications. Perfluorooctane acid (PFOA) and Perfluorooctanesulfonate (PFOS) are noteworthy for their production and use amounts, particularly as materials in the production of chemical end products (especially PFOA). On the other hand, they are also a component in many finished products, textiles, and household chemicals (especially PFOS). The environment and humans are exposed directly and indirectly to these anthropogenic perfluorinated compounds. Unlike other organic substances, PFOA is readily soluble in water and thus contaminate drinking water to a relevant degree. Since the 1970s, perfluorinated chemicals have been detected worldwide in various environmental media and human tissues.

The health assessment of PFOA and PFOS was still largely based on animal experiments and partly on occupational health data (EFSA 2008, BfR 2008, ATSDR 2009). The current TDI values are 1.5 [g / kg bw / d] for PFOA and 150 [ng / kg b.w / d] for PFOS. In animal experiments, PFOA and PFOS are moderately acutely toxic; PFOA is toxic to the liver and shows developmental, immune and reproductive toxic effects at low dose levels. The incidence of tumors (especially in liver) increases in a dose-dependent way (rat). For PFOS, changes in the thyroid and fat metabolism (rats and monkeys) have been reported to be dose-dependent. But, since the elimination rates of PFC in humans are much lower than in animals (Brede 2010), an evaluation of dose-response-relationship based solely on animal experiments is restricted.

Since 2004, the number of epidemiological studies performed has suddenly increased, particularly in connection with the announcement of regional PFC pollution of drinking water in the U.S. (about 69 000 participants) and in Germany. In addition to cross-sectional studies, also population-based HBM studies (e.g. Fromme et al. 2007) and additional laboratory analyzes and evaluations of national survey data (e.g. NHANES / USA) and sub-studies within the framework of ongoing cohort studies (especially from Scandinavia) were performed. Statistically significant associations between PFOA and PFOS concentrations in blood serum and the examined health endpoints have been reported in epidemiological studies in recent years. The influence of PFOA and PFOS are difficult to separate, since the PFOA / PFOS plasma concentrations are usually correlated ($r \sim 0.7$) under common environmental conditions. An exception is the American C8 study (Frisbee et al. 2010), in which a long-term intake of PFOA through contaminated drinking water was the main source of exposure.

Significant associations of different health outcomes to the level of existing burden levels in the population and for PFOA in highly exposed cohorts (drinking water) have been reported, mostly controlling for other influencing factors such as age, BMI, behavioral patterns, smoking, etc. Such associations have been shown for: fertility and time to desired pregnancy (Fei et al. 2009); sperm quality (Joensen et al. 2009); partly for birth weight and birth size (Fei et al. 2007/2008 , Hamm et al. 2010 , Andersen et al. 2010); for the period up to weaning (Fei et al. 2010). A tendency to low birth weights (Chen et al. 2012) is also seen for PFOS burdens.

No evidence of a tendency for premature births was seen in the C8 study; However, Darrow et al. (2013) point to a significant association of PFOA and PFOS body burdens to the occur-

[1] The views and scientific assessments presented in this abstract and talk are those of the speaker and they need not reflect those of the BGV/Hamburg.

rence of pregnancy-induced hypertensive disorder. The available data is nevertheless inconsistent. For example, no corresponding association with birth weights has been shown for the Norwegian population (Whitworth et al. 2012), for British girls at birth, and not in the postnatal development (Maisonet et al. 2012). On the basis of the data of the Faroe 1997-2000 birth cohorts, Grandjean et al. 2012 show that a higher PFC body burden is associated with reduced immune response (determination of antibody titers after vaccination) at 7 years of age (Grandjean and Budtz-Jørgensen 2013). The motor and cognitive development of children and adolescents seem to be uninfluenced. The data for the incidence of hyperactivity and attention deficit are inconsistent. The relationship between high PFOA/PFOS burden and delays in age at puberty (questionnaire and hormonal status analysis) for girls and boys (Steenland et al. 2010, Lopez-Espinosa et al. 2011, Christensen et al. 2011) is reported as a critical effect seen in a population study as well as in the C8 study. Two animal studies are demonstrating effects of PFOA on the development of the female mouse mammary gland in comparable dose ranges. The results of Kristensen et al. (2013) for the Danish birth cohort (1988-1989) point to the same direction.

A relatively consistent link between PFOA/PFOS serum concentration and changes in lipid metabolism has been shown for all age groups and in both genders. The PFC burden is associated with higher total cholesterol, especially the LDL fraction (Steenland et al. 2009, Frisbee et al. 2010). Relatively consistent associations are reported for an uric acid increase with exposure (Steenland et al. 2010). The available data on the possible effects on thyroid metabolism in adulthood indicate to a higher thyroid disease incidence, for men at PFOS concentrations higher than the reference values and in women for higher PFOA concentrations (Melzer et al. 2010). Winquist and Steenland (2014) show corresponding consistent associations in the C8 study, while Wang et al. (2013) have reported corresponding trends for PFOS in a Norwegian birth cohort (2003-2004). Lopez-Espinosa et al. (2012) show associations with thyroid metabolism of PFOA and PFNA for exposed children. Knox et al. (2011) show an association of the a younger age at menopause with higher PFOA/PFOS plasma concentrations; in particular for PFOS as an association to the measured perimenopausal and postpausal estradiol serum concentrations.

Steenland et al. (2013) show a highly significant association between the incidence of ulcerative colitis and PFOA exposure history, without association to Crohn's disease, rheumatoid arthritis, type I diabetes and multiple sclerosis. New insights on the incidence of cancer arise from the C8 study: Barry et al. (2013) point to a consistently rising risk of testicular and kidney cancer for higher cumulative PFOA exposure. The results confirm the evidence of malignant and nonmalignant kidney diseases seen in highly exposed workers (Steenland and Woskie 2012). Hardell et al. (2014) indicate to significant interaction between individual risk factors and PFC loads as influence factors for the prostate cancer incidence risk.

The results from epidemiological studies indicate that effects occur in some cases even at normal levels of burden seen actually in the general population. A critical overview of existing results from epidemiological studies can be found for example in Olsen et al. (2009) for prenatal development, in Steenland et al. (2010) for findings from the C8-study and in Post et al. (2012) for PFOA from water contamination. On the basis of the epidemiological literature no indication for the existence of a threshold dose is seen. For several health endpoints a steeper increase of the dose-effect relationship in the low dose range is documented. The state of knowledge gained by the epidemiology of PFOA and PFOS over the last years has to be considered. Several of the observed health effects associated to PFC exposure are classified as adverse. This new state of knowledge must be compiled and evaluated within an appropriate updated risk assessment that might support prevention.

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Regulatory assessment under REACH

(Regulatorische Bewertung unter REACH)

Thomas Schulz

Department Toxicology of Chemicals, Federal Institute for Risk Assessment

Perfluorooctanoic acid (PFOA) is a very persistent chemical and has been detected in the environment, in organisms and human blood. The long half-lives of elimination from blood gave rise to concern.

The German Competent Authority (CA) prepared in 2009 in co-operation with industrial partners a Chemical Safety Report (CSR) in accordance with the REACH provisions. The CSR was submitted to the European Commission.

In March 2010 Norway submitted an Annex VI-Dossier to ECHA proposing a classification according to CLP Carc. 2, H351, Repr. 1B, H360D, STOT RE1, H372, Acute Tox 4, H332, Acute Tox. 3, H301, Eye Irrit. 2, H319. After discussion in the Risk Assessment Committee (RAC) the proposal was slightly modified and the classification was decided as follows: Carc. 2, H351, Repr. 1B, H360D, Lact., H362, STOT RE1, H372 (liver), Acute Tox 4, H332, Acute Tox. 4, H302, Eye Dam. 1, H318. This classification was published in the 5th adaptation to technical progress to the CLP regulation on October, 3rd, 2013 and enters into force on January 1st, 2015.

Based on the classification Repr. 1B PFOA has been considered as a substance of very high concern according to Art. 57c. Additionally, PBT (persistence, bioaccumulation and toxicity) properties were also evaluated (accord to Art. 57d) and a dossier in accordance with Annex XV was submitted by the German and Norwegian CA in 2012. In June 2013 PFOA was included into the Candidate List. Now consumers have the chance to get information about the presence of PFOA in products. However, authorization itself seems not to be the appropriate risk management option because it is expected that most of the PFOA enters the EU in imported products.

Finally, new developments in the regulation of PFOA will be discussed.

Session II - Toxicokinetics and Carry over

Carryover from Soil to Plants in a Long-Term Lysimeter Experiment and a Nutrient Solution Experiment

(Aufnahme aus dem Boden in die Pflanze – Lysimeter- und Gefäßversuch)

Thorsten Stahl

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a) A long-term lysimeter experiment to investigate the leaching of perfluoroalkyl substances (PFASs) and the carryover from soil to plants – Results of a pilot study[1]

To study the behavior of PFASs in soil and the carryover from soil to plants, technical mixtures of PFOA and PFOS at a concentration of 25 mg/kg soil were applied to 1.5 m^3 monolithic soil columns of a lysimeter. Growth samples and percolated water were analyzed for PFASs throughout a period of 5 years. In addition to PFOA/PFOS plant compartments and leachate were found to be contaminated with short-chain PFASs (PFBA, PFBS, PFPeA, PFHxA, PFHxS and PFHpA). Calculation showed significant decreasing trends ($p < 0.05$) for all substances tested in the growth samples. In order to detect possible monotonic decreasing trends for all PFOA and PFOS data from the years 2007 to 2011 separately for grain and straw, the rank correlation coefficient according to Spearman was calculated and tested for deviation from zero. Statistically significant decreasing trends were found for PFOA both in straw and grain as well as for PFOS in straw, whereas a significant decreasing trend for PFOS in grain was not detected. This is the result of the fact that in the years 2008, 2009 and 2010 no PFOS concentrations above the limit of quantification (1.0 µg/kg) were found, however a value above the LOQ was seen in 2011. This may be due to the type of plant (barley), which possibly takes up PFOS preferentially.

Short-chain PFASs and PFOA pass through the soil much more quickly than PFOS. Of the 360 g of PFOA and 367.5 g of PFOS applied to the soil 96.88% PFOA and 99.98% PFOS were still present in the soil plot of the lysimeter after a period of five years. Plants accumulated 0.001% PFOA and 0.004% PFOS. Loss from the soil plot through leachate amounted to 3.12% for PFOA and 0.013% for PFOS.

b) Effects of chain length and pH on the uptake and distribution of perfluoroalkyl substances in maize (Zea mays)[2]

Maize is the most important grain crop grown for human nutrition, animal fodder and biogas production worldwide. Nonetheless, no systematic studies have been undertaken on these plants to examine the uptake mechanisms for perfluoroalkyl substances (PFASs) dependent upon chain length and pH value. The aim of the present study was therefore to determine the influence of chain length (C4 to C10) and pH value (pH 5, pH 6, pH 7) on the uptake and distribution of seven perfluoroalkyl carboxylic acids (PFCAs) and three perfluoroalkane sulfonic acids (PFSAs) by maize in nutrient solution experiments under controlled conditions in a climate chamber. A pH-dependent uptake was observed for perfluorodecanoic acid (PFDA) with an uptake rate of 2.51 µg g^{-1} at pH 5 compared to 1.52 µg g^{-1} root dry weight (DW) per day (d) at pH 7. Perfluorobutanoic acid (PFBA) had the highest uptake rate within the group of PFCAs with an average of 2.46 µg g^{-1} root DW d^{-1} and perfluorooctane sulfonic acid (PFOS) had the highest uptake rate (3.63 µg g^{-1} root DW d^{-1}) within the group of PFSAs. The shoot:root ratio for shorter-chain PFCAs (≤ C7) and PFBS (C4) was > 2.0, which indicates that shorter-chain PFASs are transferred predominantly and at higher concentrations to the shoot. In contrast, long-chain PFCAs such as perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA) and perfluorodecanoic acid (PFDA) as well as the PFASs perfluorohexane sulfonic acid (PFHxS) and perfluorooctane sulfonic acid (PFOS) accumulated at higher concentrations in the roots of maize plants with a shoot:root ratio of < 1.0.

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Occurrence of PFAS in food of plant origin

(Aufnahme von PFAS in Pflanzen unter Freilandbedingungen)

Dr. Josef Müller, Fraunhofer IME, Schmallenberg

The investigation was performend within the framework of the EU FP7 project Perfood. The uptake of Perfluoroalkyl acids (PFAAs) from fortified soil into crops under field conditions was studied. The work complements a comparable study performed under controlled conditions (greenhouse hydroponic systems) at the University of Amsterdam.

Corn, peas, radish and lettuce were planted on soils fortified to 4 PFAS levels (0.1 mg/kg, 1 mg/kg, 5 mg/kg and 10 mg/kg + unfortified soil). The tests were performed using soil lysimeters which allowed the collection of drainage water.

After harvest the plant organs were analyzed for their contents of 11 perfluorocarboxylic acids (C_4-C_{14}) und 2 perfluorosulfonic acids (PFBS and PFOS). In addition the PFAS were determined in the matrices soil, soil pore water and lysimeter drainage water.
An overview is given over the performance of the study including site preparation, plant growing, analyses and results obtained.

Metabolism and carry over of PFAAs in ruminants

(Metabolismus und Carry over bei Wiederkäuern)

Janine Kowalczyk, Helmut Schafft

Department Safety of Food Chain, Federal Institute for Risk Assessment

1. Carry over of PFAAs from naturally contaminated feed into tissues and milk of dairy cows

For feeding study, six lactating cows (Holstein Friesian) were fed with PFAA-contaminated feed (grass silage and hay), which grew on a PFAA-contaminated farmland in Lower Saxony, Germany. After 28 days, three cows were slaughtered whereas the others were fed with non-contaminated feed for another 21 days before they were also slaughtered. For PFAA analysis, samples were taken from blood plasma, milk, liver, kidney and muscle tissue (*M. longissimus dorsi*). All samples were analysed by the Chemical and Veterinary Analytical Institute Münsterland-Emscher-Lippe using HPLC-MS/MS (Ehlers 2012).

In plasma and milk, highest concentration were found for PFHxS and PFOS during exposure period. PFBS and PFCAs (PFHxA, PFHpA and PFOA) concentration were constantly low during PFAA feeding and decreased under detection limit when PFAA feeding has stopped. In contrast, PFHxS and PFOS did not reached detection limit until 21 days of depuration period. In milk, half-life of PFOS was estimated to be 56 days (van Asselt et al. 2013). Liver was the target organ for PFAA accumulation. Corresponding to tissue mass, estimated fraction of ingested PFAAs was highest in muscle tissue followed by liver and kidney. Overall, estimation of the PFAA distribution volume indicated that the longer chain length, the higher the accumulation in tissue and milk of dairy cows (Kowalczyk et al. 2013).

2. Study on PFAA degradation in rumen of cows using RUSITEC

Since carry over of PFAAs in tissue of ruminants has been shown to differ from that of monogastrics, hypothesis derived that ruminal fermentation affected toxikokinetics of PFAAs. The effect of microbial fermentation on PFAAs in rumen was evaluated using the *in vitro* Rumen Simulation Technique (RUSITEC), which was performed at the University of Veterinary Medicine Hannover. In experiment A, bags filled with PFAA contaminated hay were tested in six fermentation vessels, inoculated with ruminal microorganisms. In experiment B, same feed was used, but fermentation vessels were conducted under sterile conditions to determine the amount of PFAAs possibly metabolized by ruminal microorganisms or adsorbed to surfaces of RUSITEC equipment.

Release of PFSAs from feed into fermentation fluid was found to be faster for PFBS than for PFOS, whereas differences among PFCAs were not observed. Proportions of PFAAs recovered in the fermentation fluids decreased by increasing chain lengths for the PFSAs and PFCAs. In contrast, levels in fermented feed increased with increasing chain length. The attachment of microorganisms to feed particles was assumed to account for higher PFAA levels in fermented feeds and for lower levels in the fermentation fluids. Furthermore, partitioning of PFAAs to the solid and liquid phase in rumen was particularly influenced by chain length and functional group of PFAAs. Under sterile conditions (experiment B), 99 % of the initial PFAA concentration was totally recovered. In comparison, the total recovered PFAA concentration in experiment A was significantly lower (87 %). The missing percentages of PFAAs seemed to be very high and could not be explained by adhesion of PFAAs to the surfaces of RUSITEC equipment. Therefore, microbial-mediated effects on disappeared PFAAs were suspected.

Since there are optimal reductive conditions for microorganisms in rumen, degradation of PFAAs in rumen seems to be possible. However, results do not univocally indicate if PFAAs were degraded by ruminal fermentation. Therefore, further research is required to elucidate if PFAAs could be degraded by ruminal microorganisms.

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Modeling of toxicokinetics of PFAA in pigs and laying hens

(Modellierung der Toxikokinetik bei Schwein und Legehenne)

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Perfluoroalkyl acids (PFAAs) are perfluoroalkyl substances (PFAS) with a wide range of unique properties and applications. They are very useful in the industry and in consumer products as surface active agents. Unfortunately, they are also persistent in the environment, with bioaccumulative as well as toxic properties (PBT). PFAAs are known to biomagnify in the food chain, becoming absorbed and concentrated in plants, animals, eggs and milk. Here, we study the transfer of a mixture of PFAAs from contaminated feed into the edible tissues of fattening pigs and into laying hens and their eggs. The feed was formulated using hay and barley cultivated and harvested from a contaminated region in Lower Saxony, Germany. Four perfluoroalkyl sulfonic (PFSAs) and three perfluoroalkyl carboxylic acids (PFCAs) were quantifiable in several matrices, such as plasma, muscle tissue and egg yolk using HPLC-MS/MS.

For pigs, as percentages of unexcreted PFAA, the substances accumulated in plasma (up to 51%), fat and muscle tissues (collectively meat 40% to 49%), liver (under 7%) and kidney (under 2%) for most substances. An exception was perfluorooctanesulfonic acid (PFOS), with lower affinity for plasma (23%) and higher for liver (35%). A toxicokinetic model is developed to quantify absorption, distribution and excretion of PFAAs and to calculate elimination half-lives. Perfluorohexanoic acid (PFHxA), a PFCA, had the shortest half-life with 4.1 days. PFSAs are eliminated more slowly (e.g. half-life of 634 days for PFOS). We provide evidence for a fast equilibrium between plasma and edible tissues in pigs, which means that plasma elimination and edible tissue elimination half-lives are identical.

For laying hens, the elimination kinetics are much faster than in pigs. PFAAs were intensively transferred into eggs, and within the egg almost completely enriched in the yolk. Highest levels in yolk were detected for PFOS and perfluorohexanesulfonic acid (PFHxS), followed by perfluorooctanoic acid (PFOA) and low levels of perfluoro-butanesulfonic acid (PFBS). The largest percentages of PFAAs in the body circulated in blood while PFAAs were fed. Similarly to the pig, the liver was a preferential target organ for PFOS accumulation. With the exception of PFOA, only PFSAs ($C \leq 8$) could be detected in breast muscle. After the elimination period of 42 days, PFAAs could not be detected in tissue samples of laying hens, with the exception of low PFHxS levels. However, PFOS and PFHxS were still measurable in yolk, indicating that 42 days of elimination were insufficient for complete PFAAs elimination.

Comparatively, PFAAs in pigs display longer elimination half-lives than in most organisms, but still shorter than in humans. In laying hens, the elimination kinetics through egg yolk are fast, so that the accumulation in tissues of laying hens was considerably lower than in the tissues of fattening pigs. In both organisms, the chemical end-group (sulfonic vs. carboxylic acid) has a large influence; PFSAs are more bioaccumulative than PFCAs. The chain length plays a secondary role. PFAAs can become a risk for consumers of animal products when the feed becomes contaminated. The results provide the basis for generating consistent regulations by relating maximum levels in animal products to maximum levels in animal feed. The toxicokinetic models may also be used by risk managers in the form of tools to guide their decisions in a PFAA crisis situation.

Session III - Exposure of consumer

PFAS in the environment

(PFAS in der Umwelt)

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Per- and polyfluorinated alkyl substances (PFAS) are ubiquitously present in the environment. There are numerous sources of environmental emissions such as manufacturing of PFAS, production and processing of fluoropolymers and side chain fluorinated polymers, use of treated articles and waste disposal. The compounds are distributed world-wide within the environment via surface water (e.g. from waste water treatment plants to rivers and oceans) and air. Humans are mainly exposed to PFAS via food, drinking water and air (Lindström et al., 2011, Butt et al., 2010).

Perfluorinated substances are persistent in the environment. Long chain perfluorinated alkyl substances containing eight or more perfluorinated carbon atoms have been identified to show persistent and bioaccumulative properties. Perfluorooctanoic acid (PFOA) is additionally toxic (Repr. 1B, Carc 2, STOT RE 1). PFOA as well as perfluorinated carboxylic acids with 11 to 14 perfluorinated carbon atoms (C_{11-14} -PFCAs) have recently been identified as Substances of Very High Concern under the European Chemicals Regulation REACH and were added to the Candidate List (ECHA 2013).

Polyfluorinated alkyl substances such as 8:2 fluorotelomer alcohol (8:2 FTOH) which contain a nonfluorinated carbon chain moiety are degraded in the environment to perfluorinated alkyl substances and are therefore called precursor substances (Butt et al., 2013). Polyfluorinated substances are widely used as surfactants and to manufacture side-chain fluorinated polymers. Fluorinated polymers provide e.g. textiles and paper with water, oil, and dirt repellent properties. However, residual monomers are either washed out or evaporate into the surrounding air during the use stage of the respective articles. Surfactants are used e.g. in carpet- and textile-care products and are distributed continuously into the air when applied. It has been shown that indoor air and dust levels especially of outdoor shops and new offices which have been furnished with soil proof carpets have high levels of PFAS (precursors as well as the persistent perfluorinated carboxylates and sulfonates) compared to outdoor air (Schlummer et al., 2013).

Due to regulatory activities during the last years in the US, Canada and Europe industry has been shifting the use of long-chain PFAS to shorter chain PFAS. The result is that the concentrations of some long chain PFAS are decreasing in the environment and also in blood of European and North American people. Short chain PFAS contribute to >50% of the total PFAS in groundwater (Eschauzier et al., 2013).

Short chain perfluorinated alkyl substances are persistent in the environment but due to the higher mobility of these substances the bioaccumulation potential is most probably lower compared to the long chain PFAS. However, the high mobility causes a fast transfer of the substances into groundwater. To date no efficient technologies are known to remove short chain PFAS from water. Thus, a permanent exposure of humans via drinking water may be possible in the future.

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Human biomonitoring of PFCs - internal levels and total exposure

(PFC-Gesamtbelastung aufgrund von bevölkerungsbezogenen Daten aus dem Humanbiomonitoring)

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Perfluorinated compounds (PFCs) are a large group of chemicals produced since several decades and can currently be detected in many environmental media and biota, as well as in humans. Because of their persistence and their potential to accumulate they are of toxicological concern.

Comprehensive data on the internal exposure of the general population from different areas of the world are available. Whilst in European studies, serum and plasma concentrations ranged from 1 to 116 µg/l for perfluorooctane sulfonate (PFOS) and from 0.5 to 40 µg/l for perfluorooctanoate (PFOA), the concentrations in the US and Canada are somewhat higher reaching values up to 656 µg/l (PFOS) and 88 µg/l (PFOA) [1]. Overall, a clearly decreasing trend of the internal exposure was found in the last years, especially for PFOS.

In cord serum, the values for PFOS and PFOA ranged from 0.3 to 2.8 µg/l and from 0.5 to 4.2 µg/l, respectively. Different studies found mean concentration ratios in cord blood in relation to maternal serum of 0.3-0.6 for PFOS and 0.7-1.5 for PFOA [2].

Only limited data are available for infants and toddlers. In one of our studies, we observed median levels of 3.0 and 1.9 µg/l for PFOS and 6.9 and 4.6 µg/l for PFOA in serum samples of children at the age of six and nineteen months, respectively. Overall, we found low levels of PFCs in cord sera and an increase in concentrations through the first months of infant life in this study [2].

The mechanism by which PFCs are transferred from mother's blood to breast milk is not clear, but it is well known that PFCs are strongly bound to the protein fraction in blood and therefore the possibility entering the breast milk is limited. In breast milk samples from Germany, PFOS ranged from <0.03 to 0.11 µg/l (median: 0.04 µg/l), while PFOA was detected only in some samples as were all other PFCs. Although the concentrations in breast milk were low, this intake led to a body burden at the age of six months similar (PFOS) or higher (PFOA) than that found in adults [3].

Using a simplified toxicokinetic model, the total daily exposure could be back-calculated from the concentrations of phthalate metabolites excreated in the urine. The average (and high) level of daily exposure calculated from medians and 95th percentiles or maximum values including all potential routes amounts to 1.6 ng/kg body weight (8.8 ng/kg body weight) for PFOS and 2.9 ng/kg body weight (12.6 ng/kg body weight) for PFOA [1, 4]. The majority of exposure can be attributed to the oral route, mainly to diet. Overall, the contribution of PFOS and PFOA precursors to total exposure seems to be limited.

Besides this background exposure of the general population, a specific additional exposure may occur which causes an increased PFC body burden. This has been observed in populations living near PFC production facilities or in areas with environmental contamination of PFCs. The consumption of highly contaminated fish products may also cause an increase in PFC body burdens.

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Migration of poly- and perfluorinated compounds from food contact materials into food

(Migration von PFAS aus Kontaktmaterialien für Lebensmittel)

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Perfluoroalkylated substances (PFAS) represent a class of environmental contaminants that comprise a vast array of chemical entities being without exception of anthropogenic origin. Due to their special physico-chemical properties, which differ significantly from those of other halogenated compounds, they are extremely persistent against biodegradation or non-biological disintegration. Attributable to their chemical resistance, PFAS are widely used for an enormous assortment of consumer goods, including household products. In addition PFAS are also utilised as additives and coatings of contact materials for food like paper and cardboard. The physico-chemical properties of PFAS, such as their hydrophobic and oleophobic properties make them ideal substances applied as finishing components for paper and cardboards in this area. Examples for such applications comprise sandwich wrappings, microwavable popcorn bags and cup cake liners as well as fast food containers like pizza boxes. Although paper and cardboard surface treatments with PFAS are only indirect sources for the contamination of food with perfluorinated substances, these materials represent potential sources of oral exposure for consumers. However, the exact contribution of perfluorinated packaging materials to the overall perfluoro-contamination of food and beverages is currently not well characterised.

In 2010 the European Commission had taken account of this situation and recommended “Member States should monitor during 2010 and 2011 the presence of perfluoroalkylated substances in food”.

In this study we explored the migration of perfluorinated carboxylic acids (PFCA) and fluorotelomer alcohols (FTOH) from food contact material (FCM). The selection of FCM was performed with respect to the use pattern of typically fluorine-containing FCM. Four application profiles were distinguished:

a) long-term packaging at fridge temperatures
b) long-term packaging at ambient temperatures
c) short-term packaging at elevated temperatures (~80°C)
d) baking applications (temperatures up to 220°C).

Real food items and food simulants were applied, including Poly(2,6-diphenyl-p-phenylene oxide), particle size 60-80 mesh, pore size 200 nm, which is defined as stimulant for dry food by Commission Regulation 10/2011.

For each group of FCM a set of migration test conditions was investigated, at which contact time and temperature as well as food items varied in a reasonable range. Before and after the migration contact, FCM, food items and food simulants were extracted and analyzed for PFSA, PFCA and FTOH by LC tandem mass spectrometer.

Dietary Exposure to Selected Perfluoroalkyl Acids (PFAAs) in Four European Regions

Lebensmittelexposition ausgewählter Perfluoralkylsäuren (PFAS) in vier europäischen Regionen

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The dietary exposure to PFAAs was estimated in four selected European states (Belgium, the Czech Republic, Italy, and Norway) representing Western, Southern, Eastern, and Northern Europe. The harmonised sampling program designed in the EU project PERFOOD[2] was targeted at identifying PFAAs in food items that are most important both in terms of consumption and based on known high contamination pattern. Seven PFAAs have been selected for a dietary exposure assessment, namely perfluorohexanoic acid (PFHxA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnDA), perfluorohexanesulfonic acid (PFHxS) and perfluorooctane sulfonic acid (PFOS). Food consumption data for exposure estimations were taken from the Comprehensive Food Consumption Database built up by EFSA. It provides most recent national food consumption data that have been classified in a harmonised manner.

Event though low limits of quantifications (LOQ) could be archived in PERFOOD, 60% to 90% of the measurements were below the LOQ. The estimated average dietary exposure for adults (18-64 years) is generally below 0.5 ng/kg BW per d for all seven PFAAs (lower bound approach). Considering high consumption of food groups that contribute most to the exposure does not result in estimates exceeding 2 ng/kg BW per d. Thus, based on the TDIs proposed by EFSA for PFOS (150 ng/kg BW per d) and PFOA (1500 ng/kg BW per d), a concern can not be identified.

There are distinct dietary exposure patterns from region to region as a result of different food consumption and contamination patterns. Foods of plant origin (e.g. fruits and vegetables) are most important for the dietary exposure to PFHxA, PFOA and PFHxS, while the consumption of foods of animal origin (particularly fish & seafood) mostly contributes to the dietary exposure to PFDA and PFUnDA. For the dietary exposure to PFNA and PFOS, food of animal and plant origin contribute with equal importance.

In addition, some food items were sampled and analysed from a hot spot (area of a former industrial plant that resulted in environmental exposure to PFAAs) in Belgium. Higher dietary exposures to PFOS and PFOA are estimated as a consequence of the consumption of hen eggs in particular. High chronic consumption of hen eggs from the hot spot, id est one small egg every day, would even lead to a dietary exposure of PFOS that exceeds the TDI by a factor of 2.3.

In conclusion, region to region differences as well as the relative importance of food of different origin for each PFAA should be paid more attention in further research. The consumption of some food items from hot spot areas may result in intake estimates that exceed the established TDI.

[2] EU 7th Framework Programme (FP7)-KBBE-2008-2B, project no. 227525

Human exposure to perfluoroalkyl substances

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Per- and polyfluoroalkyl substances (PFASs) are a class of man-made chemicals which have been produced since the 1950s for a wide range of commercial applications. In recent years, there has a growing concern about human health effects related to the ubiquitous presence of perfluoroalkane sulfonic acids (PFSAs) and perfluoroalkyl carboxylic acids (PFCAs) in human blood serum samples. Knowledge about the primary exposure pathways is essential for the risk assessment and predicting future trends in exposure of these chemicals. In this presentation I will give an overview of the science directed to understanding the exposure pathways of PFASs and highlight areas where more research is needed.

Dietary intake has been suggested as a major pathway of human exposure to perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA). Difficulties associated with the analysis of PFASs in food samples have hampered estimations of dietary intake. However, recent advances in analytical chemistry have dramatically improved the ability to measure PFASs in food matrices at low picogram per gram concentrations and allowed more accurate determination of dietary exposure. Current estimates of dietary intake of PFOS and PFOA are typically a factor of 5 to 10 higher than the exposure from other quantified exposure pathways including drinking water, ingestion of house dust and inhalation of precursor compounds which can be metabolized to PFCAs or PFSAs. High exposure scenarios with different PFAS treated products can, however, result in an elevated total exposure to PFCAs and PFSAs via ingestion of house dust or inhalation of indoor air. It should be noted that major food categories contributing most to the total dietary exposure of PFCAs and PFSAs will vary for different homologues as a consequence of their different physico-chemical properties. For the bioaccumulative long-chain PFCAs and PFSAs, dietary intake of fish, meat and egg will be the primary vectors of exposure. The higher water solubility of short-chain PFCAs and PFSAs, on the other hand, will make drinking water and consumption of vegetables comparatively more important exposure pathways for these compounds. Despite the recent advances in analytical techniques, the mechanisms of food contamination are not very well understood. Thus, more mechanistic research on the accumulation of PFASs in different food webs is needed to understand human exposure to legacy and emerging PFASs.