

TOXIC AND HAZARDOUS SUBSTANCES LITIGATION

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IN THIS ISSUE

The authors provide an update to the October 2009 Defense Counsel Journal article on science and regulation concerning perfluorooctanoic acid (“PFOA”). They review a recent study that attempts to link blood concentration of PFOA with negative health effects, specifically delayed onset of puberty in children.

PFOA Update



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In the October 2009 *Defense Counsel Journal*, we wrote about science and regulation concerning perfluorooctanoic acid (“PFOA”) and discussed implications for litigation.¹ This update will review a recent study that attempts to link blood concentration of PFOA with negative health effects, specifically delayed onset of puberty in children.² The continuing inability to pinpoint a specific source of PFOA exposure and the difficulty for prospective plaintiffs to translate questionable and inconclusive science into successful litigation will also be discussed.

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,”³ 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.⁴ In 2006, the eight major PFOA manufacturers signed onto the EPA’s global stewardship program, whereby they agreed to “commit to working toward the elimination of [PFOA and precursor chemicals that can break down into PFOA] from emissions and products by 2015.”⁵ Despite the phase-out plan, studies continue to search for a link between PFOA concentration in human blood and various negative health effects,⁶ and litigation against PFOA manufacturers persists.

B. C8 Science Panel Study

One source of studies on the potential effects of PFOA on the human population is the C8 Science Panel, which was formed as part of a class action settlement with DuPont over the chemical releases from its Washington Works plant in West Virginia.⁷ The Panel is made up of three epidemiologists who collect data “on health status and C8 exposure in the Mid-Ohio Valley Communities [that were] potentially affected” by the release of PFOA from the DuPont plant.⁸

On September 30, 2010, the Panel released a “status report” summarizing the findings of “a statistical analysis of the relationship between levels of [PFOA] and perfluorooctane

¹ Bruce J. Berger, *The Trouble with PFOA: Testing, Regulation, and Science Concerning Perfluorooctanoic Acid and Implications for Future Litigation*, 76 Def. Couns. J. 460 (2009).

² The C8 Science Panel, *Status Report: Patterns of Age of Puberty Among Children in the Mid-Ohio Valley in Relation to Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)*, Sept. 30, 2010, http://www.c8sciencepanel.org/pdfs/Status_Report_C8_and_puberty_27Sept2010.pdf [hereinafter “Status Report”].

³ Lab Science News: PFOA and Related Compounds, <http://www.caslab.com/News/pfoa-and-related-compounds.html> (last visited Nov. 29, 2010).

⁴ See U.S. Environmental Protection Agency website, at <http://www.epa.gov/oppt/pfoa/index.html> (last visited Nov. 29, 2010).

⁵ U.S. Environmental Protection Agency 2010/2015 PFOA Stewardship Program website, <http://www.epa.gov/oppt/pfoa/pubs/stewardship/> (last visited Nov. 29, 2010).

⁶ See, e.g., Stephanie J. Frisbee, et al., *Perfluorooctanoic Acid, Perfluorooctanesulfonate, and Serum Lipids in Children and Adolescents*, Arch. Pediatr. Adolesc. Med. 2010; 164(9): 860-869 (finding that “observed associations between increasing PFOA and PFOS and elevated total cholesterol... warrants further study”).

⁷ See www.c8sciencepanel.org.

⁸ *Id.*

sulfonate (PFOS)⁹ measured in the blood serum of the children who participated in the C8 Health Project, and puberty.”¹⁰ The researchers “examined data for 3076 boys and 2931 girls” who had been “residents for at least a year in the six water districts which had been contaminated with PFOA” and who had been aged 8-18 at the time they participated in the C8 Health Project survey in 2005-2006.¹¹ By comparing the level of PFOA and PFOS in the participants’ blood with the time at which they reached puberty, the researchers found, for boys, “a clear relationship of reduced odds of having reached puberty with increasing PFOS . . . but not PFOA,” and an association for girls between “higher exposure to either PFOA or PFOS” and “reduced odds of having reached puberty.”¹²

Several news articles trumpeted the Panel’s status report as linking PFOA exposure to delayed puberty.¹³ There are various reasons, however, for viewing these results with caution. First, the status report is just that – a brief summary of a study reported on the C8 Science Panel’s website. Although the study authors intend to submit their findings “to a peer-reviewed scientific journal,”¹⁴ they have not yet done so, and thus their results have not been either vetted or verified. Second, the

methodology of the study does not lend itself to consistent and reliable results. For one thing, it cannot be replicated, as the researchers did not conduct a controlled scientific study; they merely reviewed data gathered from the individuals “who consented to be in the Science Panel studies.”¹⁵ For another thing, they based their findings on both an *estimated* average “chance of reaching puberty” and, for girls, on *self-reported* onset of menarche.¹⁶ Most importantly from a legal standpoint, the Panel reported only a “relationship” or an association, which is far from finding the causation needed to determine liability. Finally, previous studies on the relation between PFOA and PFOS and the onset of puberty have reported contrasting results.¹⁷

Indeed, the Panel itself recognized that “[c]aution is needed in interpreting these results”¹⁸ because a correlation does not

⁹ PFOS is a related polyfluoroalkyl chemical, or PFC.

¹⁰ Status Report, *supra* note 2.

¹¹ *Id.*

¹² *Id.*

¹³ See, e.g., Ken Ward, Jr., *Breaking News: C8 Panel Finds Exposure Could Change the Age at Which Children Reach Puberty*, The Charleston Gazette, Sept. 30, 2010, <http://blogs.wvgazette.com/watchdog/2010/09/30/breaking-news-c8-panel-finds-exposure-could-change-the-age-at-which-children-reach-puberty/>; Jonathan Starkey, *Study Links DuPont Chemical To Delayed Puberty*, The News Journal, Oct. 1, 2010, <http://blogs.delawareonline.com/delawareinc/2010/10/01/study-links-dupont-chemical-to-delayed-puberty/>.

¹⁴ Status Report, *supra* note 2.

¹⁵ *Id.*

¹⁶ The researchers themselves acknowledged a potential problem with the fact that “menarche was self-reported.” *Id.*

¹⁷ See Ward, *supra* note 13 (noting a University of Cincinnati study that “found an *earlier* onset of puberty – measured in the study by breast maturation – in girls exposed to PFOA” and a study in Environment International which “reported no impact from PFOS exposure on puberty age in young girls, as measured by the onset of the menstrual cycle”) (emphasis added); see also Christensen KY, *Exposure to Polyfluoroalkyl Chemicals During Pregnancy is Not Associated with Offspring Age at Menarche in a Contemporary British Cohort*, 37 Environ. Intl. 129-35, Epub. Sept. 16, 2010 (concluding that “study participants had nearly ubiquitous exposure to most PFCs examined, but PFC exposure did not appear to be associated with altered age at menarche of their offspring”); Susan Pinney, et al., *Perfluorooctanoic Acid (PFOA) and Pubertal Maturation in Young Girls*, <http://isee.conference-services.net/reports/template/onetextabstract.xml?xsl=template/onetextabstract.xml&conferenceID=1651&abstractID=312130>.

¹⁸ Status Report, *supra* note 2.

indicate causation. As the Panel noted, “it may be that growth changes associated with puberty lead to changes in PFOA and PFOS blood levels, rather than these compounds having any effect on age at puberty.”¹⁹ In addition, “other factors might be leading to both changes in the age of puberty and differences in PFC uptake.”²⁰ The Panel took into account other potential explanations, such as “smoking, alcohol intake, obesity and household family income,” and did not find any difference in the results, but acknowledged that “there may be other such causes, and further research is needed.”²¹

C. Identifying a Source

Although the C8 Science Panel was formed to study the potential health effects of PFOA on a population believed to be particularly affected by the emissions from DuPont’s Washington Works plant, it cannot determine if any PFOA found in that population actually originated from the plant. Indeed, PFOS, which the Panel found in the Washington Works-area residents and correlated with delayed puberty, was not even used by DuPont.²²

Moreover, the ubiquitous nature of PFCs makes determining the source of an individual’s exposure a near-impossible task. For example, researchers have recently concluded that exposure to polyfluoroalkyl

phosphate esters (PAPs), which can be metabolized into PFCAs, including PFOA, and are “used in food-contact paper packaging and have been observed in human sera . . . should be considered as a significant indirect source of human PFCA contamination.”²³ The study noted that, “due to the long human serum half-life of PFOA, biotransformation of even low-level diPAP exposure could over time result in *significant* exposure to PFOA.”²⁴ Thus, as several news articles warned, “[c]hemicals applied to fast-food wrappers and microwave popcorn bags are migrating into the food and being ingested by consumers.”²⁵ Therefore, even if the C8 Science Panel could definitively link delayed

¹⁹ *Id.*

²⁰ Jeffrey Saulton, *C8 Panel Releases Puberty Study*, Parkersburg News and Sentinel, Oct. 1, 2010, <http://www.newsandsentinel.com/page/content.detail/id/539666/C8-panel-releases-puberty-study.html?nav=5061> (quoting Dr. Tony Fletcher, member of the C8 Science Panel).

²¹ *Id.*

²² See Todd Baucher, *Delayed Puberty*, WTAP News, Sept. 30, 2010, <http://www.wtap.com/home/headlines/104112824.html> (noting that PFOS “is not manufactured or used by DuPont”); Starkey, *supra* note 13.

²³ Jessica C. D’eon & Scott A. Mabury, *Exploring Indirect Sources of Human Exposure to Perfluoroalkyl Carboxylates (PFCAs): Evaluating Uptake, Elimination and Biotransformation of Polyfluoroalkyl Phosphate Esters (PAPs) in the Rat*, Environ. Health Perspect., Nov. 8, 2010, <http://ehp03.niehs.nih.gov/article/info%3Adoi%2F10.1289%2Fehp.1002409>.

²⁴ *Id.* (emphasis added). This study’s results must also be viewed with caution, as the researchers dosed rats “intravenously or by oral gavage” with various mixtures of “monoPAP or diPAP chain lengths” and extrapolated from the “bioavailability” of the diPAPs to conclude that humans could be ingesting the chemicals applied to fast-food wrappers. *Id.* We have previously discussed the dangers of attempting to draw conclusions about human health from animal studies. See, e.g., Berger, *supra* note 1. Regardless of the study’s faults, however, it lends credence to the notion that no one can definitively state where any person’s possible PFOA (or PFOS) exposure originated.

²⁵ *Food Wrapper Chemicals May Leach Into Food*, United Press Int’l, Inc., Nov. 8, 2010, http://www.upi.com/Health_News/2010/11/08/Food-wrapper-chemicals-may-leach-into-food/UPI-62541289275374/; see also *Chemicals in Fast Food Wrappers Show Up in Human Blood*, Env’t News Svc., Nov. 8, 2010, <http://www.ens-newswire.com/ens/nov2010/2010-11-08-01.html>; *Dangerous Chemicals in Food Wrappers Likely Migrating to Humans*, Science Daily, Nov. 9, 2010, http://www.sciencedaily.com/releases/2010/11/101108140917.htm?sms_ss=digg&at_xt=4ce1faba269ded64.0

puberty to increased blood levels of PFCs, it could not determine the exact source of the children's exposure.

D. Recent Litigation

Although it is difficult – if not impossible – to pin purported PFOA exposure on any one potential source, and the effects of PFOA on human health are far from certain,²⁶ litigation over PFOA emissions still abounds.²⁷ This is so even though “[b]etter control of emissions and the voluntary phase out of PFOA use in the United States” have led to a decline in the overall levels of PFCs in human blood, both in the Washington Works-plant area and across the nation.²⁸

Recently, a couple of local water suppliers have sued PFOA and PFOS manufacturers for “contaminating” their wells.²⁹ A Resource

Conservation and Recovery Act (“RCRA”) citizen suit filed by an Ohio water association whose well fields are located directly across the Ohio River from DuPont's Washington Works plant survived a motion to stay proceedings pursuant to the doctrine of “primary jurisdiction.”³⁰ DuPont had argued that “EPA is actively engaged in addressing the alleged problems with respect to the release of PFOA from the Washington Works Plant,” and thus the claims in the case involved “issues within the ‘special competence of an administrative body.’”³¹ The court, however, found itself well suited to determine the relevant issues in the case, especially considering that Congress specifically authorized citizen suits in the RCRA statute.³²

Although a court of law may be a suitable forum for determining whether PFOA presents a danger to health or the environment, any particular plaintiff may not be able to present sufficient evidence of personal harm to warrant a lawsuit. For example, a Florida water supplier's claims for strict liability, nuisance, trespass, and negligence were all dismissed on summary judgment for “fail[ure] to demonstrate an injury in fact for the purposes of Article III

²⁶ See, e.g., 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.”). Efforts, however, continue on various fronts. For example, on November 16, 2010, the EPA issued a new list of 134 chemicals, including PFOA and PFOS, that will be screened for their potential to be endocrine disruptors. See *EPA Adds C8 to Endocrine Disruptor Screening List*, The Charleston Gazette, Nov. 16, 2010, <http://blogs.wvgazette.com/watchdog/category/c8/>; see also U.S. Environmental Protection Agency, *News Release: EPA to Expand Chemicals Testing for Endocrine Disruption*, Nov. 16, 2010, <http://yosemite.epa.gov/opa/admpress.nsf/eef922a687433c85257359003f5340/5f77e9903c4e2e48852577dd005bc7ce!OpenDocument> (“Endocrine disruptors are chemicals that interact with and possibly disrupt the hormones produced or secreted by the human or animal endocrine system, which regulates growth, metabolism and reproduction.”).

²⁷ See, e.g., *Ex parte 3M Co., Inc.*, 42 So.3d 1228 (Ala. 2010) (issuing writs of mandamus regarding venue issues in suits alleging negligent dumping of “biosolids,” including PFOA, on farmland, grasslands, and in water supplies).

²⁸ Saulton, *supra* note 20.

²⁹ See *Emerald Coast Utils. Auth. v. 3M Co.*, No. 3:09cv361/MCR/MD, 2010 U.S. Dist. LEXIS 103000

(N.D. Fla., Sept. 29, 2010); *The Little Hocking Water Ass'n, Inc. v. E.I. DuPont De Nemours & Co.*, No. 2:09-CV-1081, 2010 U.S. Dist. LEXIS 89175 (S.D. Ohio Aug. 30, 2010).

³⁰ *The Little Hocking Water Ass'n, Inc.*, 2010 U.S. Dist. LEXIS 89175.

³¹ *Id.* at *7-8.

³² See *id.* at *14 (“[A]lthough Defendant has entered into a Consent Order with the EPA regarding the presence of PFOA in drinking water, the question of whether PFOA is a hazardous waste and whether it presents an imminent and substantial endangerment to health or the environment can properly be determined by this Court, especially in view of the authority granted by Congress for citizens to pursue such claims.”).

standing.”³³ Although the plaintiff sought damages for costs and expenses for having to test, treat, and monitor its water for the presence of defendants’ chemicals, the court noted that the plaintiff’s “water supply has never been contaminated above any EPA advisory level” due to PFOA and PFOS, and no expert testified that the “existing levels were harmful.”³⁴ Moreover, there was “no reliable evidence in [the] record that these chemicals are associated with harm in humans,” and even assuming such harm, there was no evidence showing “at what level they produce such harm and, further, how that level relates to levels in this case.”³⁵

E. Conclusion

Despite ever more studies and continuing litigation, plaintiffs still face an upward struggle in trying to claim damages based on alleged PFOA exposure. Although the C8 Science Panel – and others – persists in looking for links between PFOA and human ailments, there is still no reliable evidence linking levels of PFOA in humans to demonstrable negative health effects, much less any evidence that proves actual causation. Even if such effects could be shown, any plaintiff would still have to demonstrate individualized harm caused by a specific, identifiable source. Without such evidence, there can be no liability.

³³ *Emerald Coast Utils. Auth.*, 2010 U.S. Dist. LEXIS 103000, at *2-3.

³⁴ *Id.* at *32.

³⁵ *Id.* at *43.



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