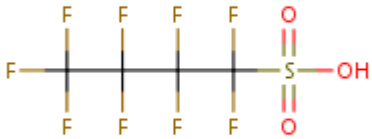


## Draft EHS Summary of Perfluorobutanesulfonic acid for the MA TURA Science Advisory Board Meeting – April 11, 2018

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| <p><b>CAS # 375-73-5</b></p>  | <p><b>Perfluorobutane sulfonic acid (PFBS)</b><br/>           Synonym<sup>1</sup>s: EINECS 206-793-1; Nonafluorobutanesulfonic acid; Nonafluoro-1-butanesulfonic acid; 1,1,2,2,3,3,4,4,4-Nonafluoro-1-butanesulfonic acid; 1-Perfluorobutanesulfonic acid;<br/>           Perfluorobutanesulfonic acid<br/> <b>RTECS #<sup>2</sup>:</b> EK5930000<br/> <b>EINECS #<sup>3</sup>:</b> 206-793-1<br/> <b>Molecular Weight<sup>4</sup>:</b> 300.0969<br/> <b>Molecular Formula<sup>5</sup>:</b> C<sub>4</sub>H-F<sub>9</sub>O<sub>3</sub>-S<br/> <b>Common Salts:</b><br/>           Perfluorobutane sulfonate, potassium salt, CAS # 29420-49-3</p>   |
| <b>PHYSICAL CHARACTERISTICS</b>  |  |
| <p><i>Primary Use</i></p>  | <p>“Potassium perfluorobutane sulfonate (K-PFBS, CAS # 29420-49-3) is a commercial product. In addition, PFBS is a degradation product and potential impurity in perfluoroalkane sulfonyl-based electrochemical fluorination products (Buck et al. 2011a). PFSA’s such as PFBS were released directly into the environment during the historical manufacture and use per- and poly-fluoroalkyl substances (Buck et al. 2011a).”<sup>6</sup></p> <p>“Perfluorobutane sulfonate (PFBS)(CASRN 375-73-5) and its related salts called potassium perfluorobutane sulfonate (K+PFBS)(CASRN 29420-49-3) are polyfluorinated compounds (PFCs) manufactured for use in paints, cleaning agents, and water-impermeable products (Rosal et al., 2010)”.<sup>7</sup></p> <p>“Perfluoroalkanesulfonate anions are good leaving groups. Esters of perfluoroalkanesulfonic acids are therefore used as strong alkylating agents in preparative chemistry.”<sup>8</sup></p> <p>In 2003, 3M Co. began replacement of PFOS in Scotchgard® with PFBS and related substances.</p> <p>The starting material for manufacture of the substances is perfluorobutane sulfonyl fluoride (PBSF)... The main application area identified is water and stain repellent protection for leather, textiles and carpets and porous hard surfaces, representing 25-50 t/year of PFBS moieties in mixtures. Minor application areas include surfactants for inks, paints, waxes, etc.; flame retardants for polycarbonate; mist suppressants for metal plating, and surfactants for fluxes for production of electronics.<sup>9</sup></p> |
| <p><i>Physical state, odor at room temperature &amp; pressure</i></p>  | <p>Colorless liquid<sup>10</sup><br/>           Potassium PFBS = solid<sup>11</sup></p>  |
| <p><i>Melting point; Boiling point</i></p>   | <p>For Perfluorobutane sulfonate, potassium salt; <b>MP</b> = 188 deg C; <b>BP</b> = 447 deg C<sup>12</sup></p>  |

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|   | <p>“PFBS (free acid), <b>BP</b> = 200 deg C; K+PFBS (potassium salt), <b>BP</b> = 76-84 deg C”<sup>13</sup></p> <p>Potassium PFBS, MP = 270 deg C (exp.)<sup>14</sup></p>  |
| <i>Solubility</i>   | <p>Perfluorobutane sulfonate, potassium salt = 4,340 mg/L in water (estimated)<sup>15</sup> “Exists in its anionic form in aqueous medium”<sup>16</sup></p> <p>“PFBS (free acid), solubility in water (mg/L) = 56.6 @ 24 deg C; K+PFBS (potassium salt), solubility in water (mg/L) = 46.2 @ 20 deg C”<sup>17</sup></p> <p>Potassium PFBS, water solubility = 52.6-56.6 mg/L (exp.)<sup>18</sup></p> |
| <i>Specific Gravity</i>   | 1.811 g/mL at 25 deg C <sup>19</sup>   |
| <b>SAFETY/PHYSICAL HAZARDS</b>  |  |
| <i>Vapor Pressure</i>   | <p>Perfluorobutane sulfonate, potassium salt<sup>20</sup> = <math>1.49 \times 10^{-6}</math>;</p> <p>K+PFBS (potassium salt) = <math>9.15 \times 10^{-8}</math> (mm Hg at 20 deg C)<sup>21</sup></p> <p>Potassium PFBS, <math>&lt; 1.22 \times 10^{-5}</math> Pa (exp.)<sup>22</sup></p>   |
| <i>Flammability</i>   | Not found  |
| <i>Flashpoint</i>   | Not found  |
| <i>Flammability Rating</i>  | Not found  |
| <i>Auto Ignition Point</i>  | Not found  |
| <i>Combustion products</i>  | Special hazards arising from the substance or mixture: Carbon oxides, Sulfur oxides, Hydrogen fluoride <sup>23</sup>   |
| <i>Explosivity (UEL, LEL, shock sensitive)</i>  | Not found  |
| <i>Oxidizer</i>   | Not found  |
| <i>Corrosivity</i>  |  |
| <i>pH</i>   | No data <sup>24</sup>  |
| <i>Reactivity</i>   | Reacts violently with water [ <i>TURI note: This is possibly for the anhydride form</i> ]; Incompatible materials: Strong oxidizing agents <sup>25</sup>   |
| <i>Viscosity</i>  | Not found  |
| <i>Odor Threshold</i>   | Not found  |
| <i>Particle size, shape, respirable fraction</i>  | Not found  |
| <i>Other physical hazards associated with process: Heat, gases under pressure, noise, vibration, ergonomic hazard</i> | Not found  |
| <b>HEALTH HAZARDS</b>   |  |
| <b>Acute Toxicity</b>   |  |
| <i>Oral LD<sub>50</sub></i>   | <p>Rat = 430 mg/kg (RTECS) (<i>Study Information posted to Lib Guide as Bayer 1982</i>)</p> <p>Potassium PFBS has low acute toxicity via the oral route with an oral median lethal dose (LD<sub>50</sub>) &gt; 2,000 mg/kg bw in rats. A single oral dose</p>  |

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|  | was administered by gavage to fasted animals. No treatment-related adverse clinical observations, mortality, changes in body weight or gross pathology were found (NICNAS, 2005). <sup>26</sup>   |
| <i>Dermal LD<sub>50</sub></i>              | Potassium PFBS and PFBSF have low acute toxicity via the dermal route with dermal LD <sub>50s</sub> > 2,000 mg/kg bw. A single dose of each chemical was administered under semi-occlusive conditions for 24 hours. There were no clinical signs of reaction to treatment, nor dermal response to treatment observed in any animal throughout the studies (NICNAS, 2005; REACH). <sup>27</sup>  |
| <i>Inhalation LC<sub>50</sub></i>          | A median lethal concentration (LC <sub>50</sub> ) (for PFBSF) was not determined but is considered to be > 5,000 ppm (62 mg/L) (REACH). <sup>28</sup>   |
| <i>Intraperitoneal LD<sub>50</sub></i>     | Not found   |
| <b>Chronic or Sub-chronic Toxicity</b>     |   |
| <i>IARC rating</i>                         | Not found on IARC website   |
| <i>Carcinogenicity</i>                     | Not found on Prop 65 list (as of 5/05/17);  |
| <i>Neurotoxicity</i>                       | "Neurological alterations were reported in the 28-day (Premedica Redfield 2001) but not the 90-day oral study (Leider et al 2009b and York 2003a) in rats. The results from the 28-day study are difficult to interpret. Treated males did differ from control males, however, the decreases in tail flick, rotorod latency and foot splay did not exhibit a dose-response at the doses tested. In contrast, treated females exhibited an increase in rotorod latency. The 90 day study, which included a FOB and motor activity assessment but not a peripheral neuropathy assessment per se, did not report any treatment related effects. A database UF was incorporated into the derivation of the subchronic and chronic RfDs, in part, to address the need for additional neurological testing." <sup>29</sup><br>"Additional studies regarding neurological effects are warranted." <sup>30</sup><br>PC12 cell in vitro study: Concentration-effect curve is biphasic, evoking developmental neurotoxicity through direct actions on replicating and differentiating neurons. PFBS retarded differentiation into both ACh and DA phenotypes, an effect not seen with PFOS, PFOA or PFOSA. Both TH and ChAT were significantly reduced indicating likelihood of impaired function for both neurotransmitters. Rank order of adverse effect from the 4 substances: PFOSA > PFOS > PFBS ~ PFOA. <sup>31</sup> |
| <i>Developmental/Reproductive Toxicity</i> | Not found on Prop 65 list (as of 5/05/17);<br>"In a two-generation reproduction study (Lieder et al. 2009a), parental-generation (P) rats were dosed orally by gavage with 0, 30, 100, 300, and 1,000 mg K-PFBS for 10 weeks prior to and through mating (males and females), as well as during gestation and lactation (females only). First generation (F1) pups were dosed similarly, beginning at weaning.  |

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Second generation (F2) pups were not directly dosed but potentially exposed to PFBS through placental transfer and nursing, and the study was terminated 3 weeks after their birth. **The NOAEL in the parental generations (P and F1) was 100 mg/kg/day.** In the 300 and 1,000 mg/kg/day dose group rats, there were (1) increased liver weight (absolute or relative) and corresponding increased incidence of adaptive hepatocellular hypertrophy (male only) and (2) increased incidence of minimal to mild microscopic findings in the medulla and papilla of the kidneys (male and female). There were no K+PFBS treatment-related effects on fertility or reproduction among the P or the F1 rats. There were no microscopic changes in male or female reproductive organs, and no biologically relevant effects on sperm parameters, mating, estrous cycles, pregnancy, and natural delivery in the P- or F1-generations. There were no K+PFBS treatment-related effects on survival of pups in the two-generation study. Litter size and average pup birth weight per litter were not statistically significantly different from controls in any dose group. In the F1-generation, terminal body weight was reduced in males at 1,000 mg/kg/day. Preputial separation was slightly delayed (approximately 2 days) at this dose, a finding consistent with the body weight reduction. Essentially no effects were observed in the F1 females. F2 pups had normal bodyweights. The reproductive NOAEL was >1,000 mg/kg/day in both generations.”<sup>32</sup>

“An oral developmental study and a 2 generational study have been conducted in rats. Both studies identified a NOAEL for developmental effects 5-fold higher than the critical study (Lieder et al 2009b) NOAEL (MN DPH 2011, Page 3)”.<sup>33</sup>

“An oral 2 generation study in rats has been conducted. No treatment related effects on female reproductive parameters were noted. Decreased number of spermatids per gram testes and increased incidence of abnormal sperm were noted at doses >15-fold higher than the critical study (Lieder et al 2009a) NOAEL. Mating and fertility parameters were unaffected (MN DPH 2011, Page 3)”.<sup>34</sup>

“The potassium salt of PFBS (K+PFBS) had no effect on 3β or 17β-HSD activity in human or rat testes microsomes, even at high concentrations (Zhao et al., 2010)”.<sup>35</sup>

“Potassium perfluorobutane sulfonate (PFBS-K) has in one study been assessed for developmental and reproductive effects in rats at maternal doses until 1 g/kg/day. No adverse effect on embryo/fetal development was noted, and no significant alterations were observed

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|  | <p>in a two-generation study (Lau et al. 2004).<sup>36</sup></p> <p>Our preliminary evidence suggests that exposure to PFBS may increase the risk of female infertility due to endometriosis (Wang et al, 2017).<sup>37</sup></p> <p>“...when PFBS (200 and 500 mg/kg/day) was orally administered to pregnant mice (PFBS-dams) on days 1-20 of gestation; their female offspring (PFBS-offspring) exhibited decreased perinatal body weight and delayed eye opening compared with control offspring. Vaginal opening and first estrus were also significantly delayed in PFBS-offspring, and diestrus was prolonged. Ovarian and uterine size, as well as follicle and corpus luteum numbers, were reduced in adult PFBS-offspring. Furthermore, pubertal and adult PFBS-offspring exhibited decreases in serum estrogen (E2) and progesterone (P4) levels with the elevation of luteinizing hormone levels (Feng 2017).”<sup>38</sup></p>   |
| <p><i>Genotoxicity/Mutagenicity</i></p>    | <p>Not found in CCRIS, GENE-TOX (as of 5/05/17)</p> <p>“PFBS (0-2,000 μM) did not induce ROS or oxidative DNA damage in Hep G2 cells, and lactate dehydrogenase release into the cell media following exposure to PFBS was the same as in control cells (&lt; 5 %) (Eriksen et al., 2010).”<sup>39</sup></p> <p>“Potassium PFBS was not mutagenic (up to 5,000 μg/plate) to <i>Salmonella typhimurium</i> (TA98, TA100, TA1535 and TA1537 strains) or <i>Escherichia coli</i> (WP2 uvrA strain) with or without metabolic activation. The chemical was negative (up to 5,000 μg/mL) in a chromosomal aberration test using Chinese hamster ovary W-B1 cells with or without metabolic activation (NICNAS, 2005).</p> <p>PFBSF was negative (with and without metabolic activation) in Ames test (up to 5,000 μg) in both <i>S. typhimurium</i> (TA135, TA 1537, TA98 and TA100 strains) and <i>E. coli</i> (WP2uvrA/pKM101 strain), and in a chromosomal aberration test using human lymphocytes (REACH).”<sup>40</sup></p> |
| <p><i>Endocrine Disruption/Thyroid</i></p> | <p>Found on TEDX List of Potential Endocrine Disruptors<sup>41</sup></p> <p>“The cytotoxicity of eight PFASs, including PFBA, PFHxA, PFBS, and PFHxS was assessed in the human placental choriocarcinoma cell line JEG-3 (Gorrochategui et al. 2014). Only the long chain PFAS (PFOS, PFDoA, PFNA, PFOA) showed significant cytotoxicity but it was showed that PFBS (+ PFOS and PFOA) acted as aromatase inhibitors in placental cells. This inhibitory effect of the short chain PFBS was considered particularly important, because it is often considered a safe substitute of PFOS.”<sup>42</sup> “(Gorrochategui et al. 2014)....suggesting that PFBS could potentially alter the equilibrium between androgens and estrogens. These researchers noted inhibition of aromatase activity by PFBS occurred in these cells at very low levels, as PFBS uptake by the cells</p>   |

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|  | <p>was poor (below the limit of detection)."<sup>43</sup><br/>         "Additional studies regarding thyroid effects are warranted."<sup>44</sup><br/>         Overall, this study demonstrated that PFAA mixture could have the potential of multi-generational endocrine disruptors in <i>O. latipes</i> (Lee et al 2017).<sup>45</sup><br/>         "Notably, decreases in serum total thyroxine (T4) and 3,3',5-triiodothyronine (T3) levels were observed in fetal, pubertal, and adult PFBS-offspring in conjunction with slight increases in thyroid-stimulating hormone (TSH) and thyrotropin-releasing hormone levels. In addition, PFBS-dams exhibited significant decreases in total T4 and T3 levels and free T4 levels and increases in TSH levels, but no changes in E2 and P4 levels. These results indicate that prenatal PFBS exposure (&gt;= 200 mg/kg/day) causes permanent hypothyroxinemia accompanied by deficits in perinatal growth, pubertal onset, and reproductive organ development in female mice. (Feng 2017)"<sup>46</sup><br/>         "Gender-related differences were found; PFOS, PFHxS, PFBS, and PFOA levels were higher in males (p&lt;0.05), and the mean concentration of <math>\Sigma_8</math>PFASs was 1.5 times greater in males (6.02 ng/mL) than in females (4.15 ng/mL). PFOS and <math>\Sigma_8</math>PFASs were significantly negatively correlated with FT3 and FT4 and positively correlated with TSH while PFPeA and PFHxA were significantly positively correlated with TGAb and TMAb in all the samples."... PFBS was only detected in 27% of the samples, and was therefore not reported in the table of 'Pearson correlation coefficients between serum thyroid hormones and PFASs concentrations in different thyroid disease groups'. (Li et al 2017c)<sup>47</sup><br/>         Transcriptional effects of PFBS on thyroid related genes, <i>Pax 8</i> and <i>Hex</i> were studied (Naile et al 2012).<sup>48</sup></p> |
| <p style="text-align: center;"><i>Immunotoxicity</i></p>       | <p>"Corsini et al. (2012) made an <i>in vitro</i> characterization of the immunotoxic potential of several perfluorinated compounds, including PFBS. Cells of the human promyelocytic cell line THP-1 were incubated with PFBS (0.1-10 µg/mL) in the presence of lipopolysaccharide (LPS) or phytohemagglutinin (PHA) in order to examine the effects on the inflammatory cytokine response. PFBS inhibited the release of the tumor necrosis factor-α (TNF-α) and interleukin (IL) IL-10, but IL-6 and interferon-γ (IFN-γ) were unaffected. In THP-1 cells, PFBS also inhibited the protein NF-κB activation by inhibiting LPS-induced phosphorylation of P65, necessary for NF-κB transcription, and prevented I-κB kinase degradation. PPAR-α was not activated (Corsini et al., 2012)."<sup>49</sup></p>   |
| <p style="text-align: center;"><i>Other organ toxicity</i></p> | <p>"PFBS minimally inhibited 11β-HSD2 in human and rat kidney microsomes. The potency of inhibition declined with the length of the</p>   |

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|   | <p>carbon chain. PFBS has a four-carbon chain (Zhao et al., 2011).<sup>50</sup></p> <p>The liver toxicity and peroxisome proliferation potency of PFAS in rats increase with the carbon chain length until C9. PFBS is much less liver toxic than PFOS but large doses may damage the liver, kidneys and blood. The doses of PFBS required to produce similar increases in the enzyme hepatic acyl CoA oxidase activity (a measure of liver proliferation) was about 50 times higher than those of PFOS and PFHxS (Lau et al 2007). PFBS had also a relatively low PPAR<math>\alpha</math> activity in the liver (Wolf et al. 2008).<sup>51</sup></p>   |
| <b>Skin, Eye and Respiratory Effects</b>    |   |
| <i>Irritant – Skin, Eye, or Respiratory</i> | <p>Xn: R36 (eye irritation)(for Potassium PFBS under HSIS – Safe Work Australia)<sup>52</sup> “Not irritating to the skin, is irritating to the eye...”<sup>53</sup></p> <p>“Potassium PFBS was not irritating to rabbit skin when applied as a single dose of 500 mg for four hours under occlusive conditions. No signs of erythema or oedema were observed at 60 minutes or at 24, 48 and 72 hours (NICNAS, 2005).”<sup>54</sup></p> <p>“Potassium PFBS was found to be an irritant to rabbit eyes treated with 80 mg of the chemical for 24 hours. Corneal opacity, iritis, and redness and oedema of the conjunctivae were observed at 1, 24, 48 and 72 hours post treatment. By day 21, the scores had reversed in all but one animal (NICNAS, 2005).”<sup>55</sup></p> |
| <i>Corrosive – S, E, or R</i>               | H314 – Causes severe skin burns and eye damage. <sup>56</sup>   |
| <i>Permanent Damage – S, E, or R</i>        | Not found   |
| <i>Sensitizer– S &amp; R</i>                | <p>“Not a skin sensitizer”<sup>57</sup></p> <p>“Based on the negative results observed for potassium PFBS in a guinea pig maximisation test (GPMT) and for PFBSF in a local lymph node assay (LLNA), the chemicals in this group are not considered skin sensitisers; Potassium PFBS showed no evidence of skin sensitisation in a GPMT (NICNAS, 2005).”<sup>58</sup></p>   |
| <i>Asthmagen – Initiator or Exacerbator</i> | <p>Not found in AOEC database (as of 5/14/17)</p> <p>“In a study from Taiwan PFAS serum levels including of PFBS were reported to be significantly higher in children with asthma compared to children without asthma (Dong et al. 2013).”<sup>59</sup></p>   |
| <i>Skin Absorption, Kp</i>                  | Not found   |
| <i>LOAEL</i>                                | <p>Based on histopathological findings in the kidneys, the high dose of 600 mg/kg-day<sup>60</sup></p> <p>Based on kidney histopathology changes observed in F0- and F1-generation males and females, 300 mg/kg-day<sup>61</sup></p>  |
| <i>NOAEL</i>                                | <p>NOAEL for male rat hematological effects = 60 mg/kg/day; NOAEL for female rat = 600 mg/kg/day<sup>62</sup></p> <p>NOAEL for parental generations was 100 mg/kg bw/day in repro study (Lieder et al. 2009a).<sup>63</sup></p>   |

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|   | <p>“In a 90-day study, SD rats were treated with potassium PFBS via gavage at 60, 200 and 600 mg/kg bw/day. A no observed adverse effect level (NOAEL) of 200 mg/kg bw/day was established based on necrosis at the limiting ridge of the forestomach. Kidney hyperplasia was also reported at 600 mg/kg bw/day. However, an independent expert reported on the histopathology of the kidneys indicating that there were no consistent changes observed in the kidneys and the renal effects were not secondary to treatment (NICNAS, 2005).</p> <p>In a 28-day study with potassium PFBS in SD rats (0, 100, 300 and 900 mg/kg bw/day), a no observed effect level (NOEL) was established as 100 mg/kg bw/day in males based on significant decreases in serum phosphorus and potassium at doses of 300 and 900 mg/kg bw/day. A NOAEL was established as 300 mg/kg bw/day in females and males based on significant increase in kidney and liver weights, respectively, in animals that received 900 mg/kg bw/day. No histopathological findings were reported (NICNAS, 2005).”<sup>64</sup></p>   |
| <p><i>Benchmark Dose Response (BMD)</i></p> | <p>“Therefore, the BMDL<sub>10</sub> of 78.7 mg/kg-day based on increased incidence of kidney hyperplasia in females from the subchronic-duration study is selected as the point of departure (POD) for derivation of the subchronic p-RfD.”<sup>65</sup> See Pages 14-15 of the U.S. EPA 2014a document for additional details.</p>  |
| <p><i>Toxicokinetics</i></p>                | <p>“Based on the available data for potassium PFBS, the chemicals appear to be rapidly absorbed and excreted in animals (monkeys) by the kidneys with up to 87 % excreted in the urine within 24 hours post dosing. The chemical was shown to actively bind to human albumin protein in serum (94 % binding at 100 % physiological concentrations of albumin) with negligible binding to the other liver-manufactured proteins gamma globulin, alpha globulin, fibrinogen, alpha-2-macroglobulin, transferrin and beta lipoproteins. Apparent saturation of the binding above 100 ppm in rat, human and monkey serum was observed in this study (NICNAS, 2005). ... Bogdanska et al., (2014) determined the tissue distribution in male mice after a 1–5-day dietary exposure to environmentally relevant levels of 35S-labelled PFBS. PFBS was detected in all tissues examined; however, the tissue levels and tissue to blood ratios of PFBS were in all cases significantly lower than those of PFOS under similar circumstances. In most cases, tissue levels plateaued after 3 days.”<sup>66</sup></p> <p>In rats, the mean terminal serum PFBS elimination half-lives, after i.v. administration of 30 mg/kg PFBS, were: males 4.51±2.22 h (standard</p> |



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error) and females  $3.96 \pm 0.21$  h. In monkeys, the mean terminal serum PFBS elimination half-lives, after i.v. administration of 10 mg/kg PFBS, were: males  $95.2 \pm 27.1$  h and females  $83.2 \pm 41.9$  h. Although terminal serum half-lives in male and female rats were similar, without statistical significance, clearance (CL) was significantly greater in female rats ( $469 \pm 40$  mL/h) than male rats ( $119 \pm 34$  mL/h) with the area under the curve (AUC) significantly larger in male rats ( $294 \pm 77 \mu\text{g}\cdot\text{h}/\text{mL}$ ) than female rats ( $65 \pm 5 \mu\text{g}\cdot\text{h}/\text{mL}$ ). These differences were not observed in male and female monkeys. Volume of distribution estimates suggested distribution was primarily extracellular in both rats and monkeys, regardless of sex, and urine appeared to be a major route of elimination. Among 6 human subjects (5 male, 1 female) followed up to 180 days, the geometric mean serum elimination half-life for PFBS was 25.8 days (95% confidence interval 16.6–40.2). Urine was observed to be a pathway of elimination in the human. (Olsen 2009)<sup>67</sup>

“Chengelis et al. (2009b) compared the toxicokinetic behaviour of PFBS and PFHxA ... In rats, the terminal half-lives of PFBS were shorter for female rats (0.64 hours) than males (2.1 hours) after a single i.v. dose.”<sup>68</sup>

PFBS was identified in the serum of retired fluorochemical workers in the US. The mean serum elimination half-life was 27.7 days and ranged from 13.1 to 45.7 days. The main route of elimination was in the urine. The substance was also found in rat livers. (Olsen 2009)<sup>69</sup>

“In some workers with a lower than the animals but long-term exposure to PFBS as potassium salt, the mean serum elimination half-life of PFBS was determined to be 25.8 days in humans. However, that doesn’t mean that the substance is excreted, because analysis of Spanish human autopsy tissues revealed that the highest concentration of PFBS was found in lung tissues, however, PFBS also accumulated in liver, kidney and bone but not in brain (Perez et al. 2013).”<sup>70</sup>

“The mechanisms of toxicity may involve partitioning into lipid bilayers. PFBS disrupted different model phosphatidylcholine lipid assemblies indicating a potential for PFBS to alter cell membrane properties (Oldham et al. 2012). However, the effects of PFBS were not as pronounced as those seen with longer chain PFAS. Another toxicity mechanism may be an effect on lipid metabolism, As PFBS modestly reduced plasma triglycerides in a study with mice (Bijland et al. 2011).”<sup>71</sup>

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| <i>Synergistic or Antagonistic Effects</i>                     | “Additivity endpoint(s): Hematological (blood system), Hepatic (liver) system, Renal (kidney) system” <sup>72</sup>  |
| <b>Environmental and Human Health Exposure and Risk Values</b> |  |
| <i>RfC/RfD</i>   | Not found in the IRIS database (as of 5/05/17);<br>0.0042 mg/kg-d (laboratory animal), 0.42 mg/kg-d (human equivalent dose) <sup>73</sup><br>RfD = 0.02 mg/kg-day, oral, rat (Lieder et al. 2009b) <sup>74</sup><br>SRfD = 0.2 mg/kg-day, oral, rat (Lieder et al. 2009b) <sup>75</sup><br>“VTR = 0.08 mg/kg/day; Critical impact/effect: Renal effects (Tubular hyperplasia), Lieder et al 2009b (this is Lieder et al 2009a on the LG)” <sup>76</sup>  |
| <i>ATSDR-MRL</i>   | Not found on the ATSDR March 2016 List (as of 5/05/17)   |
| <i>Adverse Effect Levels: DNEL, PNEC, PNEL</i>                 | Not found  |
| <b>Health Based Exposure Limits</b>                            |  |
| <i>NIOSH-REL/IDLH/Ceiling Limits</i>                           | Not found in the NIOSH Pocket Guide  |
| <i>OSHA-PEL</i>  | Not found in the Z Tables  |
| <i>ACGIH TLV-TWA</i>   | Not found in RTECS (as of 5/05/17)   |
| <i>TLV-STEL</i>  | Not found in RTECS (as of 5/05/17)   |
| <i>Biomonitoring Action Limits</i>                             | Not found  |
| <i>Drinking Water Standards</i>                                | “Clean Water Act Requirements: Monitoring requirements for unregulated contaminants. (a) General applicability. This section specifies the monitoring and quality control requirements that must be followed if you own or operate a public water system (PWS) that is subject to the Unregulated Contaminant Monitoring Regulation (UCMR), as specified in paragraphs (a)(1) and (2) of this section. In addition, this section specifies the UCMR requirements for State and Tribal participation. For the purposes of this section, PWS "population served," "State," "PWS Official," "PWS Technical Contact," and "finished water" apply as defined in section 141.35(a). The determination of whether a PWS is required to monitor under this rule is based on the type of system (e.g., community water system, non-transient non-community water system, etc.), and its retail population, as indicated by SDWIS/Fed on December 31, 2010. ... Contaminant: perfluorobutanesulfonic acid; CAS Registry No.: <b>375-73-5</b> ; Analytical Method: EPA 537; Minimum Reporting Level: 0.09 ug/L; Sampling location: EPTDS (entry points to the distribution system); Period during which monitoring to be completed: 1/1/2013-12/31/2015.” <sup>77</sup> |
| <i>Other</i>   | Subchronic Noncancer Health Risk Limit = 9 ug/L <sup>78</sup><br>Chronic Noncancer Health Risk Limit = 7 ug/L <sup>79</sup>  |
| <b>ENVIRONMENTAL &amp; ECO-SYSTEM HAZARDS</b>                  |  |
| <b>Persistence</b>   | “PFBS is persistent” <sup>80</sup>   |

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|                               | <p>“Concerns about PFBS and other PFCs stem from the resistance of these compounds to hydrolysis, photolysis, and biodegradation, which leads to their persistence in the environment (Sundstrom et al., 2012).”<sup>81</sup></p> <p>Quinete et al. (2010) studied the degradability of some PFBS in two traditional OECD ready biodegradability screening tests, the manometric respirometry test (OECD 301F) and the closed-bottle test (OECD 301D) with River Rhine water as inoculum. PFBS did not show any significant biodegradation over the 28 day duration of the tests.<sup>82</sup></p>   |
| <p><i>Bioaccumulation</i></p> | <p>“It is also mentioned by Lassen et al. (2013) that the bioaccumulation of PFOS and other PFAS is higher in the marine environment than in soil. These findings are believed to be valid also for the short-chain perfluorinated carboxylic and sulfonic acids and their salts. According to a number of reports (e.g. Ellis et al. (2004), Butt et al. (2010), Martin et al. (2013)), the acids are not very bio-accumulative in themselves but precursors such as fluorotelomer alcohols and acrylates accumulate and are subsequently transformed in the organs of animals to the corresponding acids, which are retained in the body.”<sup>83</sup> <a href="#">Checked against Danish EPA references; that statement is supported by Butt 2010 fish study and Hagen 1981 rat study, and transformation in the atmosphere is supported by Ellis 2004.</a></p> <p>“PFBS is not bioaccumulative or toxic to aquatic organisms (NICNAS 2005, <a href="#">Giesy et al. 2010</a>)”.<sup>84</sup> <a href="#">Checked against Dewitt references: NICNAS 2005 references a 3M unpublished 2001 study by Wildlife International: flow through bioconcentration test with bluegill sunfish; results: tissue concentration appeared to reach steady state between day 3 and 7; steady-state BCF ranged from 0.113 – 0.43. PFBS eliminated during depuration phase, estimated half-life 1.3-2.9 days. Giesy 2010 references six 3M unpublished 2001 studies by Wildlife International with results of LC<sub>50</sub> &gt;1,000 mg/L and NOEC &gt;700 mg/L for bluegill and other aquatic species (see aquatic toxicity below).</a></p> <p><a href="#">BSAFs measured based on wet weight of organisms of PFASs in earthworms at 3 exposure concentrations; PFBS BSAF: 0.021 ± 0.004 [at 100 ng/g]; 0.011 ± 0.000 [at 200 ng/g]; 0.008 ± 0.000 [at 500 ng/g].</a></p> <p><a href="#">“According to our study, PFHxA, PFHpA, PFOA, PFBS and PFHxS, which have seven or less fluorinated carbons, displayed distinct bioaccumulative ability. This could be due to the active ingestion of soil through the gut and the high protein content of the earthworms.”<sup>85</sup></a></p> |

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### Presence in biota and the environment:

FBSA (perfluoro-1-butane-sulfonamide) is a major metabolite in post 2002 Scotchgard fabric protector and precursor to PFBS (sulfonamide substitutes NH<sub>2</sub> for OH, the sulfonamide breaks down to form the sulfonate). FBSA was quantifiable in the fish samples and is more than likely the penultimate precursor of PFBS. While nearly all fish samples were < MLOD for PFBS, FBSA was detected in 32 of 33 samples of remote Canadian freshwater fish (2009-10) with concentrations ranging from <0.01 ng/g w.w. (detection limit) to 0.44 ng/g w.w. (vs. industrialized Great Lakes sites with 3.17 ng/g w.w.)

PFBS was detected in marine fish: flounder muscle (Netherlands) 80.12 ng/g w.w. (exceeded only by PFOS)<sup>86</sup>

In a study in Spain FBSA was not found in gull eggs<sup>87</sup> and PFBS was not found in herring gull eggs in a Great Lakes study.<sup>88</sup>

Llorca et al (2009) found two short chain PFASs, PFPeA and PFBS, in samples of whole fish (striped mullet, anchovies and young hake), swordfish fillets and hake roe taken from fish markets in Spain.<sup>89</sup>

Chinese study downstream of Beijing Airport: The highest surface water concentrations were observed for PFBS (2.90–51.3 ng/L).<sup>90</sup>

In addition, significantly increasing temporal shifting trends of PFOS to PFBS were observed in the dolphin liver samples (Lam et al, 2016).<sup>91</sup>

Significantly higher concentrations of perfluorobutane sulfonic acid (PFBS) and perfluorohexane sulfonic acid (PFHxS) were found among women who lived in districts modeled to have received contaminated drinking water compared to unaffected districts both in 1996-1999 and 2008-2011, indicating that the contamination was already present in the late 1990s (Gyllenhammar et al, 2015).<sup>92</sup>

In Uppsala County, Sweden, temporal trends of PFAAs and FOSA in pooled samples of blood serum from nursing women were sampled between 1996 and 2010. Levels of PFBS increased over the time period (0.014 ng/g 1996 to 0.101 ng/g in 2010) (Glynn 2012; Glynn 2012a).<sup>93</sup>

“Total fluorine (TF), extractable organic fluorine (EOF) and poly- and per-fluorinated compounds (PFCs) were measured in eight dated cores of sediment taken along with 27 surface sediments from Lake Michigan in 2010. Based on rates of sedimentation, total concentrations of PFCs ( $\Sigma$ PFCs) reached a maximum in the later 1990s and early 2000s. This result is consistent with rapid changes in production and subsequent sedimentation. Perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) are still the predominant PFCs in the cores, but in surface sediments, concentrations of perfluorobutane sulfonate (PFBS) and perfluorobutanoic acid (PFBA) are now occurring at concentrations comparable to those of PFOS and PFOA. This

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|  | <p>observation is consistent with shifts in patterns of production and use in the US and Canada. Concentrations of TF in sediments were greater than those of EOF. This result is consistent with a larger proportion of un-extractable fluorinated material in both surface sediments and in cores” (Codling 2014).<sup>94</sup></p>   |
| <i>BAF, BCF, BMF</i>   | <p>In a subtropical food web study, PFBS was a dominant PFC in tidal shrimp pond water at ~6.8 ng/L, but was not detected in any of the biota samples, indicating that PFBS is not bioaccumulative in this system (Loi 2011).<sup>95</sup></p> <p>Bioaccumulation factors (BAFs) of the aquatic plants indicated the absorption of PFASs were effective. BAFs in submerged plants basically increased with increasing chain length accordingly (Zhou et al, 2017a).<sup>96</sup></p>  |
| <i>Aquatic Toxicity: LC<sub>50</sub>, EC<sub>50</sub>, ErC<sub>50</sub>, NOAEC/NOEC</i>  | <p>Bluegill, 96 hr LC<sub>50</sub> = 6,452 mg/L, NOEC = 6,452 mg/L (<i>TURI note: LC<sub>50</sub> = NOEC? Don't have access to original Wildlife Int'l study</i>);<br/>         Fathead minnow, 96 hr LC<sub>50</sub> = 1,938 mg/L, NOEC = 888 mg/L;<br/> <i>Daphnia magna</i>, 48 hr LC<sub>50</sub> = 2,183 mg/L, NOEC = 886 mg/L, 21-day study, chronic NOEC = 707 mg/L.<sup>97</sup></p> <p>“These data indicate that PFBS is not very toxic to freshwater organisms, with effect levels only being observed at a concentration greater than 700 mg/L”.<sup>98</sup></p> <p>Zebrafish larvae (<i>Danio rerio</i>), 144 hr, EC<sub>50</sub>, embryotoxicity = 450 mg/L<sup>99</sup></p> <p>“Only a single review citing results on the sulfonate PFBS-K was identified (NICNAS, 2005). The short chain PFAS showed to be practically non-toxic to algae with effects concentrations &gt; 1,000 mg/L.”<sup>100</sup></p>  |
| <i>Wildlife Toxicity: LC<sub>50</sub>, EC<sub>50</sub>, ErC<sub>50</sub>, NOAEC/NOEC</i> | <p>“PFBS acute and chronic toxicity studies in Mallard and Northern Bobwhite Quail have been reported (Newsted et al. 2008). In acute dietary studies with juvenile mallards and northern bobwhite quail, 10-day-old mallards and quail were exposed to 1,000, 1,780, 3,160, 5,620 or 10,000 mg/kg feed, wet weight (ww) for 5 days and the birds were then fed an untreated diet and observed for up to 17 days. No treatment-related mortalities were observed in the study up to 10,000 mg/kg, ww feed. Body weight gains of quail exposed to 5,620 or 10,000 mg/kg feed were statistically less than that of unexposed controls. Weight gain of mallards exposed to 10,000 mg PFBS/kg feed was statistically less than that of controls. There were no statistically significant effects on feed consumption of either species. The no observed adverse effect concentration (NOAEC) for mallards and quail were 5,620 and 3,160 mg/kg, ww feed, respectively. In a reproduction study, adult quail were exposed to nominal dietary concentrations of</p> |

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|   | <p>100, 300, or 900 mg/kg, ww feed for up to 21 weeks. There were no treatment-related mortalities or effects on body weight, weight gain, feed consumption, histopathology measures, or reproductive parameters evaluated in the study when compared to the control group. The dietary NOAEC was 900 mg/kg, ww feed.”<sup>101</sup></p> <p>Using available avian studies showing no biomagnification, <b>Giesy et al 2010</b> assumed BAF/BMF of 1.0, and calculated a Water Quality Criteria for protection of aquatic predatory birds Avian Wildlife Value (AWV) according to Great Lakes Initiative guidance methods of 17 mg/L PFBS (geometric mean of 3 values ranging from 13-24). Value is lower than 47 mg/L AWV for PFOS.<sup>102</sup></p>   |
| <i>Breakdown/degradation /combustion products</i>     | <p>“Perfluorinated acids are not biodegradable neither under aerobic nor under aerobic environmental conditions in water or soil (Lassen et al., 2013).”<sup>103</sup></p> <p>“According to NICNAS (2005), no biodegradation of potassium perfluorbutane sulfonate (PFBS-K) is expected.”<sup>104</sup></p>   |
| <i>Anaerobic degradation</i>                          | <p>“The rate constant for the vapor-phase reaction of perfluorobutanesulfonic acid with photochemically-produced hydroxyl radicals has been estimated as <math>1.4 \times 10^{-13}</math> cu cm/molecule-sec at 25 deg C(SRC) using a structure estimation method(1). This corresponds to an atmospheric half-life of about 115 days at an atmospheric concentration of <math>5 \times 10^{+5}</math> hydroxyl radicals per cu cm(1). Perfluorobutanesulfonic acid is not expected to undergo hydrolysis in the environment due to the lack of functional groups that hydrolyze under environmental conditions (2). Perfluorobutanesulfonic acid does not contain chromophores that absorb at wavelengths &gt;290 nm(2) and, therefore, is not expected to be susceptible to direct photolysis by sunlight(SRC).”<sup>105</sup></p> |
| <i>Aerobic degradation</i>                            | <p>“Using a closed-bottle test (OECD 301D), an inoculum from the Rhine River and incubated at 20 deg C for 28 days in the dark perfluorobutanesulfonic acid starting at 73 mg/L was biodegraded &lt;3%. Using a manometric respirometry test (OECD 301F) with activated sludge, 100 mg/L perfluorobutanesulfonic acid was biodegraded &lt;1% in 40 days. In a fixed bed bioreactor test using Rhine River inoculum, perfluorobutanesulfonic acid did not biodegradable in the 28 day test run at room temperature in the dark(1).”<sup>106</sup></p>  |
| <i>Other observable ecological effects (e.g. BOD)</i> | <p>The composition of PFASs in water changed with time, perfluorobutane sulfonate (PFBS) was the predominant compound in spring and summer, while long-chain PFASs, perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS), started to increase in autumn and winter. (<b>Zhou et al, 2017a</b>).<sup>107</sup></p> <p>The observed dose-response PFAS-induced effects were to some extent</p>  |

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|  | related to their cytotoxicity: the EC <sub>50</sub> -values of most influential PFAS-treatments increased (PFOS < PFNA < PFOA << PFBS), and higher-doses of these chemicals induced a larger impact. Major spectral alterations were mainly attributed to DNA/RNA, secondary protein structure, lipids, and fatty acids. Finally, PFOS and PFOA caused a decrease in A6 cell numbers compared to controls, whereas PFBS and PFNA did not significantly change cell population levels. Overall, this work highlights the ability of PFASs to alter A6 cells, whether forming monolayers or differentiated into dome structures, and the potential of PFOS and PFOA to induce cell death (Gorochategui et al 2016). <sup>108</sup>   |
| <i>Fate and Transport: Aquatic</i>       | (See atmospheric below)  |
| <i>Fate and Transport: Terrestrial</i>   | “Short-chain PFAAs are generally more mobile than their long-chain homologues; they are not retarded in soil and have already been detected in groundwater”. <sup>109</sup>  |
| <i>Fate and Transport: Atmospheric</i>   | <p>“The ionic PFASs such as the carboxylic and sulfonic acids (PFCAs and PFSA) are considered to undergo long-range transport mainly via the aquatic environment (Ahrens, 2011), not least via the oceanic currents while atmospheric long-transport have been postulated to involve mainly the neutral PFASs such as the precursors known as fluorotelomer alcohols (FTOHs). These have properties ensuring sufficient residence time in the atmosphere for long-range transport while at the same time being sufficiently reactive to be transformed by various oxidation reactions into the corresponding carboxylic acids and/or other products including other fluorotelomer species (Ellis et al., 2004)”.<sup>110</sup></p> <p>The global distribution and long-range transport of PFASs were investigated using seawater samples collected from the Greenland Sea, East Atlantic Ocean and the Southern Ocean in 2009-2010. PFBS was one of five most frequently detected PFAS, with concentrations ranging from &lt;1.6 to 45 pg/L (Zhao 2012).<sup>111</sup></p> |
| <i>Transport Issues</i>                  | “The long-term fate of these substances is transport to deep ocean water and/or sediment burial”. <sup>112</sup>   |
| <i>Factors affecting bioavailability</i> |  |
| <b>Global Environmental Impacts</b>      |  |
| <i>Ozone Depletion Potential (ODP)</i>   | Not found  |
| <i>Global Climate Change</i>             | Not found  |
| <i>Greenhouse Gas Production</i>         | Not found  |
| <i>Acid Rain Formation</i>               | Sulfur oxides are combustion products  |
| <b>Special Reports</b>                   |  |
| <i>EU</i>                                | <b>Human Health Tier II Assessment for Perfluorobutanesulfonate (PFBS) and its direct precursors, NICNAS,</b>  |

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[https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment\\_id=1429](https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1429)  
**Environment Tier II Assessment for Perfluorobutanesulfonic Acid and its Direct Precursors**, NICNAS, updated April 26, 2017,  
<https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/perfluorobutanesulfonic-acid-and-its-direct-precursors>  
**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>  
**Perfluoroalkylated substances: PFOA, PFOS and PFOSA** - Evaluation of health hazards and proposal of a health based quality criterion for drinking water, soil and ground water, Environmental project No. 1665, 2015  
<http://www2.mst.dk/Udgiv/publications/2015/04/978-87-93283-01-5.pdf>

Notes on chemical research: Not found in NIOSH Pocket Guide

### **ToxPlanet folders available:**

ATSDR; Australian Gov't – NICNAS; CDC; CTD – Comparative Toxicogenomics Database  
Danish EPA – Publications; EFSA – European Food Safety Authority; EPA; HSDB  
ITER – International Toxicity Estimates for Risk Assessment  
MAK Collection for Occupational Health & Safety; Minnesota Department of Health;  
NTP; PubChem; RTECS; TEDX

<sup>1</sup> [www.expub.com](http://www.expub.com); Chemical Identity Page for Perfluorobutanesulfonic acid.

<sup>2</sup> [www.expub.com](http://www.expub.com); RTECS for Perfluorobutane sulfonic acid.

<sup>3</sup> [www.expub.com](http://www.expub.com); Chemical Identity Page for Perfluorobutane sulfonic acid.

<sup>4</sup> U.S. National Library of Medicine, ChemIDplus, a Toxnet Database, entry for “Perfluorobutanesulfonic acid.”, accessed online at: <https://chem.nlm.nih.gov/chemidplus/rn/375-73-5>

<sup>5</sup> U.S. National Library of Medicine, ChemIDplus, a Toxnet Database, entry for “Perfluorobutanesulfonic acid.”, accessed online at: <https://chem.nlm.nih.gov/chemidplus/rn/375-73-5>

<sup>6</sup> **DeWitt 2015:** DeWitt, Jamie C. *Toxicological Effects of Perfluoroalkyl and Polyfluoroalkyl Substances*. Humana Press; 2015 edition (April 14, 2015).



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<sup>7</sup> **U.S. EPA 2014a:** U.S. Environmental Protection Agency. Provisional Peer-Reviewed Toxicity Values for Perfluorobutane Sulfonate (CASRN 375-73-5) and Related Compound Potassium Perfluorobutane Sulfonate (CASRN 29420-49-3). July 17, 2014. Accessed online at: <https://hhpprt.v.ornl.gov/>. Page 2.

<sup>8</sup> **HSDB 2017:** U.S. National Library of Medicine, Hazardous Substances Data Bank (HSDB), a Toxnet Database, entry for “Perfluorobutanesulfonic acid, CAS # 375-73-5.”, accessed online at: <https://toxnet.nlm.nih.gov/cgi-bin/sis/search2>. [Kosswig K; Sulfonic Acids, Aliphatic. Ullmann's Encyclopedia of Industrial Chemistry 7th ed. (1999-2015). NY, NY: John Wiley & Sons. Online Posting Date: June 15, 2000] **\*\*PEER REVIEWED\*\***

<sup>9</sup> **Norwegian EA 2017:** Norwegian Environment Agency. Investigation of Sources to PFBS in the Environment. Report Number M-759|2017. May 15, 2017. Accessed online at: <http://www.miljodirektoratet.no/Documents/publikasjoner/M759/M759.pdf>.

<sup>10</sup> HSDB 2017: [Sigma-Aldrich; Product Search Nonfluorobutane-1-sulfonic acid. (375-73-5). Available from, as of Dec 21, 2015: <http://www.sigmaaldrich.com/united-states.html>] **\*\*PEER REVIEWED\*\***

<sup>11</sup> **NICNAS 2017a:** Australian Government, Department of Health, National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Environment Tier II Assessment for Perfluorobutanesulfonic Acid and its Direct Precursors, Last update April 26, 2017. Accessed online at: <https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-ii-environment-assessments/perfluorobutanesulfonic-acid-and-its-direct-precursors>.

<sup>12</sup> **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. Page 49. Accessed online at: <http://www2.mst.dk/Udgiv/publications/2015/05/978-87-93352-15-5.pdf>

<sup>13</sup> U.S. EPA 2014a. Page 2.

<sup>14</sup> NICNAS 2017a.

<sup>15</sup> Danish EPA 2015b: Page 49.

<sup>16</sup> Dewitt 2015: Page 473.

<sup>17</sup> U.S. EPA 2014a. Page 2.

<sup>18</sup> NICNAS 2017a.

<sup>19</sup> HSDB 2017: [Sigma-Aldrich; Product Search Nonfluorobutane-1-sulfonic acid. (375-73-5). Available from, as of Dec 21, 2015: <http://www.sigmaaldrich.com/united-states.html>] **\*\*PEER REVIEWED\*\***

<sup>20</sup> Danish EPA 2015b: Page 49.

<sup>21</sup> U.S. EPA 2014a. Page 2.

<sup>22</sup> NICNAS 2017a.

<sup>23</sup> HSDB 2017: [Sigma-Aldrich; Safety Data Sheet for Nonfluorobutane-1-sulfonic acid. Product Number: 562629, Version 4.2 (Revision Date 09/08/2015). Available from, as of December 15, 2015: <http://www.sigmaaldrich.com/safety-center.html>] **\*\*PEER REVIEWED\*\***

<sup>24</sup> U.S. EPA 2014a. Page 2.

<sup>25</sup> HSDB 2017: [Sigma-Aldrich; Safety Data Sheet for Nonfluorobutane-1-sulfonic acid. Product Number: 562629, Version 4.2 (Revision Date 09/08/2015). Available from, as of December 15, 2015: <http://www.sigmaaldrich.com/safety-center.html>] **\*\*PEER REVIEWED\*\***

<sup>26</sup> **NICNAS 2017b:** Australian Government, Department of Health, National Industrial Chemicals Notification and Assessment Scheme (NICNAS). HUMAN HEALTH TIER II ASSESSMENT FOR Perfluorobutanesulfonate (PFBS) and its direct precursors. Accessed online at: [https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment\\_id=1429#cas-A\\_375-73-5](https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1429#cas-A_375-73-5).

<sup>27</sup> NICNAS 2017b.

<sup>28</sup> NICNAS 2017b.

<sup>29</sup> **MN DPH 2011:** Minnesota Department of Health. 2011 Health Risk Limit for Groundwater, Perfluorobutane sulfonate. Web Publication Date: March 21, 2011. Accessed online at: <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfbs.pdf>. Page 3.

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<sup>30</sup> MN DPH 2011. Page 1.

<sup>31</sup> **Slotkin 2008:** Slotkin, TA, et al. Developmental Neurotoxicity of Perfluorinated Chemicals Modeled in Vitro. *Environmental Health Perspectives* **116**:716-722 (2008).

<sup>32</sup> Dewitt 2015: Pages 473-474.

<sup>33</sup> MN DPH 2011. Page 3.

<sup>34</sup> MN DPH 2011. Page 3.

<sup>35</sup> **EFSA 2014:** Ricardo-AEA Ltd. For European Food Safety Authority (EFSA). Extensive literature search and provision of summaries of studies related to the oral toxicity of perfluoroalkylated substances (PFASs), their precursors and potential replacements in experimental animals and humans – Area 1: Data on toxicokinetics (absorption, distribution, metabolism, excretion) in in vitro studies, experimental animals and humans; Area 2: Data on toxicity in experimental animals; Area 3: Data on observations in humans. EFSA supporting publication 2014:EN-572. Accessed online: <http://onlinelibrary.wiley.com/doi/10.2903/sp.efsa.2014.EN-572/abstract>. Page 73.

<sup>36</sup> Danish EPA 2015b: Page 31.

<sup>37</sup> **Wang 2017:** Wang B, et al. Perfluoroalkyl substances and endometriosis-related infertility in Chinese women. *Environ Int.* 2017 May; **102**:207-212.

<sup>38</sup> **Feng 2017:** Feng X, et al. Exposure of Pregnant Mice to Perfluorobutanesulfonate Causes Hypothyroxinemia and Developmental Abnormalities in Female Offspring. *Toxicological Sciences*, **155(2)**, 2017, 409-419.

<sup>39</sup> EFSA 2014. Page 75.

<sup>40</sup> NICNAS 2017b.

<sup>41</sup> TEDX: Search for 375-73-5, Accessed online, 2/7/17, <http://www.endocrinedisruption.org/endocrine-disruption/tedx-list-of-potential-endocrine-disruptors/chemicalsearch?sname=&x=0&y=0&action=search&sall=1&searchfor=any&scas=375-73-5&searchcats=all>

<sup>42</sup> Danish EPA 2015b: Page 29.

<sup>43</sup> **CA 2015:** CA Scientific Guidance Panel Biomonitoring California. Mar 13, 2015. Potential Designated Chemicals: Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs) (p.11 referencing Nilsson et al 2013). Accessed online at: [http://www.biomonitoring.ca.gov/sites/default/files/downloads/PotenDesigPFASs\\_031315.pdf](http://www.biomonitoring.ca.gov/sites/default/files/downloads/PotenDesigPFASs_031315.pdf).

<sup>44</sup> MN DPH 2011. Page 1.

<sup>45</sup> **Lee 2017:** Lee JW, et al. Multi-generational xenoestrogenic effects of Perfluoroalkyl acids (PFAAs) mixture on *Oryzias latipes* using a flow-through exposure system. *Chemosphere*. 2017 Feb; **169**:212-223.

<sup>46</sup> **Feng 2017:** Feng X, et al. Exposure of Pregnant Mice to Perfluorobutanesulfonate Causes Hypothyroxinemia and Developmental Abnormalities in Female Offspring. *Toxicological Sciences*, **155(2)**, 2017, 409-419.

<sup>47</sup> **Li 2017c:** Li Y, et al. Perfluorinated alkyl substances in serum of the southern Chinese general population and potential impact on thyroid hormones. *Scientific Reports* **7**, 43380 (2017).

<sup>48</sup> **Naile 2012:** Naile JE, et al. Transcriptional effects of perfluorinated compounds in rat hepatoma cells. *Chemosphere* **86** (2012) 270-277.

<sup>49</sup> Danish EPA 2015b: Page 32.

<sup>50</sup> EFSA 2014. Page 73.

<sup>51</sup> Danish EPA 2015b: Page 31.

<sup>52</sup> NICNAS 2017b.

<sup>53</sup> Dewitt 2015: Page 473.

<sup>54</sup> NICNAS 2017b.

<sup>55</sup> NICNAS 2017b.

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