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Commentary

PFAS Exposure, Reproductive Impacts, Cancer, and other Health Effects: No Longer an Emergent Threat

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DESCRIPTION

Our study found that southern NH residents exposed to industrial per and Polyfluoroalkyl Substances (PFAS) air emissions experience significantly higher rates of thyroid, prostate, and bladder cancers. Additionally, the cancer incidences are strikingly similar to that of the residents of Bennington, Vermont, another region with documented high PFAS exposure from industrial emissions [1]. Residents in both towns have been exposed to decades of industrial PFAS air emissions and PFAS-contaminated drinking water.

PFAS is an umbrella term for more than 12,000 or possibly as many as almost 21 million man-made chemicals that contain at least one Carbon-Fluorine (C-F) bond [2]. The strength of the C-F bond makes PFAS persistent, bioaccumulative, and toxic and these properties are imparted to the emissions produced during manufacturing and are conveyed to the end products produced [3].

While PFAS have been used in everything from Teflon™ pans and rain jackets, to Scotchgard™ and fast-food processing and packaging since the 1950's, the public and scientific community have only learned about them in the last decade [4]. In the meantime, the public has been unknowingly exposed to PFAS by ingesting contaminated drinking water and eating food that has been in contact with PFAS-coated food packaging or fast-food grill sheets. Perfluorooctanoic Acid (PFOA), one of the PFAS compounds, and other PFAS have been detected in 98% of serum samples from humans over age 12 in the US. Our ability to identify PFAS in environmental media and serum has been hampered by industry non-disclosure and intellectual property claims [5].

However, robust epidemiological evidence supports the association between PFOA exposure and cancer [6]. Studies of health outcomes in 69,000 people exposed to PFOA from DuPont's Washington Works plant in West Virginia concluded that PFOA exposure was "more probably than not" associated with testicular and kidney and renal pelvis cancers, ulcerative colitis, thyroid disease, hypercholesterolemia, and pregnancy-induced hypertension [7].

Previous research also indicates that PFOA exposure is also associated with female breast cancer, low birth weight, immune disruption, and cardiovascular impacts. In addition, but not limited to, associations have been found between PFAs exposure and longer time to pregnancy, increased risk of miscarriage, and reduced vaccine response [8]. PFAs exposure has also been associated with an increased risk of other cancers including melanoma, ovarian cancer, and non-Hodgkin lymphoma.

In addition to cancers and chronic disease, PFAS exposure disrupts male hormonal pathways potentially leading to male infertility [9]. Several authors report that in human in vitro studies PFOA, Perfluorononanoic acid (PFNA), Perfluorodecanoic acid (PFDA), Perfluorooctanesulfonic acid PFOS and Perfluorohexane Sulfonate (PFHxS) interfere with estrogen receptor signaling.

Historically, industry-sponsored studies found exceptionally high serum PFAS levels and reproductive hormone disruptions in male plant workers [10]. PFAS exposure was associated with testosterone and estradiol disruptions. Serum PFOA levels in the 68 male factory workers ranged up to 115 parts per million which is 28 million times the average serum levels in the US between 1999 and 2010 [11].

Animal and human epidemiologic studies of the general male population found associations between PFAS and Leydig cell tumors in rats and reproductive hormone disruptions in animals and humans [12].

PFAS exposure is also associated with male reproductive organ development and sperm quality parameters. Several studies reported negative associations between semen quality and PFAS exposure; with up to a 35% reduction in sperm morphology [13]. In one study, PFAS industrial air emissions are associated with a reduction in semen quality, testicular volume, penile length, and anogenital distance (agd) [14].

PFAS chemicals were once termed "emerging contaminants" but now the science is clear–exposure is associated with many chronic diseases, cancers, and reproductive impacts. In addition, PFAS chemicals have been found to cross the placenta, bioaccumulate, and cause gender-specific hormone disruption in

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children [15]. The scientific community lags far behind the industry in understanding the health effects of these sex-specific and sometimes sex-dimorphic endocrine disruptors. Non-monotonic, non-linear dose responses are typical of this chemical class and challenge traditional exposure risk assessment and regulatory program decisions [16].

Viewed in isolation, any one of these outcomes should raise eyebrows, but the full body of evidence should sound alarm bells about the public health crisis future generations will face [17].

In June 2022, the US EPA lowered the lifetime health advisory to 100,000 times lower than the agency's 2009 advisory for only two PFAS chemicals [18]. To date, the EPA has failed to enact promised enforceable drinking water standards and classify PFAS as a hazardous waste under CERCLA pushing states across the country to act [19].

The road to regulation is long and fraught with industry pushback, so clinicians and scientists need to support and be abreast of developing regulations but in the meantime recognize the impacts of PFAS exposures to protect public health more effectively.

REFERENCES

1. Agency for Toxic Substances and Disease Registry. Toxicological profile for perfluoroalkyls. 2009.
2. Aimuzi R, Luo K, Chen Q, Wang H, Feng L, Ouyang F, et al. Perfluoroalkyl and polyfluoroalkyl substances and fetal thyroid hormone levels in umbilical cord blood among newborns by prelabor caesarean delivery. Environ Int. 2019;130:104929.
3. Barry V, Winquist A, Steenland K. Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. Environ Health Perspect. 2013;121(11-12):1313-1318.
4. Benninghoff AD, Bisson WH, Koch DC, Ehresman DJ, Kolluri SK, Williams DE. Estrogen-like activity of perfluoroalkyl acids *in vivo* and interaction with human and rainbow trout estrogen receptors *in vitro*. Toxicol Sci. 2011;120(1):42-58.
5. Biegel LB, Liu RC, Hurtt ME, Cook JC. Effects of ammonium perfluorooctanoate on Leydig-cell function: *in vitro*, *in vivo*, and *ex vivo* studies. Toxicol Appl Pharmacol. 1995;134(1):18-25.
6. Calafat AM, Wong LY, Kuklenyik Z, Reidy JA, Needham LL. Polyfluoroalkyl chemicals in the US population: data from the National Health and Nutrition Examination Survey (NHANES) 2003–2004 and comparisons with NHANES 1999-2000. Environ Health Perspect. 2007;115(11):1596-1602.
7. Centers for Disease Control and Prevention, U. S. Department of Health and Human Services. National Report on Human Exposure to Environmental Chemicals. 2022.
8. Cook JC, Murray SM, Frame SR, Hurtt ME. Induction of Leydig cell adenomas by ammonium perfluorooctanoate: A possible endocrine-related mechanism. Toxicol Appl Pharmacol. 1992;113(2):209-217.
9. Costa G, Sartori S, Consonni D. Thirty years of medical surveillance in perfluorooctanoic acid production workers. J Occup Environ Med. 2009;51(3):364-372.
10. Cui Q, Pan Y, Wang J, Liu H, Yao B, Dai J. Exposure to per-and polyfluoroalkyl substances (PFASs) in serum versus semen and their association with male reproductive hormones. Environ Pollut. 2020;266:115330.
11. Del Fiore P, Cavallin F, Mazza M, Benna C, Monico AD, Tadiotto G, et al. Per-and polyfluoroalkyl substances (PFAS) exposure in melanoma patients: A retrospective study on prognosis and histological features. Environ Health. 2022;21(1):126.
12. di Nisio A, Sabovic I, Valente U, Tescari S, Rocca MS, Guidolin D, et al. Endocrine disruption of androgenic activity by perfluoroalkyl substances: Clinical and experimental evidence. J Clin Endocrinol Metab. 2019 Apr;104(4):1259-1271.
13. Gilliland FD. Fluorocarbons and human health: Studies in an occupational cohort. University of Minnesota. 1992.
14. Grandjean P, Heilmann C, Weihe P, Nielsen F, Mogensen UB, Budtz-Jorgensen E. Serum vaccine antibody concentrations in adolescents exposed to perfluorinated compounds. Environ Health Perspect. 2017;125(7):077018.
15. Henry ND, Fair PA. Comparison of *in vitro* cytotoxicity, estrogenicity and anti-estrogenicity of triclosan, perfluorooctane sulfonate and perfluorooctanoic acid. J Appl Toxicol. 2013;33(4):265-272.
16. Jensen TK, Andersen LB, Kyhl HB, Nielsen F, Christesen HT, Grandjean P. Association between perfluorinated compound exposure and miscarriage in Danish pregnant women. PloS One. 2015;10(4):e0123496.
17. Joensen UN, Bossi R, Leffers H, Jensen AA, Skakkebæk NE, Jorgensen N. Do perfluoroalkyl compounds impair human semen quality? Environ Health Perspect. 2009;117(6):923-927.
18. Kim S, Chen J, Cheng T, Gindulyte A, He J, He S, et al. PubChem in 2021: New data content and improved web interfaces. Nucleic Acids Res. 2021;49(D1):D1388-D1395.
19. Kjeldsen LS, Bonefeld-Jorgensen EC. Perfluorinated compounds affect the function of sex hormone receptors. Environ Sci Pollut Res Int. 2013;20:8031-8044.