The Trouble with PFOA: Testing, Regulation and Science Concerning Perfluorooctanoic Acid and Implications for Future Litigation

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I N THE April 2007 Defense Counsel Journal, we wrote about “endocrine disrupters” (“EDs”), widely used synthetic chemicals that are believed to disrupt the body’s normal hormonal functions.1 This article will provide an update on this topic, reviewing recent testing, regulation, science, and litigation regarding one alleged ED, perfluorooctanoic acid (“PFOA”).

A. Background

PFOA, also known as C8, is a synthetic chemical that has been used in the manufacture of commercial products such as non-stick cookware, stain-resistant clothing and carpets, food wrappers, and firefighting foam, and has many industrial uses as well. Although “[t]here is still controversy over PFOA’s toxicity,”2 PFOA has raised health concerns because it is persistent in the environment, found at low levels in the blood of the general U.S. population, remains in the human body for a long time, and has been linked to adverse health effects in laboratory animals.3

In 2006, the United States Environmental Protection Agency (“EPA”) instituted — and eight manufacturers signed onto — a Global Stewardship Program designed to phase out PFOA by the year 2015.4 Recent findings of contaminated soil and drinking water and new and continuing studies on human health effects, however, ensure the potential for future litigation.5

B. Testing and Regulation

PFOA fears have spurred recent action in several states. In Alabama, allegedly high levels of PFOA and other perfluorochemicals (“PFCs”) were found in grazing land near Decatur, prompting concern that they had spread to milk and

5 See, e.g., Lab Science News, supra note 2 (stating that because of the persistence of PFOA “in the environment and the bioaccumulation and biomagnification in the food chain [it] will continue to be in the environment long after manufacturing ceases.”).
meat and seeped into the water supply.6 In response, EPA issued a short-term Provisional Health Advisory limit for PFOA in drinking water of 0.4 parts per billion (ppb).7 Tests of nearby wells and ponds revealed that, out of 51 samples, 25% exceeded that limit,8 although PFOA was present in local drinking water systems at levels “well below anything that could be a health risk.”9 The exact source of the chemicals is unknown, but a public meeting held in June 2009 discussed the status of the EPA investigation.10 The director of the EPA’s water protection division has ordered potential contributing companies to “expand a survey of private wells.”11

Drinking water supplies are also being tested in Georgia and Minnesota. In the former, the local news rushed to report elevated PFOA levels before the EPA had even seen test results.12 In the latter, the suspected source of local contamination is a firefighting foam made by 3M that allegedly “is flushed into storm sewers or left to seep into the ground” after being used “in training exercises, often on city-owned property adjacent to municipal wells.”13 Indeed, plaintiffs’ attorneys are trolling for clients.14 California may be another future site for lawsuits, as the Carcinogen Identification Committee, which advises the state EPA’s Office of Health Hazard Assessment (OEHHAA), recently recommended PFOA for “the highest priority review” for inclusion in its Proposition 65 list of hazardous chemicals.15

As part of a 2005 settlement with EPA, DuPont is testing whether fluorotelomers in its consumer products are breaking down into PFOA and other perfluorinated chemicals.16 On January 8, 2009, the federal Environmental Appeals Board17 approved a three-year extension for the completion of the tests, provoking some public outcry.18

8 See Rebecca Renner, Are Perfluorochemicals Widespread in Biosolids?, 43 ENVTL. SCI. & TECH. 5164 (2009).
10 See Houston, supra note 9.
Under the program, “DuPont must provide replacement water supplies for anyone whose drinking water contains more than the EPA-recommended level of 0.4 parts per billion.” Residents near the company’s Chamber Works plant in New Jersey, however, are calling for a stricter measure, the 0.04 micrograms per liter (equivalent to 0.04 ppb in water) level recommended by the state’s Department of Environmental Protection (DEP).

C. Science

i. Environmental Studies

Environmental testing in Alabama, Minnesota, and elsewhere involves sampling the PFOA levels in the water or soil of a given area and, using a “safety” level extrapolated from the limited health studies available, determining whether the local human population is at risk. There are several problems with this approach, including the uncertain derivation of the safety level, as illustrated by the competing figures promulgated by the EPA and New Jersey’s DEP.

Researchers from DEP and Rutgers University used a “risk assessment approach” to derive “a health-based drinking water concentration protective for lifetime exposure” to PFOA of 0.04 micrograms per liter. This figure was based on two main assumptions: first, that there is a quantifiable connection between PFOA in drinking water and the blood serum of the local population; second, and more importantly, that PFOA in human blood relates to identifiable adverse health effects. The researchers determined that there is approximately a 100:1 serum to drinking water concentration ratio using two different studies. The 100:1 ratio was observed in one study of a highly contaminated area in Ohio. The researchers then found “a similar ratio” by reviewing data from five Ohio and West Virginia water districts with lower PFOA concentrations. While PFOA serum levels seemed to rise with the level in drinking water in these five lower-concentration areas, the researchers caution that, because of various unknowns, “the mean or median drinking water concentrations in these districts cannot be accurately estimated from the range of concentrations.”

To determine at what level PFOA in drinking water may pose a risk to humans, the researchers turned to a 2005 EPA draft effects, and then assessing a risk for such effects in the general population. Id.

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21 Health risk assessments do not determine disease causation, but “are used to estimate whether current or future chemical exposures will pose health risks to a broad population.” CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY, A GUIDE TO HEALTH RISK ASSESSMENT, at 4, available at http://oehha.ca.gov/pdf/HRSguide2001.pdf (last visited Sept. 8, 2009). They involve identifying any potential health problems a chemical may cause, determining what levels, patterns, and duration of exposure may create negative health
24 Post et al., supra note 22, at 4549.
25 Id. These unknown factors include “the number of people served by each [point of entry], as well as the variation of concentration over time.” Id.
risk assessment, which “identified toxicological end points in experimental animals,” mainly rats. We have previously noted the limited usefulness of animal studies in determining potential human health effects. Many problems arise in attempting to extrapolate results in animals to human beings. For example, any correlation between the effect of high blood concentration of PFOA in animals and the minuscule concentrations in humans is purely speculative. As one science panel notes, although “[a]nimal studies suggest that [PFOA] exposure can cause some cancers, but up to this point the few human studies which have been done have shown no clear increase in cancer . . . . Animal evidence indicates C8 can damage the liver, but again, the few human studies which have been done do not support this.”

In addition, animal studies have not always proven consistent, even with each other. There is a “tremendous species difference in elimination” of PFOA, and “also gender differences” found in some animals, but not in others. In fact, studies have concluded that “[i]t is clear that there are vast species differences in the toxicokinetics of PFAAs.”

The New Jersey researchers used an uncertainty factor ("UF") of 10 to account for interspecies extrapolation, but note that there was some question at EPA of “whether comparison on this basis fully addresses interspecies toxicokinetic differences, and whether an interspecies UF of 3 rather than 10 is appropriate.” The New Jersey study also used a “standard UF of 10 for subchronic to chronic exposure” to explain the difference between their 0.04 ppb figure for lifetime PFOA exposure and the 0.4 ppb provisional level for short-term exposure advised by EPA. Although a factor of 10 generally is applied to extrapolate chronic exposure from subchronic exposure, the assumption on which these levels are based are not necessarily valid. At least one study has questioned the necessity of using such a high factor. Finally, the New Jersey study

26 Id. at 4550. The most sensitive end point, 0.04, was extrapolated from the No Observed Adverse Effect Level (NOAEL) for “decreased body weight and hematological effects in the adult female rat.”

27 Berger & Junk, supra note 1, at 115–17.

28 A recent draft toxicological profile by the Agency for Toxic Substances & Disease Registry declined to derive a minimal risk level (MRL) for PFOA exposure, because of the lack of useful human studies and the fact that, “at this time, derivation of MRLs for PFOA or PFOS based on animal studies would be highly uncertain, in part, because of large interspecies differences in the toxicokinetics of perfluoroalkyls for which mechanisms are not completely understood.”

29 C8 Science Panel, http://www.c8sciencepanel.org/why.html (last visited Sept. 8, 2009). See also Christopher Lau et al., Perfluoroalkyl Acids: A Review of Monitoring and Toxicological Findings,

30 Id. at 385 (“It is clear that an understanding of body burden is crucial for interspecies extrapolation of toxicological effects.”). PFAAs, or perfluoroalkyl acids, include PFOA and PFOS.

31 Id. at 4552. The researchers compared subchronic and chronic
notes that there have been recent “data not considered” by EPA in developing its endpoints.36 Mice, the study asserts, may be a more appropriate model for health effects extrapolation than rats, and “[r]ecent mouse developmental studies show significant effects not seen in the rat.”37 These effects also have not been seen in humans.38 The New Jersey researchers also assert that evaluation of “other studies of similar or shorter duration not considered by USEPA . . . could result in a short-term health-based concentration below” 0.4 micrograms per liter.39 However, the referenced studies are animal studies, and therefore should be seen only as hypothesis-generating, potentially indicating the need for further study.40

Because PFOA is pervasive both in the environment and in human beings,41 it is
difficult to pinpoint its exact source, and the mechanism for its transfer to the human body is still unknown.42 One recent study found that dust inhalation may contribute to human exposure to PFOA.43 Another group of researchers found polyfluoroalkyl phosphoric acid diesters (“diPAP”s), a group of PFCs that break down into PFOA and are used in food wrappers, at low-level concentrations in human blood.44 The findings “suggest that diPAPs are contributing a significant portion of the PFOA found in human blood . . . maybe 10 percent or more.”45 According to the New Jersey study, non-drinking water sources “are assumed to provide 80% of exposure” to PFOA in human serum.46 Therefore, in trying to derive target PFOA concentration levels in drinking water from health end points in serum, these researchers assumed a “relative source contribution” factor of 20%.47 The scientific validity of such as assumption is unknown.

Using “default values”48 to extrapolate the risk of PFOA exposure from a particular environmental source may be a sufficient accommodation for devising a protective, prospective health measurement. This 2007 studies, which found PFOA “in approximately 98% of the population”) (last visited Sept. 8, 2009).

42 See Lab Science News, supra note 2 (“Exposure may occur through consumption of contaminated food or water, or through the use of products containing these compounds, but not all sources are known or understood.”); Lau et al., supra note 29, at 385 (“While monitoring studies have clearly shown the presence of PFAAs worldwide, the sources and pathways of exposure are unknown.”).

43 Jonas Bjorklund et al., Perfluoroalkyl Compounds (PFCs) in Indoor Dust: Concentrations, Human Exposure Estimates, and Sources, 43 ENVTL. SCI. & TECH. 2276 (2009).


46 Post et al., supra note 22, at 4550.

47 Id.

48 Id.
methodology does nothing, however, to aid in reliably determining a source of causation or liability for litigation.

ii. Human Studies

A more accurate “health-based drinking water concentration level” requires more definitive information about the actual human health consequences of PFOA exposure. Researchers currently are trying to find significant trends in the general human population, but more extensive research on occupational exposure to PFOA has continued to yield inconsistent results. Although epidemiological and medical surveillance studies of U.S. chemical plant workers have been conducted for decades, “[n]o consistent association between serum fluorochemical levels and adverse health effects has been observed.” 49 A 30-year medical surveillance study of male workers at a PFOA production plant noted that, despite an association of total cholesterol and uric acid with PFOA serum level, “[n]o clinical evidence of any specific trouble or disease has been recorded over the 30 years.” 50

The lack of reliable information on what health effects PFOA exposure may have on humans, if any, drives one large, multi-study project in the Mid-Ohio Valley. 51 As part of a settlement reached in a class action lawsuit against DuPont regarding PFOA that had leached into the groundwater surrounding its West Virginia Washington Works Plant, an independent panel of epidemiologists conducted a year-long survey of about 70,000 area residents. Ten separate studies were designed to look for any connections between the heightened levels of PFOA found in the blood of people living near the plant and such adverse health effects as impaired liver and immune function, cancer, and poor birth outcomes. As the panel noted, “There have been preliminary results in our study area indicating that some residents have C8 in their blood, but that doesn’t tell us what, if anything, C8 might be doing to human health.” 52 Preliminary results have not yet been peer reviewed. 53 One of the available status reports concludes that the panel “found little or no support” for a relationship between PFOA and miscarriage, preterm birth, or low birth weight. 54 Another report notes that some “statistically significant associations” were found between PFOA blood levels and levels of immunoglobulins, but “the magnitude of the changes across the observed exposure range is small and most

49 Lau et al., supra note 29, at 373. The European Food Safety Authority (EFSA) reports, “Epidemiological studies in PFOA-exposed workers do not indicate an increased cancer risk. Some have shown associations with elevated cholesterol and triglycerides, or with changes in thyroid hormones, but overall there is no consistent pattern of changes.” Opinion of the Scientific Panel on Contaminants in the Food chain on Perfluorooctane Sulfonate (PFOS), Perfluorooctanoic Acid (PFOA) and Their Salts, 653 THE EFSA JOURNAL 1, 4 (2008).
50 Giovanni Costa et al., Thirty Years of Medical Surveillance in Perfluorooctanoic Acid Production Workers, 51 J. OCCUPATIONAL & ENVTL. MED. 364 (2009).
51 See C8 Science Panel, Why Further Study is Necessary, www.c8sciencepanel.org/why.html (“[T]here is very little reliable information on what, if anything, C8 does to people.”).
52 Id.
53 The related C8 Health Project and C8 Science Panel recently published their first papers in a peer-reviewed journal. These papers, however, do not yet discuss any alleged connections between PFOA and adverse health. Instead, respectively, they describe the project and conclude that PFOA blood serum levels are high in the populations that lived near or worked at the plant. See Stephanie J. Frisbee et al., The C8 Health Project: Design, Methods, and Participants, ENVTL. HEALTH PERSP. doi:10.1289/ehp.0900379 (online Jul. 13, 2009), available at http://www.ehponline.org/members/2009/0800379/0800379.pdf (last visited Sept. 8, 2009); Kyle Steenland et al., Predictors of PFOA Levels in a Community Surrounding a Chemical Plant, 117 ENVTL. HEALTH PERSP. 1083 (2009).
values remain within normal ranges."\(^{55}\) Moreover, the researchers caution that the "associations found between [these] immune biomarkers and PFOA do not necessarily indicate that PFOA is the cause of changes observed."\(^{56}\) One report on an observed correlation between higher serum levels of PFOA and an increase in cholesterol admits that "the cross-sectional design of our study . . . prohibits knowing whether an increase in cholesterol may have followed or preceded an increase in PFOA."\(^{57}\) Rather, it is "possible that . . . increased lipids could lead to increased retention of PFOA/PFOS in the blood."\(^{58}\)

Other recent human studies involve much smaller samplings of a general population and, given limitations and lack of corroboration, merely highlight the need for further study. One study of 1,240 women from the Danish National Birth Cohort found a correlation between higher maternal levels of PFOA and a longer time to pregnancy ("TTP").\(^{59}\) Although the researchers identified a trend, the dose-response relationship was not consistent.\(^{60}\) Given the nature of the way women were recruited for the study and the relatively low lack of participation, there may also be sampling concerns. Pregnant women were recruited for the cohort through their general practitioners, but only "[a]pproximately 50% of all general practitioners in Denmark participated in the study, and approximately 60% of invited women accepted the invitation to participate."\(^{61}\) After that, the researchers randomly selected 1400 women from mothers who provided a blood sample, gave birth to a single live born child, and completed four telephone interviews.\(^{62}\) Questions also may be raised about information bias given the essential information gleaned from interviews and the sensitive nature of the study. In addition, although potential confounders like maternal age at delivery, pre-pregnancy body mass index, and alcohol consumption before pregnancy were considered and adjusted for, the researchers admit that they "did not have information on some important determinants of TTP, including frequency and timing of intercourse, and sperm quality."\(^{63}\) As this study so far stands alone, it is unknown how reliably its results may be extrapolated to the general population. Finally, even if a genuine link between PFOA levels and TTP may be determined, a causal relationship is far from certain, especially since a "biological mechanism by which exposure to PFOS and PFOA may reduce fecundity is unknown."\(^{64}\)

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\(^{56}\) Id.  
\(^{58}\) Id.  
\(^{60}\) Id. at 1203 tbl. II. In a strong causal relationship, one would expect to find that occurrence of illness increased with increasing levels of the suspected causal agent. In this study, however, the odds ratio for infertility decreased among groups with increasing levels of PFOA in their blood serum before it increased again. Id.  
\(^{61}\) Id. at 1201.  
\(^{62}\) Excluding 160 women with unplanned pregnancies or unknown TTP brought the final number to 1,240. Id.  
\(^{63}\) Id.  
\(^{64}\) Id. at 1204. The authors go on to note that animal studies have shown some possible mechanisms, but even these are inconsistent with each other, and a study on rats "may not apply to man, since the rodent estrous cycle is not an ideal
Another study dealing with the Danish population found that higher levels of PFOA in a small cohort group of young men were “associated with fewer normal sperm.”\footnote{Lilla N. Joensen et al., Do Perfluoroalkyl Compounds Impair Human Semen Quality?, 117 ENVTL. HEALTH PERSP. 923 (2009).} This study alone is of limited use, however, in linking PFOA to infertility. The researchers used a nonrandom sample of only 105 men,\footnote{Id. at 926 (noting that “this selection affects the homogeneity of the group when correlations are analyzed for the group as a whole [which] could potentially influence the subsequent analysis of semen quality by bias or confounding and may affect the general applicability of the results”).} and the results were inconclusive. Out of many factors tested — including sperm count and motility — only morphology showed a statistically significant association. This alone does not indicate a potential for impairment of fertility.\footnote{See S.E. Chia et al., What Constitutes a Normal Seminal Analysis? Semen Parameters of 243 Fertile Men, 13 HUMAN REPRODUCTION 3394 (1998) (“Normal sperm morphology is but one of many parameters for assessment of fertility.”). The study found that even fertile men had “a low mean percentage of normal sperm morphology,” but with a normal distribution. Id. Another study found that, together with the percentage of motile sperm, what may matter is the mean number of abnormalities observed per abnormal sperm. Pierre Jouannet et al., Male Factors and the Likelihood of Pregnancy in Infertile Couples. I. Study of Sperm Characteristics, 11 INT’L J. ANDROLOGY 379 (1988).} All of the men in the Danish study had a sufficiently high percentage of morphologically normal sperm.\footnote{See Serdar Gunalp et al., A Study of Semen Parameters with Emphasis on Sperm Morphology in a Fertile Population: An Attempt to Develop Clinical Thresholds, 16 HUMAN REPRODUCTION 110 (2001); Joensen et al., supra note 65, at 924 tbl. 2. In addition, “Sperm morphology evaluation is considered to be a highly subjective procedure . . . and a lack of objective measurements . . . continues to be a problem.” S. Parastie, The Importance of Sperm Morphology in the Evaluation of Male Infertility, http://www.gfmer.ch/Endo/PGC_network/Sperm_morphology.htm (last visited Sept. 8, 2009). It is also worth noting that “morphology was not adjusted for confounders,” and, as with the TTP study, the “mode of action of [PFOA] is not clear.” Joensen et al., supra note 65, at 924, 926.}

Other general population studies have found no significant correlations between PFOA levels in blood and adverse health effects. High-level exposure to PFOA has been associated with liver, pancreatic, and testicular tumors in animals, but the few epidemiological studies of cancer risk in humans with exposure at levels greater than the general population have shown “suggestive but inconsistent associations.”\footnote{Kristen T. Eriksen et al., Perfluorooctanoate and Perfluorooctanesulfonate Plasma Levels and Risk of Cancer in the General Danish Population, 101 J. NAT’L CANCER INST. 605 (2009).} The first study to look for any correlation between PFOA levels in blood and rates of cancer revealed “no clear difference in incidence rate ratios” of prostate, bladder, pancreatic, or liver cancer.\footnote{Id. at 924, 926.} High levels of PFOA in maternal blood in rodents have been associated with developmental effects in their young, such as delayed learning, delayed eye opening, and a decrease in motor function. However, a recent human study found “no convincing associations between developmental milestones in early childhood and levels of PFOA or PFOS as measured in maternal plasma early in pregnancy.”\footnote{Fei et al., supra note 38, at 1391.} In another study, researchers “observed no correlation” between PFOA levels in maternal blood of Japanese women and birth weight and size.\footnote{Noriaki Washino et al., Correlations between Prenatal Exposure to Perfluorinated Chemicals and Reduced Fetal Growth, 117 ENVTL. HEALTH PERSP. 660 (2009). The researchers note that their results are inconsistent with two previous reports, and “cannot fully explain the inconsistencies,” even after using different confounder adjustments. Id.}

D. Litigation

How does the available science affect future litigation? The cases launched thus far seem to indicate an uphill battle faced by prospective plaintiffs. Without being able to prove — or, it seems, even allege — actual physical injury, PFOA plaintiffs recently have attempted a variety of tactics.
One large, multidistrict suit (consolidating 22 cases of plaintiffs claiming, among other things, that DuPont made false representations of the safety of its non-stick cookware coatings and failed to warn of potential health risks), sought only "recovery solely for economic damage."73 Plaintiffs did not press on after failing to achieve class action status, and the case was dismissed.74

Class action status also was denied in three drinking water contamination suits filed in New Jersey and West Virginia district courts.75 In all of the suits, the plaintiffs listed a variety of claims, but focused solely on medical monitoring.76 States have different rules for medical monitoring claims, if allowed at all, but the basic requirements that plaintiffs must prove include: (1) they have been significantly exposed to a toxic substance, (2) such exposure may cause serious disease, and (3) they are at a “distinctive increased risk of disease” due to that exposure.77 Noting that “[t]he certification of a class action under Federal Rule of Civil Procedure 23 is a two-step process,” the court in Rhodes v. E.I. DuPont de Nemours & Co. moved right to the second step and found that the plaintiffs’ case could not be certified under Rule 23(b).78 Specifically, “a proposed class must be ‘cohesive’ to be certified under Rule 23(b)(2),” but the class was not cohesive because the claims “require individualized inquiries that are not conducive to common treatment.”79 The court in Rowe v. E.I DuPont de Nemours & Co. first found that the proposed class there did meet the requirements of Rule 23(a),80 but then determined that the class could not be certified under Rule 23(b) because of too many individualized issues.81 The nature of the medical monitoring claim itself made class action status inapplicable in both cases, as none of the plaintiffs could show significant exposure or a significantly increased risk of disease on a class-wide basis.82 The plaintiffs’ attempt to rely on the environmental risk assessment approach

75 Rowe v. E.I. DuPont de Nemours & Co., No. 06-1810, 2008 WL 5412912 (D.N.J. Dec. 23, 2008) (consolidating the motions from two separate suits in one opinion), Rhodes v. E.I. DuPont de Nemours & Co., 253 F.R.D. 365 (S.D. W.Va. 2008). The Rhodes court noted that although the suit was filed because PFOA levels in the water district in question had exceeded the level at which DuPont had entered into a state court settlement over the same Washington Works plant (see Leach v. E.I. DuPont de Nemours & Co., No. 01-C-608, 2002 WL 1270121 (W.Va.Cir.Ct. Apr. 10, 2002)), “[t]hat level did not constitute a concession by DuPont about the quantity of C-8 that must be in drinking water to effect a significant exposure.” Rhodes, 253 F.R.D. at 375.
76 See Rowe, 2008 WL 5412912 at *4 (“[B]oth potential classes have focused all their attention on the medical monitoring aspect of the case and completely ignored the other claims listed in their complaints.”); Rhodes, 253 F.R.D. at 372 (“The plaintiffs . . . have failed to provide any argument or analysis as to their causes of action, with the sole exception of their claim for medical monitoring.”).
77 Rowe, 2008 WL 5412912 at *5.
78 Rhodes, 253 F.R.D. at 370, 374.
79 Id. at 371, 374.
80 Specifically, the proposed class satisfied the requirements of numerosity, commonality (there were several issues of law and fact common to all class members), typicality ("all plaintiffs’ medical monitoring claims arise from the same course of conduct by DuPont and are based on the same legal theory"), and adequacy of representation. Rowe, 2008 WL 5412912 at *6-9.
81 Id. at *20 ("The presence of so many individualized issues precludes a finding of cohesiveness, which renders certification under 23(b)(2) inappropiate."). Certification was also found inappropriate under Rule 23(b)(1) (without class certification, there was “no danger of DuPont being exposed to conflicting obligations in terms of Plaintiffs’ medical monitoring claims”) and Rule 23(b)(3) (because the common questions do not predominate, a class action would not be a superior means of adjudicating the case). Id. at *10, *22.
82 See id. at *15. ("The reality is that the element of significant exposure is fraught with individualized issues [and the same issues] preclude a class-wide finding of increased risk of disease."). See also Rhodes, 253 F.R.D. at 375–79.
to prove these factors failed.\textsuperscript{83} Even if the few studies showing a weak positive correlation between PFOA exposure and human health effects were fully credited, plaintiffs still would need to prove their own individual risks and need for monitoring, based on the actual level of PFOA in their blood and their own personal health histories.\textsuperscript{84}

Another tactic in environmental exposure cases may be to allege property damage. After class action status was denied and health claims were stripped from their case, several Minnesota plaintiffs pursued their suit against 3M, which has not made perfluorochemicals since 2002, by alleging that PFCs in their water had decreased the property value of their homes.\textsuperscript{85} The jury refused to award damages.\textsuperscript{86}

E. Conclusion

PFOA contamination suits likely will fail unless proven deleterious health effects to humans, at exposure levels present in the plaintiffs, are directly linked to its existence in the environment. The scientific evidence does not yet support such conclusions. Studies performed on laboratory animals are of limited use. Existing environmental health risk assessments are based on these studies — as well as questionable assumptions — and they provide only a highly conservative, protective level designed for the most sensitive members of a population. This may be a suitable method for discerning a public health concern, but it is insufficient and inapt for determining the individualized risk or actual harm needed for litigation. Presently available human studies have yielded no conclusive evidence of a direct health risk, and those that posit such risks remain uncorroborated. Finally, even if a direct link between PFOA exposure and disease risk could be ascertained as a general matter, every individual plaintiff must demonstrate that she bears such a risk herself and that her PFOA exposure derives from a specific, liable source.\textsuperscript{87}

\textsuperscript{83} See Rowe, 2008 WL 5412912 at *15 ("[T]he risk assessment method . . . establishes nothing more than an assumption of common exposure. . . . [T]he underlying assumptions are not necessarily true for all class members—indeed, they are undoubtedly false.").

\textsuperscript{84} See Rhodes, 253 F.R.D. at 379 (Even if the court gave "full credence" to the epidemiological studies offered by plaintiff’s expert, "the aggregate evidence would only show that C-8 generally causes some human diseases; it does not show that the specific exposure in this case proximately caused a significantly increased risk of disease for each proposed class member.").

\textsuperscript{85} See Bob Shaw, Minnesota: 3M Water Suit Goes to Trial Trimmed, PIONEER PRESS, May 4, 2009, available at http://www2.fluoridealert.org/Pollution/Perfluorinated-chemicals/Minnesota-3M-water-suit-goes-to-trial-trimmed (last visited Sept. 8, 2009). A state survey had reported that, "[d]espite decades of exposure to the PFCs, residents showed no increases in disease or death compared with metro-area norms." Id.


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