

TOXIC AND HAZARDOUS SUBSTANCES LITIGATION

October 2019

IN THIS ISSUE

Jeffrey Karp, Edward Mahaffey, Maxwell Unterhalter and James Wilhelm discuss how scientific studies on PFAS toxicity and potential endangerment to human health and the environment have influenced the increasing number of lawsuits against PFAS manufacturers and distributors, and other potentially liable parties.

PFAS Update: Evolving Science and Liability

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Per- and poly-fluoroalkyl substances (PFAS) are a group of man-made chemical compounds that are now ubiquitous in the environment. Federal and state governments grapple with finding the best approach to handle PFAS releases from manufacturing facilities, fire and crash training areas, and industrial and municipal waste disposal sites into soil, groundwater, surface water, and drinking water systems. A growing awareness of PFAS persistence and the possible negative health effects at low levels in the environment also has resulted in an increasing number of lawsuits against manufacturers and distributors of PFAS and other potentially liable parties. This article discusses how scientific studies on PFAS toxicity and potential endangerment to human health and the environment have influenced such litigation.

How Dangerous are PFAS?

PFAS are absorbed readily following inhalation and ingestion, and are not metabolized in humans or laboratory

animals.¹ Several federal agencies have investigated the toxicity of PFAS compounds, reviewing human epidemiological and animal laboratory studies, and reached differing conclusions.

In 2016, the United States Environmental Protection Agency (“U.S. EPA”) issued Lifetime Drinking Water Health Advisories for Perfluorooctanoic Acid (“PFOA”) and Perfluorooctane Sulfonate (“PFOS”)² in which the Agency determined that exposure to PFOA and PFOS, two of the most prevalent PFAS chemicals, may adversely affect fetuses during pregnancy and breastfed infants; harm the liver, immune system, and thyroid; and cause changes in cholesterol levels.³ The U.S. EPA also concluded that there was “suggestive evidence of the carcinogenic potential” of PFOA⁴ and PFOS⁵ in humans.⁶ As a result of its review of the scientific data, the U.S. EPA established a combined 70 parts per trillion (ppt) limit for PFOA and PFOS in drinking water systems, and recommended that providers take appropriate measures to

¹ See ATSDR Toxicological Profile for Perfluoroalkyls, Draft for Public Comment, at p. 4 (June 2018). <https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf>.

² U.S. EPA’s Drinking Water Health Advisories are assessments of scientific evidence that provide non-regulatory, non-binding information for drinking water system operators on when and how to take appropriate action to protect public health.

³ See <https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos>.

⁴ Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA) – May 2016, pp. 44 – 47. https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final_508.pdf.

⁵ https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final_508.pdf.

⁶ See also International Agency for Research on Cancer, Perfluorooctanoic acid IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, at p. 98 (December 22, 2016) (concluding that PFOA is “possibly carcinogenic to humans”). <https://monographs.iarc.fr/wp-content/uploads/2018/06/mono110-01.pdf>.

protect their customers from ingesting higher PFAS concentrations.

In 2018, the United States Department of Human Health and Services (“HHS”) Agency for Toxic Substances and Disease Registry (“ATSDR”) conducted its own review of PFAS toxicity, which was reported in its Toxicological Profile for Perfluoroalkyls.⁷ Based on its evaluation, ATSDR determined that associations existed between exposure to certain PFAS and several negative health outcomes, including: pregnancy-induced hypertension/pre-eclampsia; liver damage; increases in serum lipids, particularly total cholesterol and low-density lipoprotein cholesterol; increased risk of thyroid disease; decreased antibody response to vaccines; increased risk of asthma diagnosis; increased risk of decreased fertility; and small decreases in birth weight.⁸ These results followed a 2016 evaluation of PFAS health impacts by the HHS National Toxicology Program, which found PFOA and PFOS altered immune

function in humans.⁹ Based on its assessment of the studies, the ATSDR published adult and child “intermediate” Minimal Risk Levels (MRLs) for PFAS compounds, as follows: 78 ppt and 21 ppt for PFOA, 52 ppt and 14 ppt for PFOS, 517 ppt and 140 ppt for PFHxS, and 78 ppt and 21 ppt for PFNA.¹⁰

The values developed by U.S. EPA and ATSDR represent the highest levels at which the respective agencies concluded that exposure to PFAS would not result in associated negative health outcomes. The U.S. EPA’s exposure limits reflect an assessment of health effects for the most sensitive populations (e.g., developing fetuses and newborns), designed to protect individuals over a lifetime of exposure to PFAS in drinking water. U.S. EPA’s Lifetime Health Advisory limits do not represent definitive cut-offs between safe and unsafe conditions. Rather, they reflect the Agency’s determination of an adequate margin of protection for individuals throughout their

⁷ See ATSDR Toxicological Profile for Perfluoroalkyls.

⁸ *Id.* at pp. 5-6.

⁹ National Toxicology Program. 2016. Monograph on Immunotoxicity Associated with Exposure to Perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS).

<https://ntp.niehs.nih.gov/pubhealth/hat/noms/pfoa/index.html>.

¹⁰ ATSDR’s MRLs are presented in terms of dosage amounts [milligrams/kilogram/day], rather than concentrations [ppt]. A dose is the amount of a substance to which a person is exposed during a specified time period. The PFAS MRLs are designated “intermediate,” which refers to exposure during a time period of 15 to 364 days. MRLs designated “acute” refer to 1 to 14 days, and those that are “chronic” refer to more than 365 days.

As opposed to doses, a concentration is the amount of a substance present in a certain amount of soil, water, air or other environmental media. For drinking water exposures, doses can be used to determine equivalent water concentrations using mathematical equations. ATSDR’s MRL concentration equivalents have been presented here for ease of comparison with the U.S. EPA values. ATSDR’s PFAS dose-to-concentration calculations are based on the guidelines published in the [Public Health Assessment Guidance Manual](#) and [U.S. EPA 2011 Exposure Factors Handbook](#). See https://www.atsdr.cdc.gov/pfas/mrl_pfas.html; https://www.atsdr.cdc.gov/docs/PFAS_Public_KeyMessages_June20_Final-508.pdf.

lives from possible adverse health effects. The ATSDR MRLs provide an estimate of the amount of PFAS a person can eat, drink, or breathe each day without an appreciable risk to non-cancer health effects. However, exposure above the published “intermediate” PFAS MRLs does not mean health problems will occur. Rather, the increased exposure acts as a signal for health assessors to further evaluate possible harms to human health from that specific exposure.¹¹ While U.S. EPA and ATSDR have recommended safe ingestion exposure levels, it is unclear what specific levels of PFAS found in blood serum mean in terms of possible health effects. Blood test results that show PFAS in blood serum do not inform the need for treatment of a health problem, nor do they predict or rule-out the potential development of future health problems.¹²

Additional studies have been influential in identifying PFAS health risks. In some of the most widely publicized PFAS epidemiological studies, the C8 Science Panel¹³ announced in 2012 that human exposure to 50 ppt or more of PFOA in drinking water for one year or longer had “probable links” with certain

human diseases, including kidney and testicular cancer, ulcerative colitis, thyroid disease, preeclampsia, and medically-diagnosed high cholesterol.¹⁴ The C8 Science Panel’s studies evaluated health outcomes for nearly 80,000 community members exposed to PFOA contamination near a DuPont de Nemours, Inc. (“DuPont”) PFOA manufacturing plant in West Virginia. The panel of epidemiologists was created in connection with a 2005 settlement between DuPont and a group of plaintiffs who had alleged a variety of negative health impacts from drinking PFAS-contaminated water in order to assess whether or not a “probable link” between C8 exposure and human disease existed in the affected community. The panel’s “probable link” determinations relied both on their own findings and other studies.

The differences among the advisory exposure levels developed by U.S. EPA, ATSDR and other key authorities have drawn significant attention.¹⁵ However, the bases for these differences are largely dependent on each risk assessor’s selection of critical effects¹⁶ and uncertainty factors to derive

¹¹See https://www.atsdr.cdc.gov/docs/PFAS_Public_KeyMessages_June20_Final-508.pdf.

¹² See <https://www.atsdr.cdc.gov/pfas/pfas-blood-testing.html>.

¹³See <https://www.nytimes.com/2016/01/10/magazine/the-lawyer-who-became-duponts-worst-nightmare.html>.

¹⁴ See <http://www.c8sciencepanel.org/index.html>.

¹⁵ See Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan, Michigan PFAS Science Advisory Panel, p. 44 (December 7, 2018). World-wide health-based

advisories for PFOS and PFOA in drinking water have set safe levels ranging from 13 – 530 ppt for PFOS, and 14 – 1,000 ppt for PFOA. https://www.michigan.gov/documents/pfasresponse/Science_Advisory_Board_Report_641294_7.pdf.

¹⁶ Critical effect: For deterministic effects (a phenomenon committed to a particular outcome determined by fundamental physical principles), it is the first adverse effect which appears when the threshold (critical) concentration or dose is reached in the critical organ. See International Union of Pure

reference doses,¹⁷ as well as methodological principles regarding assumptions of exposure and relative source contribution, all of which underlie the calculations of ingested PFAS limits.¹⁸ As the Michigan PFAS Science Advisory Panel stated: “there may be only limited scientific evidence for claiming one [advisory] or the other is ‘better.’...[Risk assessment] is an art of practice more than an exact science.”¹⁹ As a result, the risk assessment results have varied depending upon the manner in which the associations between PFAS exposure and health outcomes are addressed, even as the scientific studies being relied upon have largely remained the same.

While the risk assessments conducted by U.S. EPA and ATSDR give significant weight to PFAS exposure and human health associations, the causal relationship

between PFAS toxicity and adverse human health effects remains unclear.²⁰ Potential health effects are associated with PFAS exposure where epidemiological studies have found increases in those health effects and increased levels of PFAS in blood serum. However, these health effects may be caused by a variety of different factors, such as exposure to other environmental contaminants, making it difficult to assess whether PFAS exposure alone is the cause of specific observed health problems.²¹ Although laboratory animal studies have helped to clarify whether a causal relationship exists by demonstrating health impacts in other animals, comparison of study results across species is difficult. Accordingly, the differences in elimination

and Applied Chemistry Glossary of Terms Used in Toxicology, 2nd Edition, IUPAC Recommendations, 2007. Environmental Health, Toxicology, & Chemical Information, U.S. National Library of Medicine, National Institute of Health. <https://envirotoxininfo.nlm.nih.gov/toxicology-glossary.html>.

¹⁷ Reference dose: An estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. *See id.*

¹⁸ Scientific Evidence and Recommendations for Managing PFAS Contamination in Michigan, at pp. 43-48. For example, in determining PFOS MRLs, ATSDR’s risk assessors chose a developmental outcome point-of-departure derivation that is identical to the one selected by the U.S. EPA. However, the ATSDR MRL estimate is lower (more stringent) than the U.S. EPA reference dose value because ATSDR applied a 10-fold higher uncertainty factor that includes a modifying factor due to concern that immune-toxicity may be a more

sensitive outcome than developmental toxicity. The Michigan Advisory Panel’s full report contains a comprehensive discussion of features that distinguish the risk assessments of PFOS and PFOA among the U.S. EPA, ATSDR, and the states of Minnesota and New Jersey.

¹⁹ *Id.* at pp. 43, 48. Note that while the Michigan Advisory Panel found underlying similarities between different authorities’ risk assessments, nonetheless, it concludes: “that if one accepts the probable links between PFOA exposure and health effects detected in the epidemiological literature as critical effects for health risk assessment, then 70 ppt in drinking water might not be sufficiently protective for PFOA, and possibly by extrapolation to PFOS.” *Id.* at p. 11.

²⁰ *See* ATSDR Toxicological Profile for Perfluoroalkyls at p. 5.

²¹ *See* An Overview of Perfluoroalkyl and Polyfluoroalkyl Substances and Interim Guidance for Clinicians Responding to Patient Exposure Concerns, (June 7, 2017).

half-lives,²² species differences in the mechanism of toxicity²³ and the lack of adequate mechanistic data,²⁴ and differences in measurement of exposure levels between epidemiological and experimental studies create difficulties in assessing how observed health effects in other species translate into potential health effects for humans.²⁵ Thus, while laboratory animal studies serve to supplement scientific understanding of associations demonstrated in epidemiological studies, causal relationships between PFAS exposure and human health outcomes are not yet well understood.

This lack of clarity has led some experts, such as the [2018 Australian Expert Health Panel for PFAS](#), to conclude that there is no current evidence demonstrating a large impact on

an individual's health or overall cancer risk from PFAS exposure.²⁶ The Australian panel concluded that, while there are observed health effects associated with PFAS exposure, the level of health effect reported in people with the highest exposure is still within the normal ranges for the general population.²⁷ The panel also cautioned that the hundreds of epidemiological studies addressing the association between PFAS and health outcomes²⁸ are based on only a few populations and the observed effects may be explained by confounding variables, such as age, smoking, or socio-economic status.²⁹ Similarly, in issuing its health advisories, the U.S. EPA recognized the limitations of some epidemiological studies on PFAS exposure due to the failure to control for alternative risk factors.³⁰ Other studies of PFAS-impacted populations

²² For example, the estimated elimination half-life for PFOA ranges from 8 years in humans to 1.9 hours in female rats. See ATSDR Toxicological Profile for Perfluoroalkyls at p. 4.

²³ For example, there is strong evidence that some PFAS effects observed in rodents, such as hepatotoxicity, immuno-toxicity, and developmental toxicity, involve the activation of peroxisome proliferator-activated receptor- α (PPAR α), a receptor to which humans are less responsive. Thus, the known mechanisms of toxicity of PFAS do not clearly interact with humans in a comparable manner. See ATSDR Toxicological Profile for Perfluoroalkyls at p. 4.

²⁴ Additionally, PPAR α -independent mechanisms for PFAS toxicity also are involved, and it is not known if species differences exist for these mechanisms. See ATSDR Toxicological Profile for Perfluoroalkyls at pp. 4-5.

²⁵ See ATSDR Toxicological Profile for Perfluoroalkyls at p. 4.

²⁶ See <https://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-expert-panel.htm>.

²⁷ *Id.* at 2. Health effects referenced by the Australian Panel include those identified by the U.S. EPA and ATSDR, such as increased levels of cholesterol, increased levels of uric acid in the blood serum, reduced kidney function, alterations in some indicators of immune response, altered levels of thyroid hormones, lower birth weight in babies, delayed menstruation in adolescent girls, and earlier onset of menopause in women.

²⁸ A large number of epidemiological studies are focused on the high levels of exposure surrounding the DuPont PFOA manufacturing plant in West Virginia, which was the focus of the C8 Science Panel's studies.

²⁹ See <https://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-expert-panel.htm>.

³⁰ See Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS) – May 2016, p. 10. https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final_508.pdf.

actually have rebutted the associations between cancer and PFAS exposure, finding no statistically significant increase in cancer rates in populations exposed to PFOA for multiple decades.³¹

Proving PFAS Claims

Due to the range and variability of study results, individuals who pursue PFAS litigation may find it difficult to establish a causal link between PFAS exposure and negative health impacts. In order to prevail, they generally must demonstrate that exposure to PFAS contamination has caused them harm. As discussed above, while there are studies that support a positive association, it may prove difficult to tie incidence of PFAS exposure to a particular disease or illness. On the other hand, drinking water providers in PFAS litigation do not seem to face this potential evidentiary hurdle because they are not seeking relief stemming from a specific illness or disease allegedly caused by exposure to PFAS contamination. In the cases we have reviewed, the drinking water providers typically are seeking compensation to

upgrade water treatment technologies to meet the 70 ppt PFAS health advisory limit recommended by the U.S. EPA, as well as injunctive relief to require that defendants remediate PFAS-contaminated groundwater.

For example, in a case involving PFAS contamination that allegedly migrated from a 3M Company manufacturing facility in Alabama, both the West Morgan-East Lawrence Sewer and Water Authority ("WMEL"), and individuals who received their drinking water from the WMEL, brought suit to recover damages due to the alleged release of PFAS contaminants upriver from WMEL's drinking water supply intake. The individual plaintiffs asserted that the drinking water to which they were exposed exceeded the U.S. EPA limits and ATSDR MRLs, thus causing them harm³² by increasing their risk of cancer, immunotoxicity, thyroid disease, ulcerative colitis, and high cholesterol. These plaintiffs pinned their assertions of harmful PFAS levels on the U.S. EPA's findings and those of the C8 Science Panel.³³ While the defendants admitted that PFAS concentrations

³¹ See Cancer Incidence Investigation Findings: Village of Hoosick Falls, 1995-2014. (May 2017). (PFOA contamination in the Village of Hoosick Falls' (NY) public drinking water supply was estimated to have occurred for 40 years or longer, though exact dates are unknown. In 2014, sampling results showed PFOA concentrations of 595 ppt average in the drinking water supply.) https://www.health.ny.gov/environmental/investigations/hoosick/docs/cancer_report.pdf; see also Cancer Incidence Report, Merrimack, NH. (January 2018). (Cancers associated with PFOA were not found to be higher in Merrimack, NH where

there were low levels of PFOA contamination in the drinking water system.) <https://www.dhhs.nh.gov/dphs/pfcs/documents/merrimack-cancer-012018.pdf>.

³² Measured concentrations of PFOA and PFOS in the municipal drinking water supply, prior to the installation of a new type of filter, were 100 ppt and 190 ppt, respectively.

³³ *West Morgan-East Lawrence Water and Sewer Authority, et al. v. 3M Company, et al.*, U.S. District Court No. 5:15-01750 (N.D. Ala. 2015), Consolidated Individual and Class Action Complaint at pp. 7-18

measured in the WMEL water supply exceeded the U.S. EPA's drinking water advisory limits and ATSDR MRLs, they disputed that an association existed between the PFAS in the drinking water supply and the asserted potential health outcomes.³⁴ The defendants also denied that the C8 Science Panel's "probable link" determinations accurately summarized the results of epidemiological findings,³⁵ and further disputed that the PFAS levels found in the drinking water were harmful.³⁶ Moreover, in the absence of an existing federal or state regulatory framework, the defendants have asserted that their conduct was consistent with law, applicable standards of care, and industry practice.³⁷ Litigation of the individuals' claims is ongoing.

In contrast, defendants 3M Company and Daikin America, Inc. settled with the WMEL for \$35 and \$4 million, respectively. Some commentators attribute the difference in outcomes to a less onerous evidentiary

burden facing drinking water providers who need not demonstrate a causal link between exposure to PFAS contamination and human health outcomes.³⁸ Rather, the drinking water providers may rely on the EPA's Health Advisories as evidence of a significant public health risk that must be addressed, without proving the actual occurrence of illness or disease from exposure to PFAS contamination found in the drinking water supply. For this reason, defendants in PFAS litigation appear more willing to settle with drinking water providers and municipalities than with individuals.³⁹ As a result, states and municipalities have initiated legal action promptly seeking to hold manufacturers and users liable for costs to upgrade water treatment systems and to require clean-up of PFAS contamination.⁴⁰

Yet even where states and municipalities have highlighted exposure limits, conflicting scientific evidence has sometimes weakened

(Filed 5/6/2019). (An amended complaint was filed following the WMEL and 3M Company settlement).

³⁴ *West Morgan-East Lawrence Water and Sewer Authority, et al. v. 3M Company, et al.*, U.S. District Court No. 5:15-01750 (N.D. Ala. 2015), Answers of 3M Company and Dyneon, LLC to Plaintiffs' Consolidated Individual and Class Action Complaint at pp. 8-9 (Filed 6/3/2019). (An amended complaint was filed following the WMEL and 3M Company settlement).

³⁵ *Id.* at pp. 5-6.

³⁶ *West Morgan-East Lawrence Water and Sewer Authority, et al. v. 3M Company, et al.*, U.S. District Court No. 5:15-01750 (N.D. Ala. 2015), Daikin America, Inc.'s Answer to Third Amended Class Action Complaint at p. 11 (Filed 6/3/2019).

³⁷ *Id.* at p. 18; See also Answers of 3M Company and Dyneon, LLC in *West Morgan-East Lawrence Water and Sewer Authority, et al. v. 3M Company, et al.*, U.S. District Court No. 5:15-01750 (N.D. Ala. 2015) at p. 21.

³⁸ See <https://www.circleofblue.org/2018/world/as-pfas-lawsuits-proliferate-legal-tactics-emerge/>.

³⁹ See *State of Vermont, Agency of Natural Resources v. Saint-Gobain Performance Plastics Corporation*, Docket No. 92-4-19 Bncv (2019) (Settlement provided funding arrangement that covered most of the expected \$20-to-\$25 million increase in costs to treat PFAS-contaminated drinking water.); *City of Lake Elmo v. 3M Company*, No. 0:2016-CV-02557 (D. Minn. 2017) (3M settled with the City for \$2.7 million).

⁴⁰ See <https://www.eenews.net/stories/1060469135>.

cases against PFAS manufacturers.⁴¹ In 2010, the Minnesota State Attorney General filed suit against 3M Company for \$5 billion in alleged damages to natural resources from PFAS contamination.⁴² However, a Minnesota State Department of Public Health report, released leading up to the trial, concluded that the cities affected by thirty years of exposure registered no increase in the rates of cancer or low birth-weight babies.⁴³ While the case had centered on alleged damages to the environment from PFAS contamination, the Minnesota Attorney General was expected to employ expert testimony on the health risks of PFAS exposure and thus bolster Minnesota's case.⁴⁴ The release of the Minnesota State Department of Health report appeared to mitigate the threat of those claims,⁴⁵ as the parties settled within a week of the report's release for less than one-fifth of the original amount sought.⁴⁶

In an apparent attempt to circumvent this causation issue, a firefighter claiming to have been exposed to PFOA-laden aqueous film-forming foams (AFFFs) brought suit on

behalf of all individuals residing in the U.S. with a detectable level of PFAS in their blood serum, seeking to hold PFAS manufacturers accountable to determine the chemical's true health impacts. *Hardwick et al. v. 3M Company, et al.*, 2:18-cv-01185 (S.D. Ohio 2018). In addition to monetary damages, the complaint requests the establishment of an independent panel of scientists—jointly selected by the parties and funded by defendants—to further study and evaluate the health effects of PFAS.

Although issues of causation remain in flux, litigation continues to ensue with complainants asserting claims for relief based on common law torts, including negligence (negligence per se and gross negligence), property torts (trespass), nuisance (public and private), intentional torts (battery), and product liability (defective product failure to warn and design defects). Notably, lawsuits arising from the widespread use of AFFFs containing PFOA and PFOS have grown so numerous that in November 2018, the United States Judicial Panel on Multidistrict

⁴¹ See *State of Minnesota v. 3M Company*, 27-CV-10-28862 (Hennepin County District Court) State of Minnesota Amended Complaint at p. 12 (Filed 1-18-2011). Concentration of PFAS contaminants at in soil and groundwater at disposal sites allegedly exceeded health based standards developed by the Minnesota Department of health. <http://www.mncourts.gov/Media/StateofMinnesotaavs3MCompany.aspx>.

⁴² See <https://fortune.com/2018/02/20/3m-scotchgard-pollution-lawsuit/>.

⁴³ See <https://www.twincities.com/2018/02/20/minnesota-3m-reach-settlement-ending-5-billion-lawsuit/>.

⁴⁴ See <https://www.twincities.com/2018/02/19/minnesota-vs-minnesota-agency-pushes-back-in-5-billion-3m-suit/>.

⁴⁵ See <https://fortune.com/2018/02/20/3m-scotchgard-pollution-lawsuit/>.

⁴⁶ See *The State of Minnesota and 3M settled for \$850 million following the release of the health department study.* <https://www.twincities.com/2018/02/20/minnesota-3m-reach-settlement-ending-5-billion-lawsuit/>.

Litigation [granted an order](#) to transfer and consolidate 75 cases from around the country. *In RE: Aqueous Film-Forming Foams Products Liability Litigation*, MDL No. 2873. Currently, the multidistrict litigation has grown to include more than one hundred cases.⁴⁷

While PFOA and PFOS have been phased out of manufacture and use in the United States, their unique physiochemical properties ensure that these chemicals will remain in the environment for the foreseeable future. U.S.-based chemical manufacturers have begun to replace PFOA and PFOS in their products, in some cases substituting other PFAS compounds for which health-related data is lacking.⁴⁸ These less-understood compounds are frequently short-chain PFAS, which “are less adsorbable, more persistent and more mobile in groundwater and soil” than long-chain PFAS such as PFOA and PFOS.⁴⁹ An Auburn University study has noted the possibility that these differences “may pose broader risks” to “human and ecosystem health,”⁵⁰ and thus the study argued that more research into the health effects of short-chain PFAS is urgently needed.⁵¹

A recently released set of documents, while probably contributing little new evidence on the causation question, still may hurt the PFAS manufacturers DuPont and 3M in future litigation by establishing their knowledge of PFAS risks before they were publicly acknowledged. This dossier was released by the Environmental Working Group (EWG) and, according to 3M, the documents regarding 3M had been among those released previously by the Minnesota attorney general during Minnesota’s litigation against 3M.⁵² The documents may be particularly damaging to DuPont in the context of a pending lawsuit by its own spin-off company, The Chemours Company, which accused DuPont of fraudulent underestimation of the environmental liabilities that the spin-off process left to Chemours.⁵³ The documents released by the EWG include a 1950 3M study finding that PFAS bioaccumulates in the blood of mice, 1961 DuPont research linking PFAS to enlarged livers in rabbits and rats, 1981 documents concerning decisions by both 3M and DuPont to reassign female workers due to concerns that PFAS might harm developing fetuses, and 3M and DuPont studies from the late 1980s and the 1990s

⁴⁷ See <https://www.scd.uscourts.gov/mdl-2873/>.

⁴⁸ See U.S. EPA’s PFAS Action Plan, p. 2, (February 2019). (U.S. EPA is working to investigate additional PFAS compounds such as GenX chemicals and PFBS). https://www.epa.gov/sites/production/files/2019-02/documents/pfas_action_plan_021319_508compilant_1.pdf.

⁴⁹ Fan Li, Jun Duan, Shuting Tian, Haodong Ji, Yangmo Zhu, Zongsu Wei, Dongye Zhao, “Short-chain per- and polyfluoroalkyl substances in aquatic

systems: Occurrence, impacts and treatment,” *Chemical Engineering Journal*, Volume 380, 2020, 122506, ISSN 1385-8947, (<http://www.sciencedirect.com/science/article/pii/S1385894719319096>), 3.

⁵⁰ Id. at 19.

⁵¹ Id. at 5.

⁵² Suzanne Yohannan, “As EWG Details Industry’s PFAS Findings, Dupont Fortifies Cleanup Pledges,” *Inside EPA Weekly Report* (Sept. 6, 2019).

⁵³ Id.

regarding possible links between PFAS and cancer.⁵⁴

While there appears to be ample scientific evidence of an association between PFOA and PFOS and adverse health effects, the current scientific understanding of the causal mechanisms by which PFAS affects humans remains incomplete. Expert analysis of study results has led to assertions that state regulations imposing PFAS limits are overhyped and unnecessary,⁵⁵ while others contest that current advisories are woefully inadequate to protect human health and should be more stringent.⁵⁶ Nevertheless, many more lawsuits are likely to be filed as new PFAS-contaminated sites are identified monthly. It remains to be seen whether the results of further research studies will solidify the causal link between PFAS exposure and specific illnesses or diseases.

⁵⁴ See <https://www.ewg.org/pfastimeline/>.

⁵⁵ See https://www.americanbar.org/groups/environment_energy_resources/publications/trends/2018-2019/january-february-2019/fear-and-loathing/.

⁵⁶ See Written testimony of James DeWitt, PhD, DABT Associate Professor, Department of Pharmacology and Toxicology, Brody School of Medicine, East Carolina University. Hearing on

"Protecting Americans at Risk of PFAS Contamination & Exposure," Before the Subcommittee on Environment and Climate Change, Committee on Energy and Commerce, U.S. House of Representatives. May 15, 2019. (Noting that U.S. EPA's Drinking Water Health Advisory levels are not sufficiently protective of health).

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