

Burdick v Tonoga, Inc.
2019 NY Slip Op 35265(U)
November 15, 2019
Supreme Court, Rensselaer County
Docket Number: Index No. 253835
Judge: Patrick J. McGrath
Cases posted with a "30000" identifier, i.e., 2013 NY Slip Op <u>30001</u> (U), are republished from various New York State and local government sources, including the New York State Unified Court System's eCourts Service.
This opinion is uncorrected and not selected for official publication.

At an IAS Term of the Supreme Court of the State of New York, held in and for the County of Rensselaer, in the City of Troy, New York on the 9th day of August 2019

PRESENT: HON. PATRICK J. McGRATH
Justice of the Supreme Court

STATE OF NEW YORK
SUPREME COURT COUNTY OF RENSSELAER

**JAY BURDICK, CONNIE PLOUFFE, EDWARD PLOUFFE,
FRANK SEYMOUR, SUZANNE SEYMOUR, AND EMILY MARPE,
as parent and natural guardian of E.B., an infant, and
G.Y., and infant, JACQUELINE MONETTE, WILLIAM SHARPE,
EDWARD PERROTTI-SOUSIS, MARK DENUÉ, and
MEGAN DUNN, individually, and on behalf of all similarly situated,**

Plaintiffs,

DECISION AND ORDER
Index No. 253835

- against -

TONOGA, INC. (d/b/a TACONIC),

Defendant.

APPEARANCES: FARACI LANGE, LLP
WEITZ & LUXENBERG, PC
Co-Lead Class Counsel

GREENBERG TRAUERIG, LLP
HOLLINGSWORTH, LLP
Attorneys for the Defendant

RECEIVED
RENSSELAER COUNTY CLERK
2019 NOV 25 AM 11:39
FRANK J MEROLA

McGRATH, PATRICK J., J.S.C.

This case stems from the contamination of groundwater in the Town of Petersburgh, New York with perfluorooctanoic acid (hereinafter "PFOA"). In a decision and order dated July 3, 2018, this Court granted plaintiffs' motion to certify four (4) classes. Three of those classes allege harms related to property damage and nuisance stemming from contamination of class members' property and drinking water with PFOA. The fourth class seeks the establishment of a class-wide medical

monitoring program to provide medical surveillance to class members exposed to PFOA via the municipal water supply or contaminated wells within a seven mile radius of defendant's facility. Plaintiffs assert causes of action that sound in negligence and strict liability claims related to property, negligence and strict liability claims related to PFOA ingestion, private nuisance and trespass.

Defendant brings what it characterizes as a *Frye* motion to preclude plaintiffs' expert epidemiologist Dr. David Savitz from testifying concerning the casual connection between PFOA exposure and certain health conditions. Plaintiffs challenge this characterization, arguing that the motion should not be considered under a *Frye* analysis and at most, constitutes subject matter for cross-examination or a *motion in limine*. Defendant has submitted a Reply.

The Frye Test

In determining the admissibility of expert testimony, New York follows the rule of Frye v United States, 293 F 1013 (1923), specifically, "that expert testimony based on scientific principles or procedures is admissible but only after a principle or procedure has 'gained general acceptance' in its specified field." *See also* People v Wesley, 83 NY2d 417, 422 (1994); People v Wernick, 89 NY2d 111, 115 (1996). "[G]eneral acceptance does not necessarily mean that a majority of the scientists involved subscribe to the conclusion. Rather it means that those espousing the theory or opinion have followed generally accepted scientific principles and methodology in evaluating clinical data to reach their conclusions." Zito v Zabarsky, 28 AD3d at 44, *quoting* Beck v Warner-Lambert Co., 2002 NY Slip Op 40431[U], *6-7 (Sup. Ct., New York County, 2002). "The Frye 'general acceptance' test is intended to protect[]juries from being misled by expert opinions that may be couched in formidable scientific terminology but that are based on fanciful theories." Styles v General Motors Corp., 20 AD3d 338 (1st Dept. 2005) (Catterson, J., concur) [internal quotation marks omitted].

A *Frye* inquiry is directed at the basis for the expert's opinion and does not examine whether the expert's conclusion is sound. "*Frye* is not concerned with the reliability of a certain expert's conclusions, but instead with 'whether the experts' deductions are based on principles that are sufficiently established to have gained general acceptance as reliable.'" Nonnon v City of New York, 32 AD3d 91, 103 (1st Dept. 2006), *quoting* Marsh v Smyth, 12 AD3d 307, 308 [2004]. Put another way, "[t]he court's job is not to decide who is right and who is wrong, but rather to decide whether or not there is sufficient scientific support for the expert's theory." Gallegos v Elite Model Mgmt. Corp., 195 Misc 2d 223, 225[2003]. "The appropriate question for the court at ... a [Frye] hearing is the somewhat limited question of whether the proffered expert opinion properly relates existing data, studies or literature to the plaintiff's situation, or whether, instead, it is 'connected to existing data only by the ipse dixit of the expert.'" Marsh v Smyth, 12 AD3d 307, 312 [1st Dept. 2004] (Saxe, J., concur.) *quoting* General Elec. Co. v Joiner, 522 US 136, 146 (1997).

Both parties cite two Court of Appeals cases concerning expert testimony in toxic tort cases.

First, in Parker v Mobil Oil Corp., 7 NY3d 434 (2006), the plaintiff alleged that he developed acute myelogenous leukemia (AML) from 17 years of occupational exposure to gasoline containing benzene while he worked as a gas station attendant. The plaintiff intended to call causation experts without presenting evidence of the concentration level of benzene in the gasoline. The experts employed no other methodology to establish the plaintiff's benzene exposure level. The defendants moved to preclude the plaintiff's experts under *Frye* and for summary judgment since the plaintiff's case would be meritless without expert testimony to establish causation. The trial court denied the defendants' motions and the defendants appealed. The Second Department reversed the trial court's decision and granted summary judgment to the defendants. The Court of Appeals ruled that an expert's causation opinion must establish three elements: (1) the plaintiff's level of exposure to the relevant toxin; (2) general causation, such that the toxin could in fact cause the illness and that the level of exposure would engender such illness (dose-response relationship); and (3) specific causation – the likelihood that the specific toxin did cause the plaintiff's injury. Failure to satisfy any of these elements would render an expert opinion inadmissible. However, the Court found that experts could establish chemical exposure causation in many ways, provided that whatever methods an expert uses to establish causation are generally accepted in the scientific community. The Court upheld the use of extrapolation methods such as differential diagnosis, mathematical modeling, and qualitative reasoning for causation opinions.

However, the Court did not decide *Parker* based on a *Frye* analysis; rather the Court framed the issue as one of foundation. The Court distinguished *Frye* challenges of new or novel expert theories from other reliability challenges to the admissibility of expert opinions: "The *Frye* inquiry is separate and distinct from the admissibility question applied to all evidence—whether there is a proper foundation—to determine whether the accepted methods were appropriately employed in a particular case ... The focus moves from the general reliability concerns of *Frye* to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial." *Id.* at 447; *see also Buchholz v Trump 767 Fifth Ave., LLC*, 5 NY3d 1, 9 (2005) (New York law does not permit the court to accept assertions that are "speculative or unsupported by any evidentiary foundation."). The Court in *Parker* noted that the foundation "should not include a determination of the court that such evidence is true. That function should be left to the jury." *Id.* at 425.

The Court of Appeals found that the Second Department properly excluded the opinion of the plaintiff's first expert, a toxicologist and epidemiologist, because the expert failed to demonstrate that exposure to benzene as a component of gasoline caused the plaintiff's AML. This expert's citation to an epidemiological study of refinery workers was insufficient to establish causation. While claiming that the plaintiff had "far more exposure to benzene" than the refinery workers, the expert did not establish the worker's exposure level or how the plaintiff exceeded it. Likewise, the plaintiff's second expert, a medical doctor specializing in occupational medicine and epidemiology, failed to back up his claims that the plaintiff frequently was exposed to excessive quantities of both liquid and vapor gasoline. Even though "an expert is not required to pinpoint exposure with complete precision," the expert's statement could not "be characterized as a scientific expression of . . . exposure level" at all. Both experts failed to look at the plaintiff's alleged exposure to benzene as

a component of gasoline (as opposed to benzene either by itself or in some other compound). Neither expert cited to studies to establish a relationship between gasoline exposure and AML. Thus, their opinions lacked foundation and it was right to exclude them.

In Cornell v. 360 W. 51st St. Realty, LLC, 22 NY3d762 (2014), the Court of Appeals applied the test established in Parker, which required an expert's causation opinion to establish both general causation and specific causation in complex product liability and toxic tort matters. In Cornell, the plaintiff sued for bodily injury she allegedly suffered from exposure to mold. She claimed the source of the mold was construction work performed in the basement of her apartment building. Her expert offered the opinion that there was an "association" between the mold environment and the plaintiff's symptoms, which included dizziness, headaches, rashes and respiratory problems. The Court rejected plaintiff's expert's opinion, noting that it failed to satisfy the general causation and specific causation requirements set out in Parker. The defendant established a prima facie case as to general causation establishing generally accepted standards within the relevant community of scientists and scientific organizations, that exposure to mold caused disease in three ways, none of which were claimed by the plaintiff. Similarly, the Court held that plaintiff did not establish specific causation because Plaintiff's expert had failed to make any effort to quantify plaintiff's exposure to mold, or to refute the opinion of defendants' expert that the mold was present at concentrations and distribution to be expected in a typical home.

Defendant primarily relies on that portion of the Cornell decision wherein the Court noted that "Frye focuses on principles and methodology, but these are not entirely distinct from one another... Thus, even though the expert is using reliable principles and methods and is extrapolating from reliable data, a court may exclude the expert's opinion if there is simply too great an analytical gap between the data and the opinion proffered." Cornell, *supra* at 780-81, quoting General Electric Co. v Joiner, 522 US 136, 146 (1997). The Court noted that it had previously "expressed this precept in terms of the general foundation inquiry applicable to all evidence." Cornell, *supra* at 781 citing People v. Wesley, 83 NY2d 417, 422 (1994) and Parker, *supra* at 447. I

Dr. David Savitz

David A. Savitz, Ph.D. is a Professor of Epidemiology at the School of Public Health and Professor of Obstetrics and Gynecology and Pediatrics at the Warren Alpert Medical School of Brown University in Providence, Rhode Island. He is one of three epidemiologists chosen to serve on the C8 Science Panel to evaluate the probable causal link between exposure to PFOA and the development of certain diseases. He has published eleven scientific papers in the peer-reviewed literature regarding PFOA health effects, most focused on health effects related to pregnancy and children. He served as a Peer Reviewer of the June 2018 Draft Toxicological Profile for Perfluoroalkyls (a class of chemicals that includes PFOA) by the United States Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. He chaired a scientific panel to advise the State of Michigan Science Advisory Panel on addressing the health and environmental concerns related to perfluoroalkyl substances (PFAS) exposure and provided a report entitled "Scientific Evidence and Recommendations for Managing PFAS Contamination in

Michigan.”

Dr. Savitz states that epidemiology is the study of the patterns and determinants of disease in human populations, seeking an understanding of the causes of disease in order to determine needed actions to improve the health of the public. Epidemiologists conduct and review studies of populations first to determine whether there is evidence indicative of a statistical association between some potentially causative agent and a human illness or condition. This typically requires comparing the frequency of disease in a group that has relatively elevated exposure to the frequency of disease in a group that is unexposed or has a lower level of exposure. When it is determined that those who are exposed have an elevated risk of disease relative to those who are not, he conducts analyses to make an informed judgment regarding whether it is likely that the exposure has in fact caused an elevated risk of disease. While this cannot be proven with 100% certainty, the field of epidemiology has developed clear principles and methodologic tools to make a reasoned, scientifically grounded judgment. By considering alternative explanations of the association, including biases and random error, and conducting analyses to address those alternative explanations, the case for a causal interpretation can be strengthened or weakened, depending on what is found.

He states that scientific certainty of causality is difficult to establish with any toxicants, epidemiologists are able to make informed use of available data to address questions of causality. By considering the body of scientific evidence and interpreting it with an appreciation of the underlying methodologic strengths and limitations, reliable judgments can be made, including when a causal link is more likely than not to be present.

The C8 Health Project concerned DuPont's West Virginia Washington Works Plant in southwest Parkersburg, which released PFOA into the air and Ohio River from the 1950s until the early 2000s. C8, the name given to perfluorooctanoic acid (PFOA), reached drinking water supplies by entering the groundwater and was detected in six water districts near the DuPont plant in 2002. A class action lawsuit brought by the communities against DuPont resulted in a Settlement Agreement. As part of that settlement, Brookmar Inc., an independent company, was set up and conducted a yearlong survey (August 2005 - July 2006) called the C8 Health Project. The C8 Health Project gathered information through interviews and questionnaires and collected blood samples from about 69,000 people living near the Washington Works plant in West Virginia. The settlement also established that a group of public health scientists would assess whether or not there is a probable link between PFOA exposure and disease in the community. The members of the Science Panel were jointly selected by the lawyers for the community and DuPont. The C8 Science Panel consisted of Dr. Tony Fletcher of the London School of Hygiene and Tropical Medicine, Dr. Kyle Steenland of Emory University in Atlanta and Dr. Savitz. All were chosen because of their long experience in designing and carrying out environmental health studies and the view of the parties in the settlement that they would be able to objectively generate and evaluate the evidence.

Dr. Savitz states that the C8 Health Project was unique in that it enabled the study of nearly 70,000 people whose exposure to PFOA was markedly elevated in some cases and could be reconstructed given the well-defined source of contamination.

As per the settlement, the panel was required to make a judgment regarding the evidence of a causal link between PFOA and the risk of developing a disease based on health research carried out in the Mid-Ohio Valley population, as well as other published scientific research. For each health problem of concern, the panel first generated the research results, and then in a separate activity, evaluated all the evidence to make a judgment regarding whether or not there is a probable link between PFOA exposure and that illness. The panel's interpretation and judgment regarding the concept of "probable link" was based on the potential for a causal influence of PFOA, taking into account whether observed associations were more likely to be due to some bias or artifact versus due to a causal effect of PFOA. When the panel found that a causal effect was more likely to be responsible, even if only slightly more likely, they determined that a probable link was present. As a result of the above analyses, the C8 Science Panel came to the conclusion that there was a probable causal link between PFOA exposure and six human diseases and conditions: kidney cancer, testicular cancer, ulcerative colitis, thyroid disease, hypercholesterolemia and pregnancy induced hypertension (preeclampsia).

Dr. Savitz notes that the C8 Science Panel was instructed to focus only on disease, not on changes in biomarkers that could potentially be used to predict future disease. As a result, the panel analyzed whether PFOA caused the recognized condition of "hypercholesterolemia" but not whether it generally resulted in elevation of cholesterol levels that did not yet rise to the level required to diagnose hypercholesterolemia. Similarly, the C8 Science Panel did not analyze whether elevated liver enzymes levels or uric acid levels were associated with PFOA exposure. However, Dr. Savitz states that many other researchers have addressed these associations and have concluded that there is likely to be a causal link to these elevated biomarkers as well.

With respect to thyroid disease, Dr. Savitz states that he determined that there is support in the scientific literature for a causal link between cumulative PFOA exposure and thyroid disease, specifically hyperthyroidism and hypothyroidism. This link is supported by the C8 Health Project, with some support from the analysis of National Health and Nutrition Examination Survey (NHANES) data. The C8 Health Project found a clear positive association of PFOA with hypothyroidism in men and a somewhat weaker association with hyperthyroidism in men. For hypothyroidism in women, there was a clear dose-response gradient, with the first indication of an increased risk in the third quintile of exposure which became larger in the higher exposure groups. For hyperthyroidism in woman, a dose-response relationship was found with an increase in incidence being found starting in the second quintile and continuing to rise with increasing exposure. For prospective cases (diagnosed after PFOA was measured), hypothyroidism among men increased starting in the third quintile and showed a consistently increasing risk with increasing exposure above that level, rising to a two-fold increased risk in the uppermost quintile.

Dr. Savitz opines that increasing levels of PFOA are associated with increased risk of developing ulcerative colitis based on a series of studies conducted by the C8 Science Panel. The C8 study indicated a "clear dose-response gradient of increasing risk with increasing cumulative exposure. Using a cumulative exposure measure of nanograms per milliliter (ng/mL), quartiles of the distribution were examined and each of the upper three quartiles was compared to the lowest.

Exposures above 158 ng/ml were associated with increasing risk and continued to rise with more elevated exposure.

He opines that there is consistent evidence of a strong association and dose-response relationship between PFOA exposure and kidney cancer. This opinion is based on three different studies all conducted as part of the C8 Science Panel research in the Ohio/West Virginia area. The studies consist of a geographic study by Vieira et al., an occupational study of mortality of DuPont workers by Steenland and Woskie, and a cancer incidence study that combined occupational and community cohorts by Barry, et. al.

He states that the epidemiological literature generated by the C8 Science Panel supports an association between PFOA exposure and an increased risk of developing testicular cancer. He relies on two studies that address PFOA and testicular cancer, one a geographic study in Ohio and West Virginia, and the other the study of the combined community and occupational cohort by the C8 Science Panel. The community and occupational cohort study included 32 reported incident cases of testicular cancer of which 19 were validated. Across the range of exposure, there was an increased risk of testicular cancer per log unit change in cumulative PFOA and across quartiles of exposure. Similar results were found with a 10-year lag.

Dr. Savitz opines that there "is rather clear and convincing evidence" that higher levels of PFOA are associated with higher levels of serum uric acid and that it is probable that exposure to PFOA is capable of causing increased uric acid levels. This is seen in the analyses of the C8 Health Project participants, with notable increases in average serum uric acid levels and the risk of being above the cut point defining hyperuricemia (significantly elevated serum uric acid) across the spectrum of PFOA exposure. The increase in risk was especially strong in the lower range and reflects somewhat of a ceiling effect with less of an increase across the highest levels. Evidence of this association was corroborated in studies in children and adults in other populations.

Dr. Savitz notes that a significant number of studies have found clear associations between PFOA exposure and both total and LDL cholesterol. It is his opinion based on these studies that it is probable that exposure to PFOA causes an increase in both total and LDL cholesterol. Using cross-sectional data from the C8 Health Project, he notes that Steenland et al. found clear evidence that higher levels of PFOA are associated with greater risk of hypercholesterolemia, with odds ratios across exposure quartiles and with a similar pattern for LDL cholesterol. In an analysis of the community and worker cohort developed by the C8 Science Panel, Winquist and Steenland again found increased risk of hypercholesterolemia when compared to the lowest quintile. An association with hypercholesterolemia was also found in National Health and Nutrition Examination Survey (NHNES) data where an increased risk of elevated levels of LDL cholesterol was also found. There is a strong empirical basis for concluding that higher levels of PFOA are associated with higher levels of total and LDL cholesterol, and that PFOA is associated with increased risk of hypercholesterolemia. Dr. Savitz acknowledges this is not universal across studies, some of which show no association with either total or LDL cholesterol or both. Again, generalizing across a large body of studies, he opines that the most consistent and compelling association would be with total

cholesterol in part because more studies have addressed this measure. This association is found in adults, children and adolescents, and pregnant women with some consistency. The dose-response gradient shows a rapid increase in total cholesterol in the lower range of PFOA exposure but appears to plateau, with little increased risk as exposure rises further, which may explain some of the inconsistency across studies.

He states that there is support in the scientific literature for an association between PFOA exposure and elevation of at least some liver enzymes in the blood serum, and opines that it is probable that exposure to PFOA is capable of causing an increase in liver enzyme levels in the blood. A substantial number of studies have examined the correlation between serum levels of PFOA and an array of liver enzymes. Those that are most frequently studied include ALT (alanine transferase), ALP (alkaline phosphatase), AST (aspartate aminotransferase), GGT (gamma glutamyl transferase), bilirubin (total and direct), and CCK (cholecystokinin). Many of the studies examine the entire panel of routinely assayed liver enzymes and others do so selectively. Given the large number of enzymes and large number of studies, there are an array of results which are not entirely consistent but with some patterns present. The most consistent finding is an association of PFOA with increased levels of ALT, observed in the C8 Science Panel research, in the National Health and Nutrition Examination Survey, and in some of the occupational studies.

Finally, Dr. Savitz states that there is "some evidence in the published literature for an association between PFOA exposure and the incidence of preeclampsia or pregnancy induced hypertension." He states that the study of the C8 community showed an increased risk for preeclampsia. He notes that another study of this population showed a weak association between PFOA exposure and pregnancy induced hypertension. Based upon these studies, it is his opinion and the collective opinion of the C8 Health Panel that exposure to PFOA is capable of causing preeclampsia and pregnancy induced hypertension.

Dr. Savitz states that there are other health conditions which may reach the threshold of "more probably than not are related to PFOA exposure" in the future, including prostate cancer and ovarian cancer, as well as effects on the immune system but concedes that there is only "limited evidence supporting an association between PFOA exposure and risk of prostate and ovarian cancers" at this point. Further, that while it seems "plausible that there is some increase in infections in relation to PFOA serum levels", "the research does not allow pinpointing of one type or another due to the varying results across studies. It is not even clear at this point whether viral or bacterial infections would be most likely to be affected if there is an effect."

He opines to a reasonable degree of scientific certainty that elevated PFOA exposure increases the risk of the development of certain diseases and conditions referenced above. He states that the question of a lower limit for this effect is not resolved at present but there is evidence that even in the exposure ranges near the background levels, elevated risks may be present. Because PFOA demonstrates adverse biological effects even near "background" levels, evidence does not exist for establishing a level of PFOA exposure below which no negative effects can be assured. While it is true that evidence of increased incidence of disease for some conditions listed above were

only seen in the highest exposed groups, for other outcomes such as elevated cholesterol and ulcerative colitis, increased risks were present in the near-background exposure range. Since a dose-response relationship has emerged for a number of the associated illnesses, what is clear is that as exposure increases above background so does risk of harm.

Dr. Savitz states that because drinking water has only recently become a focus of attention for PFOA contamination and because a testing of both public and private drinking water sources had detected significant levels of PFOA in many locations across the United States, it is "highly likely" that more research will be done that may add to support for an association between PFOA and other adverse human health effects in the future.

Defendants seek to preclude Dr. Savitz from testifying. Defendant provides the affidavit of Linda Dell, also an epidemiologist. She concludes to a reasonable degree of scientific study that the epidemiologic data does not support a conclusion that PFOA *causes* the diseases and conditions as stated by Dr. Savitz, noting that the C8 studies focused on a "more probable than not" standard. She notes that of the 55 diseases (including 21 cancers) or conditions studied, the C8 panel made a "more probable than not" link between PFOA and six diseases or conditions.

Defense counsel notes that the Cancer Incidence Investigation 1995-2014 conducted by the NYS Department of Health for the Village of Hoosick Falls in May of 2017 found no increased incidence of kidney or testicular cancer in the population, with fewer cases of each cancer found than expected. Dr. Savitz states that such information is routinely collected by the state cancer registry and can be used for general surveillance purposes, but is not designed to be nor is it useful for etiologic studies of the potential effect of an environmental toxicant on diseases in the population because 1) there is no direct information on the levels of PFOA in the water over the period that the person lived there or even a basis for estimating cumulative PFOA exposure; 2) there is no information on other potential causes of these cancers that may need to be taken into account to isolate any effect of PFOA, which might mask true associations or generate spurious associations; 3) the numbers of events for the cancers of particular interest are simply too small to be informative.

Counsel for the defendant claims that Dr. Savitz's techniques lack general acceptance in the scientific community, however, defendant's expert does not offer this opinion in support of the motion to preclude.

In reply, Dr. Savitz notes that his approach with the C8 Panel was based upon generally accepted principles practiced in this field and that his opinions regarding the causal link between PFOA exposure and human health effects is not novel or unique but is within the mainstream of opinions in the field. He notes that his opinions and conclusions are also supported by the over one hundred articles in his bibliography as well as the June 2018 Draft Toxicological Profile for Perfluoroalkyls which states: "The available epidemiology studies suggest links between perfluoroalkyl exposure and several health outcomes.", listing hepatic effects, cardiovascular effects, endocrine effects, immune effects, reproductive effects and developmental effects linking PFOA exposure in each of these adverse health outcomes.

Defense counsel argues that the C8 study concerned much higher levels of exposure than have been observed in Petersburg, and that it is “plainly contrary to generally accepted scientific principles to opine that because high levels of PFOA exposure are allegedly associated with certain adverse health outcomes, those same outcomes will occur at lower levels of exposure.” Again, defendant’s expert does not offer this opinion in support of the motion to preclude. Additionally, Dr. Savitz replies that the data from the NYSDOH indicates that there were 478 people tested for PFOA in their blood serum. Of those tested, 398 tested above 1.86 ug/L and 80 tested at or below that level with 8 testing non-detect. He notes that this is not surprising since the NYSDOH testing was made available to anyone that wanted to be tested and was not limited to those whose drinking water source was known to be contaminated with PFOA as was the case in the mid-Ohio Valley. Since these 80 people fall outside the proposed class definition, Dr. Savitz states that they should not be included in the calculation of an average level to compare to the C8 Health Project communities. When only considering the 398 people who meet the class definition, the mean PFOA serum level is actually 41.98 ug/L, which is lower than Little Hocking, OH and Lubeck, WV, very similar to Belpre and Tappers Plain, OH, and higher than Mason County, WV that were part of the C8 Health Project.

Finally, defense counsel argues that Dr. Savitz should not be permitted to testify concerning the results of future research, specifically, that certain health conditions may be linked to PFOA in the future.

As recognized by the Third Department, epidemiology is not novel. Jackson v. Nutmeg Tech., Inc., 43 AD3d 599, 601 (3d Dept. 2007). “[N]umerous courts have held that this field of science is the primary generally accepted methodology for demonstrating a causal relation between a chemical compound and a set of symptoms or a disease.” Nonnon v. City of New York, 32 AD3d 91, 104 (1st Dept. 2006) *citing* Soldo v Sandoz Pharmaceuticals Corp., 244 F Supp 2d 434, 532 (WD Pa 2003), Castillo v E.I. Du Pont De Nemours & Co., Inc., 854 So 2d 1264, 1270 [Fla Sup Ct 2003]; Arnold v Dow Chem. Co., 32 F Supp 2d 584 (EDNY 1999) and Conde v Velsicol Chem. Corp., 804 F Supp 972, 1025-26 [SD Ohio 1992] *affd* 24 F3d 809 (1994). The evidence offered by plaintiffs is comprised of epidemiological data, an established and reliable scientific field based on the gathering of data and the statistical analysis of the information. Ms. Dell's affidavit does not state that Dr. Savitz's conclusions and the conclusions of the C8 Science Panel are not generally accepted in the field of epidemiology or that his methodology in analyzing the various studies was novel or different from the approach epidemiologists are trained to follow in reaching such conclusions. While she addresses general concepts of epidemiology, she does not provide any specific application of these concepts in reaching her contrary conclusions regarding PFOA general causation or assert that Dr. Savitz's opinions are in any way inconsistent with these general concepts. As noted in Parker, where “[t]here is no particular novel methodology at issue for which the Court needs to determine whether there is general acceptance. Thus, the inquiry here is more akin to whether there is an appropriate foundation for the experts' opinions, rather than whether the opinions are admissible under Frye.” Parker, supra at 447. The issue before this court, therefore, is not the general acceptance of epidemiology by the relevant scientific community, but rather the application of these accepted scientific principles.

As the epidemiological testimony does not concern "novel science," *Frye's* concerns are not implicated and no pretrial *Frye* hearing is required.

Relying on specific/general causation test set forth in *Parker*, defense counsel argues that Dr. Savitz should still be precluded from testifying as his opinions only establish association rather than causation. Unlike the plaintiffs in *Parker* and *Cornell*, the present plaintiffs do not allege that PFOA has caused any illness, so the general/specific causation test set forth in *Parker* is simply not applicable.

Rather than seeking direct damages from manifest illness, plaintiffs are seeking medical monitoring as consequential damages to their ordinary negligence and property damage claims. Therefore, this Court's analysis shifts from *Parker* and *Cornell* to *Caronia v Philip Morris USA, Inc.*, 22 NY3d 439, 446 (2013), *Abusio v Consolidated Edison Co. of N.Y.*, 238 AD2d 454, 454-55 (2d Dept 1997) and *Askey v Occidental Chem. Corp.*, 102 AD2d 130, 135 (4th Dept. 1984), all of which concerned consequential rather than direct damages. In *Caronia*, the Court of Appeals determined that New York does not recognize an independent cause of action for medical monitoring and reaffirmed well established law that "[a] threat of future harm is insufficient to impose liability against a defendant in a tort context" and that "the requirement that a plaintiff sustain physical harm before being able to recover in tort is a fundamental principle of our state's tort system." *Caronia v Philip Morris USA, Inc.*, *supra* at 446. This Court has previously determined that the plaintiffs here have alleged the requisite injury via the accumulation of PFOA in their blood.¹ However, the *Caronia* Court also recognized that there "is a basis in law to sustain a claim for medical monitoring as an element of consequential damage." *Caronia v Philip Morris USA, Inc.*, *supra* at 447, quoting *Askey v Occidental Chem. Corp.*, 102 AD2d 130, 135 (4th Dept. 1984). The *Askey* court concluded that the plaintiffs could recover "reasonably anticipated consequential damages," including medical monitoring, so long as the plaintiffs could "establish with a reasonable degree of medical certainty that such expenditures [were] 'reasonably anticipated to be incurred by reason of their exposure'." *Caronia v Philip Morris USA, Inc.*, *supra*, citing *Askey, supra* at 137. On the other hand, "[c]onsequences which are contingent, speculative, or merely possible are not properly considered in ascertaining damages." *Askey, supra* at 136-37.

In this case, Dr. Savitz's affirmation indicates a clear dose response gradient that increases with PFOA exposure with respect to thyroid disease, ulcerative colitis, and kidney cancer. He found an increase in the risk of testicular cancer and high levels of uric acid and ALT across the quartiles of exposure. With respect to hypercholesterolemia, Dr. Savitz finds a dose-response gradient with a rapid increase in total cholesterol in the lower range of PFOA exposure. With respect to these specific diseases and conditions, the Court finds that Dr. Savitz has established that damages are

1

The Court in *Baker v. St.-Gobain Performance Plastics Corp.*, 232 F. Supp. 3d 233 (NDNY 2017), addressing very similar issues concerning injury and medical monitoring in a PFOA accumulation/property damage case has certified its order for interlocutory appeal, noted the Second Circuit's power to certify questions of state law to the New York Court of Appeals. Further, this Court's order denying the bulk of defendant's motion to dismiss on this basis is pending appeal in the Third Department, Appellate Division.

reasonably anticipated to flow from the invasion of the body by PFOA at or above background.

Citing the C8's own study, Dr. Savitz also concludes that exposure to PFOA is capable of causing preeclampsia and pregnancy induced hypertension. He acknowledges that a study of this same population² showed a weak association between PFOA exposure and pregnancy induced hypertension. Defendant attacks the weight and the strength of the plaintiff's contention here, which would certainly constitute a proper avenue of cross examination. However, this court cannot make a determination as to whether "such evidence is true. That function should be left to the jury." *Parker, supra* at 425. Plaintiffs' expert is properly subject to cross-examination, and the substance of his opinions is a subject for questioning. However, these issues go to credibility and to the weight to be given to the evidence.

Additionally, according to Dr. Savitz's own affidavit, there is only limited evidence supporting an association between PFOA exposure and risk of prostate, ovarian cancers and effects on the immune system at this point. Therefore, plaintiff has not established that monitoring expenditures are reasonably anticipated to be incurred based on plaintiffs' exposure at this time, and as such, this testimony is precluded.

Finally, whether expert testimony is novel or not, a trial court always has the duty to rule on the admissibility of evidence to determine its relevance. In this case, Dr. Savitz opines that other health conditions may in the future be established as probably causally linked to PFOA exposure. As noted above, a defendant may be liable for "reasonably anticipated" consequential damages. Consequences which are contingent, speculative, or merely possible are not properly considered in ascertaining damages. The Court agrees with defendant that speculation regarding the future of PFOA research is not relevant to any present cause of action at this time, and as such, this testimony is precluded.

In accordance with the foregoing, it is hereby

ORDERED that the defendant's motion to preclude Dr. David Savitz from testifying concerning an association between PFOA exposure and the risk of prostate cancer, ovarian cancer and effects on the immune system is **GRANTED**; and it is further

ORDERED that the defendant's motion to preclude Dr. David Savitz from testifying as to health conditions that may in the future be established as probably causally linked to PFOA exposure is **GRANTED**; and it is further

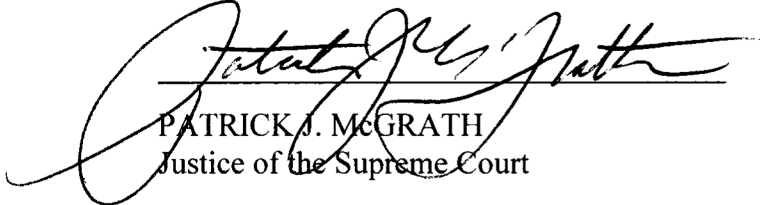
ORDERED that the balance of defendant's motion to preclude the testimony of Dr. David Savitz is **DENIED**.

This shall constitute the Decision and Order of the Court. This original Decision and Order

² See #16 in the bibliography attached to Dr. Savitz's affidavit.

is returned to Weitz & Luxenberg, PC, co-lead class counsel. All other supporting papers are being delivered by the Court to the Rensselaer County Clerk for filing. The signing and delivery of this Decision and Order does not constitute entry or filing under CPLR 2220. Plaintiffs are not relieved from the applicable provisions of that rule respecting filing, entry and notice of entry.

Dated: November 15, 2019
Troy, New York



PATRICK J. McGRATH
Justice of the Supreme Court

Papers Considered:

1. Notice of Motion; Affidavit of Thomas R. Smith, with Exhibits attached; Affidavit, Linda Dell; Taconic's Memorandum of Law in Support of Motion to Exclude Expert Testimony of Drs. Alan Ducatman, Donald Sloane Shepard and Donald R. Brandt.
2. Affidavit, David A. Savitz, Ph.D., Plaintiffs' Omnibus Memorandum of Law in Opposition to Defendant's Motion to Exclude Plaintiffs' Experts.
3. Taconic's Omnibus Reply in Support of Its Motions to Exclude Testimony of Plaintiffs' Experts; Affidavit, Jessica Kaplan, Esq., in Support of Taconic's Reply in Support of Its Motions to Exclude Testimony of Plaintiffs' Experts.

RECEIVED
RENSSELAER COUNTY CLERK
2019 NOV 25 AM 11:40
FRANK J MEROLA