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Breaking It Down: Estimating Short-Chain PFAS Half-Lives in a Human Population

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Many studies have examined the impact of long-chain per- and polyfluoroalkyl substances (PFAS) on human health,[1] but few have looked at short-chain PFAS.[2] A recent study in *Environmental Health Perspectives* estimated half-lives of both long- and short-chain PFAS in a small group of people immediately after exposure to PFAS-contaminated drinking water ended.[3]

The terms "long" and "short" refer to the length of the carbon (C) backbone of each molecule; long-chain is defined as perfluoroalkyl sulfonic acids with 6 or more C atoms, and perfluoroalkyl carboxylic acids with 7 or more C atoms.[4] Most long-chain PFAS have been phased out and replaced by short-chain varieties. Thus, long-chain varieties are sometimes called legacy PFAS.

It is generally believed that the shorter the chain, the shorter the half-life for PFAS.[2] However, this rapid excretion also makes it difficult to assess the health impacts of short-chain varieties because studies may start weeks or months after exposure has ceased.

"For an observational study of half-life in humans, it is crucial to find a proper population with a defined end of external exposure," says lead author Yiyi Xu, a research associate at the University of Gothenburg in Sweden. "Due to faster elimination of short-chain PFAS, researchers need to start the observation as soon as the external exposure stops, to capture reasonable serum levels for half-life estimation." Only a handful of previous studies have quantified the half-lives of short-chain PFAS in human populations.[5,6,7,8]

The new study took place at Sweden's Arvidsjaur regional airport, which has its own drinking water supply, separate from the municipality of Arvidsjaur. The airport water source was contaminated with PFAS, presumably from aqueous film-forming foam used at an on-site fire drill area. Some firefighting foams contain fluorinated surfactants and are a known source of both long- and short-chain PFAS pollution.[9]

When authorities discovered the water contamination at the airport in August 2018, they immediately provided clean water to employees and then contacted the authors of the new paper to quantify exposure levels. The researchers sampled serum and urine for 17 airport employees starting 11–14 days after exposure ended. The municipal supply had very low levels of PFAS, which enabled

The same chemistry that makes PFAS so persistent in the environment also enables them to quickly extinguish fires. Firefighting foam spreads and forms a film over the fire, smothering the flames and cooling the fuel. Image: © Dushlik/Shutterstock.

the researchers to confirm that employees had no substantial exposures at home after August 2018. They used a reference population[10] from Karlshamn, a city with uncontaminated water, as a basis of comparison.

"The exposure scenario was unique in that a significant exposure source existed, and then the exposure abruptly ceased. This allowed refinement of existing half-life determinations to be more accurate," says Dana Boyd Barr, a research professor of exposure science and environmental health at Emory University who was not involved in the study. "Previous human half-life determinations haven't had this clean demarcation between exposure and exposure cessation, thus introducing more error into the calculation."

Over a period of 5 months, the team collected 4–5 blood samples and 1–4 urine samples from each participating airport employee. Long-chain PFAS accounted for 90% of total serum PFAS and 50% of the contamination in water, suggesting more bioaccumulation in the employees' bodies. At initial sampling, the highest serum PFAS was found for perfluorohexane sulfonic acid (PFHxS, C6), with concentrations 102–225 times higher than in the Karlshamn reference population. Short-chain perfluoropentane sulfonic acid (PFPeS, C5) was 175–380 times higher than in the reference population.

All serum PFAS concentrations declined over time. As expected, the half-lives of long-chain PFAS were longer than those of short-chain compounds, reaching almost 3 years in some cases. For example, the researchers estimated the average half-lives for PFHxS at 2.86 years and L-PFOS (a specific form of perfluorooctane sulfonic acid, C8) at 2.91 years. In comparison, the short-chain PFPeS had an estimated average half-life of 0.63 years, and perfluoroheptane sulfonic acid (PFHpS, C7) concentrations were 1.46 years. The shortest estimated half-lives were for perfluorobutanoic acid (PFBA, C4) at 0.12 years and perfluoroheptanoic acid (PFHpA, C7) at 0.17 years.

The investigators noted that the estimated half-lives of PFOA and PFOS were shorter than previously published estimates.[10] They also found that some isomers of PFOS had shorter half-lives than others. One possible explanation is that elimination may slow down over time once exposure ceases—a phenomenon that would be uniquely captured in this study, which started almost immediately after exposure stopped.

"The findings and toxicokinetic estimates for the PFAS analyzed were in good agreement with previous analysis and lend support for the very long biological half-lives defined for most PFAS," says Scott Belcher, a research professor of toxicology at North Carolina State University who was not involved in the study. "It is important to recognize that even the short-chain PFAS are eliminated slowly, on the order of many weeks."

Belcher adds, "The slow rates of elimination for shorter-chained PFAS, which have toxicity profiles similar to long-chain PFAS, are concerning because of the increased use of short-chain PFAS as replacements for many applications."

Xu agrees. "Although short-chain PFAS are less bioaccumulative, these PFAS with the same structure of fluorine-carrying carbon chains will not degrade easily," she says. "Therefore, high short-chain PFAS contamination of drinking water is a serious environmental health problem that should be taken into account in the future."

Wendee Nicole is an award-winning science writer and editor based in Houston, Texas. She has written for *Discover*, *Nature*, *Scientific American*, and other publications.

References

1. EFSA (European Food Safety Authority). 2018. Risk to human health related to the presence of perfluorooctane sulfonic acid and perfluorooctanoic acid in food. EFSA J16 (1):e05194, PMID: 32625773, https://doi.org/10.2903/j.efsa.2018.5194.
2. Han X, Nabb DL, Russell MH, Kennedy GL, Rickard RW. 2012. Renal elimination of perfluorocarboxylates (PFCAs). Chem Res Toxicol 25(1):35–46, PMID: 21985250, https://doi.org/10.1021/tx200363w.
3. Xu Y, Fletcher T, Pineda D, Lindh CH, Nilsson C, Glynn A, et al. 2020. Serum half-lives for short- and long-chain perfluoroalkyl acids after ceasing exposure from drinking water contaminated by firefighting foam. Environ Health Perspect 128(7):77004, PMID: 32648786, https://doi.org/10.1289/EHP6785.
4. Buck RC, Franklin J, Berger U, Conder JM, Cousins IT, de Voogt P, et al. 2011. Perfluoroalkyl and polyfluoroalkyl substances in the environment: terminology, classification, and origins. Integr Environ Assess Manag 7(4):513–541, PMID: 21793199, https://doi.org/10.1002/ieam.258.
5. Conder JM, Hoke RA, de Wolf W, Russell MH, Buck RC. 2008. Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds. Environ Sci Technol 42(4):995–1003, PMID: 18351063, https://doi.org/10.1021/es070895g.
6. Olsen GW, Chang S-C, Noker PE, Gorman GS, Ehresman DJ, Lieder PH, et al. 2009. A comparison of the pharmacokinetics of perfluorobutanesulfonate (PFBS) in rats, monkeys, and humans. Toxicology 256(1–2):65–74, PMID: 19059455, https://doi.org/10.1016/j.tox.2008.11.008.
7. Russell MH, Nilsson H, Buck RC. 2013. Elimination kinetics of perfluorohexanoic acid in humans and comparison with mouse, rat and monkey. Chemosphere 93(10):2419–2425, PMID: 24050716, https://doi.org/10.1016/j.chemosphere.2013.08.060.
8. Zhang Y, Beesoon S, Zhu L, Martin JW. 2013. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environ Sci Technol 47(18):10619–10627, PMID: 23980546, https://doi.org/10.1021/es401905e.
9. Houtz EF, Higgins CP, Field JA, Sedlak DL. 2013. Persistence of perfluoroalkyl acid precursors in AFFF-impacted groundwater and soil. Environ Sci Technol 47(15):8187–8195, PMID: 23886337, https://doi.org/10.1021/es4018877.
10. Li Y, Fletcher T, Mucs D, Scott K, Lindh CH, Tallving P, et al. 2018. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Occup Environ Med 75(1):46–51, PMID: 29133598, https://doi.org/10.1136/oemed-2017-104651.