Updated EHS Summary of Perfluorohexane sulfonic acid for the MA TURA Science Advisory Board Meeting – March 29, 2017

CAS # 355-46-4 	**Perfluorohexane sulfonic acid (PFHxS)** Synonym[1]s: EINECS 206-587-1; 1,1,2,2,3,3,4,4,5,5,6,6,6-Tridecafluorohexane-1-sulfonic acid; Perfluorohexane sulfonic acid; UNII-ZU6Y1E592S **RTECS #[2]:** MO4247000 **EINECS #[3]:** 206-587-1 **Molecular Weight[4]:** 400.1109 **Molecular Formula[5]:** C6-H-F13-O3-S **Common Salts:** Tridecafluorohexane-1-sulfonic acid potassium salt, CAS # 3871-99-6[6] Tridecafluoro-1-hexanesulfonic acid, ammonium salt, CAS # 68259-08-5
PHYSICAL CHARACTERISTICS	
Primary Use	Used as surfactants, to make fluoropolymers and as water and stain protective coatings for carpets, paper and textiles[7]
Physical state, odor at room temperature & pressure	Crystalline, beige (for 3871-99-6)[8]
Melting point; Boiling point	190 deg C/452 deg C (estimated data from EPI Suite version 1.43)[9]
Solubility	Water solubility in mg/L @25C = 243.4 (estimated data from EPI Suite version 1.42)[10]
Specific Gravity	Not found
SAFETY/PHYSICAL HAZARDS	
Vapor Pressure	1.08×10^{-6} Pa @25C (estimated data from EPI Suite version 1.43)[11]
Flammability	Not found
Flashpoint	Not found
Flammability Rating	Not found
Auto Ignition Point	Not found
Combustion products	Not found
Explosivity (UEL, LEL, shock sensitive)	Not found
Oxidizer	Not found
Corrosivity	Not found
pH	Not found
Reactivity	Incompatible materials – strong oxidizing agents[12]
Viscosity	Not found
Odor Threshold	Not found
Particle size, shape, respirable fraction	Not found
Other physical hazards associated with process: Heat, gases under pressure, noise, vibration,	Not found

ergonomic hazard	
HEALTH HAZARDS	
Acute Toxicity	
Oral LD_{50}	No data in the short chain alternatives section in DeWitt; No Tox Data in ChemIDPlus
Dermal LD_{50}	Not found; No Tox Data in ChemIDPlus
Inhalation LC_{50}	Not found; No Tox Data in ChemIDPlus
Intraperitoneal LD_{50}	Not found; No Tox Data in ChemIDPlus
TDLo	Oral, mouse, 6.1 mg/kg[13]
Chronic or Sub-chronic Toxicity	
IARC rating	Not found
Carcinogenicity	Not found on Prop 65 list as of 2/15/17; Not found in CCRIS or GENETOX
Neurotoxicity	Not found in HAZMAP, NIOSH-PG or on the Scorecard list of Suspected Neurotoxicants Other studies have determined neurotoxicity in pups. Following treatment of 10 days (the peak of the brain growth spurt) old NMRI mouse pups with a single oral-oral gavage dose of the potassium salt of PFHxS (0, 0.61, 6.1 or 9.2 mg/kg b. w.), animals in the highest dose group exhibited dose–response related and long-lasting changes in both spontaneous and nicotine-induced behavior as adults (Viberg *et al.*, 2013). In a follow-up study by the authors it was shown that after 24 hours the neuroprotein levels were altered in the highly exposed mice, e.g. calcium/calmodulin-dependent kinase II (CaMKII), growth-associated protein-43 (GAP-43), synaptophysin and tau proteins, which are essential for normal brain development in mice. This was measured for both males and females, in hippocampus and cerebral cortex. There were also altered levels of neuroproteins in adult male mice explaining the results in the previous publication. These results suggest that PFHxS may act as a developmental neurotoxicant, and the effects are similar to that of PFOS and PFOA (Lee and Viberg 2013).[14] Data from the NHANES 1999-2004 and the C8-Health Project in the USA surveys showed positive association between some serum PFAA levels and attention deficit-hyperactivity disorder (ADHD) in children (Hoffman *et al.* 2010; Stein and Savitz 2011). The later study found a specific association with ADHD and PFHxS blood levels. The prevalence of ADHD plus medication increased with perfluorohexane sulfonate (PFHxS) levels, with an adjusted odds ratio of 1.59 (95% confidence interval, 1.21–2.08) comparing the highest quartile of exposure to the lowest.[15] Note: "On the basis of epidemiologic and other data available to the C8 Science Panel, they concluded that there is not a probable link between

	exposure to C8 (also known as PFOA) and neurodevelopmental disorders in children, including attention deficit disorders and learning disabilities." [16] Higher blood levels of PFOS, PFNA, PFDA, PFHxS and PFOSA (but not PFOA) were associated with significantly shorter "Impaired Response Inhibition" (IRT) during the "differential reinforcement of low rates of responding" (DRL) tasks measuring children's impulsivity (Gump *et al.* 2011). PFHxS was the second most abundant in the blood with a mean blood concentrations of about 6 ng/mL. The mean concentration of PFOS was higher and about 10 ng/mL, and the mean concentration of PFOA was about 3 ng/mL.[17]
Developmental/Reproductive Toxicity	Not found on Prop 65 list as of 2/15/17 A study of a large cohort from Avon in the UK with prenatal blood concentration (medians) of 19.2 ng/mL PFOS, 3.7 ng/mL PFOA and 1.6 ng/mL PFHxS showed that the most exposed mothers from the upper tertile gave birth to girls weighing 140 gram less than for the less exposed but at 20 months the girls with high PFOS exposure weighed 580 gram more (Maisonet *et al.* 2012). In a study from Canada there was no significant effect of PFAS on birth weight. The blood levels were, however, somewhat lower with medians of 7.8, 1.5 and 0.97 ng/mL for PFOS, PFOA and PFHxS, respectively (Hamm *et al.* 2010). That may not be a problem of the mother alone, because another Danish study found that high levels of perfluorinated acids (PFAAs) (medians: 24.5 ng PFOS/mL, 4.9 ng PFOA/mL and 6.6 ng PFHxS/mL) in blood serum were associated with fewer normal sperm cells in normal young men included in the study (Joensen *et al.* 2009). After adjusting for age, race/ethnicity, education, ever smoking, and parity, women with higher levels of PFAS had still earlier menopause than did women with the lowest PFAS levels (Taylor *et al.* 2014. Specifically, a monotonic association with PFHxS was observed: The hazard ratio (HR) was 1.42 (95% CI: 1.08, 1.87) for serum concentrations in tertile 2 versus tertile 1, and 1.70 (95% CI: 1.36, 2.12) for tertile 3 versus tertile 1).[18] The potential reproductive and developmental toxicity of perfluorohexane sulfonate (PFHxS) was studied in a study with rats dosed by gavage at 0.3, 1, 3, and 10 mg/kg/d 14 days prior to co-habitation, during cohabitation, and until the day before sacrifice (21 days of lactation or presumed gestation day 25 (if not pregnant) for females and minimum of 42 days of treatment for males). Offspring were not dosed by gavage but were exposed by placental transfer in utero and potentially exposed via milk. At all doses reductions in serum

	total cholesterol and other biochemical changes in the blood but no reproductive or developmental effects were observed, and there were no treatment-related effects in dams or offspring (Butenhoff *et al.* 2009). Thus, in this rodent study the metabolism of lipids was affected at a daily exposure for 0.3 mg/kg b. w., and liver damage was observed after exposure to 3 mg/kg b. w. per day (NOAEL = 1 mg/kg per day). A NOAEL of 10 mg/kg b. w. per day (highest concentration tested) for effects on the reproduction was determined for PFHxS.[19]
Genotoxicity/Mutagenicity	Not found in CCRIS or GENE-TOX
Endocrine Disruption/Thyroid Effects	Found on TEDX List of Potential Endocrine Disruptors[20] Data from National Health and Nutrition Examination Survey (NHANES) for the years 2007–2008 were used to evaluate the effect of PFOS, PFOA, PFNA, PFDA, PFHxS, and 2-(*N*-methyl-perfluorooctane sulfonamide) acetic acid on the levels of six thyroid function variables (Jain 2013). Levels of triiodothyronine were found to increase with the levels of PFOA ($p=0.01$), and total thyroxine levels were found to increase with increase in PFHxS levels ($p<0.01$).[21] In many PFAS toxicology studies decreased thyroid hormone levels are observed. The mechanism is a competitive binding to the thyroid hormone plasma transport protein transthyretin (TTR) that will alter/decrease the free thyroxine (T4) in blood. This competitive binding capacity of some poly- and perfluorinated compounds was studied by Weiss *et al.* (2009) with a radio-ligand-binding assay. The binding potency of the fluorinated chemicals was 12-300 times lower than for thyroxine itself and decreased in the order: PFHxS > PFOS/PFOA > PFHxA > PFBS.[22] PFHxS (and PFOS and PFOA) acts as a 17β-Estradiol (ER) agonist *in vitro* and enhanced significantly the E2-induced estrogen receptor (ER) response in human MVLN breast cancer cells (Kjeldsen *et al.* 2013).[23]
Immunotoxicity	An investigation of children aged 5 and 7 years from Faroe Island in the Atlantic showed that commonly prevalent exposures to PFOS, PFOA, PFHxS, PFNA and PFDA measured in blood serum were associated with lower anti-body responses to childhood immunizations (vaccinations) and an increased risk of antibody concentrations below the level needed to provide long-term protection against diphtheria and tetanus (Grandjean *et al.* 2012).[24]
Other organ toxicity	Many PFAS are highly potent peroxisome proliferators in rodent livers and affect mitochondrial, microsomal, and cytosolic enzymes and proteins involved in lipid metabolism (Ikeda *et al.* 1985; Van den Heuvel 1996; Upham et. al. 1998; Kudo *et al.* 2000). The liver fatty acid–binding protein (L-FABP) is a transport protein known to bind PFAS (Luebker *et al.*, 2002).

	The liver toxicity and peroxisome proliferation potency in rats depends on the carbon chain length. PFCA activated both mouse and human PPARα in a concentration dependent fashion, and activation of PPARα by PFCA was positively correlated with carbon chain length, up to C9. PPARα activity was higher in response to carboxylates compared to sulfonates. Activation of mouse PPARα was generally higher compared to that of human PPARα (Wolf *et al.* 2008). The relative activity increased from PFBS < PFOS < PFHxS < PFBA < PFHxA < PFOA. [25]
Skin, Eye and Respiratory Effects	
*Irritant – **S**kin, **E**ye, or **R**espiratory*	Skin irritation (Category 2), H315 (for 3871-99-6); Eye irritation (Category 2A), H319 (for 3871-99-6); Specific target organ toxicity – single exposure (Category 3), Respiratory system, H335 (for 3871-99-6)[26]
*Corrosive – **S, E,** or **R***	
*Permanent Damage – **S, E,** or **R***	
*Sensitizer– **S** & **R***	Not found in AOEC database
Asthmagen – Initiator or Exacerbator	Not found in AOEC database In a study from Taiwan PFAS serum levels including of PFHxS were reported to be significantly higher in children with asthma compared to children without asthma (Dong *et al.* 2013).
Skin Absorption, Kp	It is known from animal studies that the studied short chain polyfluoroalkylated substances (PFAS) are almost completely absorbed orally and by inhalation but that skin absorption may be negligible.[27]
LOAEL	
NOAEL	Thus, in this rodent study the metabolism of lipids was affected at a daily exposure for 0.3 mg/kg b. w., and liver damage was observed after exposure to 3 mg/kg b. w. per day (NOAEL = 1 mg/kg per day). A NOAEL of 10 mg/kg b. w. per day (highest concentration tested) for effects on the reproduction was determined for PFHxS.[28]
Benchmark Dose Response (BMD)	
Metabolites	The toxicokinetics of the potassium salt of PFHxS after a single intravenous exposure (10 mg/kg b. w.) was compared in rats, mice and monkeys (Sundström *et al.* 2012). Urine was the major route of excretion in male and female rats, and mean daily fecal excretion was <0.5% of administered dose at all times. Within 96 hours females excreted 28% of a dose in urine. Males excreted only about 6–7% of a dose in urine and had very much higher levels of PFHxS in blood and liver. The excretion increased with the dose. The mean serum elimination half-lives in male and female rats were calculated to 6.83 days and 1.83±0.26 days, respectively. These values are not likely to be reliable due to the short duration (24 hours). A comparison between intravenous- and oral exposures showed a PFHxS bioavailability of

	about 50%. After 10 weeks the mean serum elimination half-lives in male rats was calculated to about 29 days. In females the levels of PFHxS in the blood after 10 weeks were too low to quantify. In mice given oral doses of 20 mg PFHxS-K/kg body weight the mean serum elimination half-lives in males and females were 30.5 and 24.8 days, respectively, and not so different as for rats. Elimination in urine dominated also in mice but it was less than for rats. After 24 hours <3% of a dose was recovered in urine. In monkeys, PFHxS was much more long-lived in the blood with mean serum elimination half-lives for females and males of 87±27 days versus 141±30 days, respectively; however, this difference was not statistically significant. Less than 0.1 % of a dose was determined in the urine, thus renal elimination was very slow in monkeys.[29]
Synergistic or Antagonistic Effects	
Environmental and Human Health Exposure and Risk Values	
RfC/RfD	Not found in IRIS database
ATSDR-MRL	Not found on ATSDR-MRL March 2016 list (Note PFOA and PFOS are on the list)
Adverse Effect Levels: DNEL, PNEC, PNEL	
Health Based Exposure Limits	
NIOSH-REL/IDLH/Ceiling Limits	Not found in NIOSH-PG
OSHA-PEL	Not found in Z tables
ACGIH TLV-TWA	Not found in Z tables or in RTECS
TLV-STEL	Not found in Z tables
Biomonitoring Action Limits	
Drinking Water Standards	
Other	
ENVIRONMENTAL & ECO-SYSTEM HAZARDS (Bulk of information cited UNEP 2015a and UNEP 2015b)	
PBT	PFHxS is considered as persistent and stable in the environment and is regarded as degradation product of other perfluorinated compounds.[30]
Bioaccumulation	In one study, the Log BCFs of the C4-C7 sulfonic acids were all found to be below 1 in fish thus indicating little bioaccumulation potential of these substances in this organism group in contrast to long-chain (C11-C13) PFSAs.[31] In blood from some office workers in Boston exposed to FTOHs. PFHxA was not detectable but PFHxS reached 0.2-13 ng/mL with a geomean of 1.5 ng/mL (Fraser *et al.* 2012).[32] In retired workers from the fluorochemical producing industry serum half-lives for PFHxS (perfluorohexane sulfonate) were 7.3-8.5 years or about twice the half-lives for PFOS and PFOA (Olsen *et al.* 2007). Thus,

	the half-life for PFHxS in rats is, like for other PFAS, much shorter than in humans. However, the half-life of PFHxS is shorter in rats than the half-life (40 days) of PFOS in rats. The long residence time of PFHxS in human blood may explain the relatively low organ concentrations of this chemical compared to other PFASs measured in Spanish autopsy tissues. The highest concentration of PFHxS was found in the kidneys but 20 times lower compared to PFBA (Perez *et al.* 2013).[33] Presence in environment and biota: - Study of Spanish Jucar river basin, water and biota samples – water conc. 12.07-36.7 ng/L, detected in 13% of samples, non-detect in sediment; detected in biota 0.63 µg/kg in one fish sample (limits of quantification 0.02-2.26 µg/kg.)[34] - Harbor seals 33 µg whole body burden (compared with 2,500 µg PFOS). Concentration in tissues: thymus 10.5 ng/g wet wt, lung 8 ng/g, liver, kidney, heart, thyroid btwn 4-7 ng/g.[35] - Female herring gull eggs: 0.8 ng/g ww yolk, albumen ND - Herring gull tissue: plasma 8 ng/g ww, liver 0.8 ng/g, brain ND – 1.5 ng/g, muscle ND – 2.1 ng/g, adipose ND-0.2 ng/g[36] - Arctic food web frequency of detection: 30% capelin, 67% cod (up to 3.5 ng/g ww), 0% sediments, macroalgae, and duck liver; 50% beluga whale fetus (up to 4 ng/g), 11-14% blood and liver (up to 3.7 ng/g)[37] - Rainbow trout PFHxS half-life: carcass 11 days, blood 10 days, liver 12 days. Tissue concentrations ~0.05 – 0.1 µg/g kidney, liver, gall bladder, blood plasma, gill, gonads. <0.01 in muscle, but given that muscle is ~67% of trout by wt, could contain ~60% of total body burden of PFHxS.[38] - Human: mean concentration kidney 20.8 ng/g ww, lung 8.1 ng/g, brain 3.2 ng/g, bone 1.8 ng/g, liver 4.6 ng/g[39] Plant bioaccumulation: hydroponic (water only) uptake rate constant k_1 (per day) 2±1 in roots, 0.04±0.04 in shoots; elimination half life 0.17 days; (this rapid elimination was similar for all PFAS studied except PFBA, which had 1.83 day half life)[40] Cape Cod groundwater: detected in 55% of 20 private wells sampled, max concentration 41 ng/L. Sampling from other studies and locations, groundwater and surface water, varied from 9.3 – 32 ng/L.[41]
BAF	
BCF	Tissue half-life: Rainbow trout 10-12 days; calculated BCF 100 (liver), 76 (blood), 12 day accumulation ratio = 54-59[42]
BMF	

Ecological Toxicity	Toxicity data on PFHxS have not been available. Considering the conclusions on chain length and presence of functional groups of PFAS, it can be expected that PFHxS shows increased toxicity compared to PFBS, as well as increased toxicity compared to PFHxA.[43]
Aquatic Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	
Mammalian Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	
Wildlife Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	
Breakdown/degradation /combustion products	The compound is not expected to undergo hydrolysis or photolysis, and no biodegradation is expected.[44]
Anaerobic degradation	
Aerobic degradation	
Other observable ecological effects (e.g. BOD)	
Fate and Transport: Aquatic	The Danish Report noted that fate data on PFHxS are very sparse.[45] Based on the read-across approach, conclusions applying to the fate of PFBS can be anticipated to be valid for PFHxS as well. Thus, the compound is not expected to undergo hydrolysis or photolysis, and no biodegradation is expected. The substance was, like other PFAS, found to be poorly removed in WWTPs.[46]
Fate and Transport: Terrestrial	
Fate and Transport: Atmospheric	
Transport Issues	
Factors affecting bioavailability	
Global Environmental Impacts	
Ozone Depletion Potential (ODP)	Not found
Global Climate Change	Not found
Greenhouse Gas Production	Not found
Acid Rain Formation	Not relevant
Special Reports	
EU	**Short-chain Polyfluoroalkyl Substances (PFAS)** – A literature review of information on human health effects and environmental fate and effect aspects of short-chain PFAS, Environmental project No. 1707, 2015 http://www2.mst.dk/Udgiv/publications/2015/05/978-87-93352-15-5.pdf **Polyfluoroalkyl substances (PFASs) in textiles for children** – Survey of chemical substances in consumer products No. 136, 2015 http://www2.mst.dk/Udgiv/publications/2015/04/978-87-93352-12-4.pdf

	Survey of PFOS, PFOA and other perfluoroalkyl and polyfluoroalkyl substances – Part of the LOUS-review, Environmental project No. 1475, 2013 http://www2.mst.dk/Udgiv/publications/2013/04/978-87-93026-03-2.pdf

Notes on chemical research: Not found in NIOSH Pocket Guide; HSDB; HAZMAP

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[1] www.expub.com; Chemical Identity Page for Perfluorohexane sulfonic acid.
[2] www.expub.com; RTECS for Perfluorohexane sulfonic acid.
[3] www.expub.com; Chemical Identity Page for Perfluorohexane sulfonic acid.
[4] U.S. National Library of Medicine, ChemIDplus, a Toxnet Database, entry for "Perfluorohexane sulfonic acid.", accessed online at: https://chem.sis.nlm.nih.gov/chemidplus/rn/startswith/355-46-4
[5] U.S. National Library of Medicine, ChemIDplus, a Toxnet Database, entry for "Perfluorohexane sulfonic acid.", accessed online at: https://chem.sis.nlm.nih.gov/chemidplus/rn/startswith/355-46-4
[6] **Sigma-Aldrich SDS 2014:** Sigma Aldrich Safety Data Sheet for Tridecafluorohexane-1-sulfonic acid potassium salt, CAS# 3871-99-6, Revision Date 12/22/14, Accessed online 2/15/17 at: http://www.sigmaaldrich.com/safety-center.html
[7] NTP 2017: National Toxicology Program, Testing Status of Perfluorohexane sulfonate, PFHXS – M040005, "Known Uses". Accessed online at: https://ntp.niehs.nih.gov/testing/status/agents/ts-m040005.html
[8] Sigma-Aldrich SDS 2014.
[9] **UNEP 2012:** United Nations Environment Programme. Technical paper on the identification and assessment of alternatives to the use of perfluorooctane sulfonic acid in open applications. November 2012. Accessed online at: http://chm.pops.int/Default.aspx?tabid=2801.
[10] UNEP 2012.
[11] UNEP 2012.
[12] Sigma-Aldrich SDS 2014.
[13] www.expub.com; RTECS for Perfluorohexane sulfonic acid.
[14] **Danish EPA 2015b:** Danish Environmental Protection Agency. Short-chain Polyfluoroalkyl substances (PFAS) – A literature review of information on human health effects and environmental fate and effect aspects of short-chain PFAS. Environmental project No. 1707, 2015. Accessed online at: http://www2.mst.dk/Udgiv/publications/2015/05/978-87-93352-15-5.pdf
[15] Danish EPA 2015b. Page 35.
[16] C8 Health Panel Probable Link Reports Web Page, July 30, 2012, Accessed online at: http://www.c8sciencepanel.org/prob_link.html.
[17] Danish EPA 2015b. Pages 35-36.
[18] Danish EPA 2015b. Page 35.
[19] Danish EPA 2015b. Page 33.
[20] TEDX: Search for 355-46-4, Accessed online, 1/13/17, http://www.endocrinedisruption.org/endocrine-disruption/tedx-list-of-potential-endocrine-disruptors/chemicalsearch?sname=&x=49&y=14&action=search&sall=1&searchfor=any&scas=355-46-4&searchcats=all
[21] Danish EPA 2015b. Page 35.
[22] Danish EPA 2015b. Page 30.
[23] Danish EPA 2015b. Page 34.
[24] Danish EPA 2015b. Page 35.
[25] Danish EPA 2015b. Pages 28-29.
[26] Sigma-Aldrich SDS 2014.
[27] Danish EPA 2015b. Page 7.

[28] Danish EPA 2015b. Page 33.
[29] Danish EPA 2015b. Pages 32-33.
[30] UNEP 2012.
[31] Danish EPA 2015b. Page 51.
[32] Danish EPA 2015b. Page 25.
[33] Danish EPA 2015b. Page 33.
[34] Campo et al, 2016. Analysis of the presence of perfluoroalkyl substances in water, sediment and biota of the Jucar River (E Spain), Sources, partitioning and relationships with water physical characteristics, *Environmental Research* **147** (2016) 503-512.
[35] **Ahrens 2009:** Ahrens L, et al. Total body burden and tissue distribution of polyfluorinated compounds in harbor seals (*Phoca vitulina*) from the German Bight. *Marine Pollution Bulletin* **58** (2009) 520-525; and **Ng and Hungerbuhler 2014:** Ng, CA and Hungerbuhler K. Bioaccumulation of Perfluorinated Alkyl Acids: Observations and Models. *Environmental Science & Technology* 2014, **48**, 4637-4648.
[36] **Gebbink and Letcher 2012:** Gebbink WA and Letcher RJ. Comparative tissue and body compartment accumulation and maternal transfer to eggs of perfluoroalkyl sulfonates and carboxylates in Great Lakes herring gulls. *Environmental Pollution* **162** (2012) 40-47.
[37] **Kelly 2009a:** Supporting information for "Kelly BC, et al. Perfluoroalkyl Contaminants in an Arctic Marine Food Web: Trophic Magnification and Wildlife Exposure. *Environmental Science & Technology* 2009, **43**, 4037-4043".
[38] **Martin and Mabury 2003**: Martin JW, Mabury SA, et al. Bioconcentration and Tissue Distribution of Perfluorinated Acids in Rainbow Trout (*Oncorhynchus Mykiss*). *Environmental Toxicology and Chemistry*, **Vol. 22, No. 1**, pp. 196-204, 2003.
[39] **Perez 2013:** Perez F, et al. Accumulation of perfluoroalkyl substances in human tissues. *Environment International* **59** (2013) 354-362.
[40] **Muller 2016:** Muller CE, et al. Competing Mechanisms for Perfluoroalkyl Acid Accumulation in Plants Revealed Using an Arabidopsis Model System. *Environmental Toxicology and Chemistry*, **35(5),** pp. 1138-1147, 2016.
[41] **Schaider 2016:** Schaider, LA, et al. Septic systems as sources of organic wastewater compounds in domestic drinking water wells in a shallow sand and gravel aquifer. *Science of the Total Environment* **547** (2016) 470-481.
[42] Martin and Mabury 2003.
[43] Danish EPA 2015b. Page 62.
[44] Danish EPA 2015b. Page 62.
[45] Danish EPA 2015b. Page 62.
[46] Danish EPA 2015b. Page 62.