

**STATE OF MICHIGAN**  
**COURT OF APPEALS**

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TERI WALTERS and KIM WALTERS,  
  
Plaintiffs-Appellants,

UNPUBLISHED  
January 29, 2015

v

DONALD S. FALIK, D.D.S., d/b/a FALIK  
FAMILY DENTISTRY, ROBERT C. FALIK,  
D.D.S., and JANE DOE,

No. 319016  
Eaton Circuit Court  
LC No. 12-000658-NH

Defendants-Appellees.

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Before: MURPHY, P.J., and METER and SERVITTO, JJ.

PER CURIAM.

Plaintiff Teri Walters (hereafter “Walters”) suffered an unintended exposure to phosphoric acid contained in a dental etching solution supplied by defendants. Plaintiffs proceeded to file a negligence action against defendants. The trial court granted summary disposition in favor of plaintiffs on the question of liability, and liability is not at issue in this appeal. Subsequently, the trial court granted defendants’ motion in limine, precluding the testimony of plaintiffs’ proposed expert witness, Dr. M. Eric Gershwin, on the issue of causation and damages. Plaintiffs claimed that the exposure to the phosphoric acid in the etching solution triggered or caused Wegener’s granulomatosis (WG), an autoimmune disease that Walters was diagnosed with after the exposure. As reflected in his deposition testimony, Dr. Gershwin was prepared to testify in support of a causal connection between the exposure to phosphoric acid and WG. The trial court, exercising its role as gatekeeper, concluded that Dr. Gershwin’s opinion on causation was not supported by sufficient data and reliable scientific principles, MRE 702. We reverse and remand for further proceedings.

On October 20, 2010, Walters went to defendants’ dental office to have a permanent crown seated, and following the procedure, she was provided with what she believed was a whitening solution for her teeth. Instead, a receptionist mistakenly gave Walters an etching solution, which was never intended to be dispensed to patients. An etching solution is used to “etch” the surfaces of teeth in preparation for the application of dental restoratives, and it contains phosphoric acid, which is a caustic acid and absolutely not meant to be used for teeth whitening. Etching solution, when used properly, should only remain on a particular tooth for about 20 seconds. A product safety data sheet regarding the etching solution indicated that it is corrosive, can cause chemical burns to the skin, eyes, mouth, and throat, may cause permanent

tissue damage, is harmful if swallowed, can irritate the respiratory system, and may cause swallowing difficulties, vomiting, diarrhea, and possible shock. Etching solution should not be exposed to moist air or water. Walters refrigerated the etching solution for a few months without using it, but on February 11, 2011, she spread the etching solution in her dental tray and left the tray in her mouth overnight in an attempt to whiten her teeth. When she awoke, Walters had a burning sensation inside of her mouth, including the sides of her tongue and along the gumline.

Walters went to her doctor on February 24, 2011, and according to the associated medical record, she complained of burns in her mouth as caused by the etching solution; she was diagnosed as having a “chemical burn.” On April 5, 2011, Walters again visited her doctor, complaining of sinus trouble that had been bothersome for a month, along with fullness in one ear. The medical record documenting the visit indicated that the examination revealed raw and swollen “[n]asal mucosa, septum, [and] turbinates.”<sup>1</sup> Walters was diagnosed with sinusitis and prescribed an antibiotic. She saw her doctor again on April 14, 2011, complaining of sinus congestion and a very full feeling in her ears, which was at times painful. The medical record documenting the visit indicated that Walters had been previously prescribed two different antibiotics for the sinus problems, which were ineffective, that she was “[p]ositive for ear pain, congestion, . . . and sinus pressure,” and that her examination revealed “[m]ucosal edema and rhinorrhea.” Walters was diagnosed with acute sinusitis. On May 4, 2011, Walters saw an ear, nose, and throat doctor (ENT) to be evaluated for chronic sinusitis. The associated ENT record indicated that Walters reported a sinus infection that had been present for approximately 45 days, continual plugging and fullness in her left ear, and ongoing sinus pain and pressure. The physical examination revealed “dryness and crusting on the anterior septum on the left side” and “fairly significant swelling in the ethmoid area bilaterally with drainage.” Following numerous procedures and evaluations at a hospital, Walters was diagnosed in June 2011 with WG, which has been explained and described as follows:

[WG] is a primary vasculitis of the small blood vessels [inflammation of the blood vessels] in which a characteristic feature is production of antineutrophil cytoplasmic antibodies (ANCA), usually targeted to proteinase 3 (PR 3). The symptoms, histology, and pathogenesis go through two phases. In the first phase, the disease is confined to the airways, causing sinusitis, otitis media, tracheal stenosis and/or pulmonary nodules. Histology usually shows granulomatous lesions. The second phase starts when the disease extends to other organs . . . .

The pathogenesis of WG and other vasculitides associated with ANCA remains unclear, but probably involves an interaction between a genetic susceptibility and environmental factors. [Hamidou, Audrain, Ninin, Robillard,

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<sup>1</sup> A “turbinate” is “a small curved bone that extends horizontally along the lateral wall of the nasal passage.” *Webster’s New College Dictionary* (2005).

Muller, and Bonneville, *Staphylococcus aureus*, *T-cell repertoire*, and *Wegener's granulomatosis*, 68 *Joint Bone Spine* (June 2001), p 373.<sup>2</sup>]

Before we examine Dr. Gershwin's deposition testimony, we will review the medical and scientific articles and literature relating to WG in order to provide context for Dr. Gershwin's testimony.

In Mahr, Neogi, and Merkel, *Epidemiology of Wegener's granulomatosis: Lessons from descriptive studies and analyses of genetic and environmental risk determinants*, 24 *Clinical and Experimental Rheumatology* (Supp 41, 2006), pp S-82, S-85 to S-87, the authors stated:

The etiology of WG remains unknown. Based on a growing number of epidemiologic investigations carried out during the last 15 years, current understanding is that of a complex disease resulting from the interplay among multiple genetic and environmental risk factors.

...

Analytic epidemiology aims to identify the determination of disease occurrence with putative risk factors commonly falling into 2 major categories: genetics and environment. In that context, the term "environment" is generally used to designate all non-genetic variables . . . .

...

Genetic predisposition to WG is suggested by ethnic variation . . . and by reports of familial aggregation . . . .

...

[S]tudies are consistent in finding positive associations between crystalline silica exposure and risk of WG . . . and exposure to silica. . . . Exposure to industrial pollutants such as mercury, lead, and cadmium had been found among patients with WG but these associations were weak or statistically non-significant. Another study revealed exposure to pesticides, particulate matter, or fumes as potential risk factors for WG.

ANCA-associated vasculitides comprise WG and "environmental factors have been considered important in the development of ANCA, including: silica exposure, bacterial infection [and] in particular *Staphylococcus aureus*, viral infection . . ., and exposure to drugs such as propylthiouracil." Chen and Kallenberg, *The environment, geoepidemiology and ANCA-associated vasculitides*, 9 *Autoimmunity Reviews* (2010), pp A293-A294; see also

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<sup>2</sup> All medical and scientific articles and literature cited in this opinion were submitted to and filed in the trial court for purposes of the motion in limine.

*Staphylococcus aureus*, *T-cell repertoire*, and *Wegener's granulomatosis*, 68 *Joint Bone Spine* at 373 (“The many environmental factors capable of inducing the production of ANCA or triggering true vasculitis include toxic substances (silica), drugs . . . , and others.”). “A case-control study carried out at the National Institutes of Health (NIH) revealed an association with exposure to fumes or particulates and pesticides in patients with WG compared with healthy or rheumatic disease controls” and “exposure to metal and welding fumes has been reported to increase the risk of . . . WG . . . .” Lane, Watts, Bentham, Innes, and Scott, *Are Environmental Factors Important in Primary Systemic Vasculitis?*, 48 *Arthritis & Rheumatism* #3 (March 2003), pp 814-815. “A history of high solvent exposure at any time was associated with . . . WG.” *Id.* at 818. In Duna, Cotch, Galperin, Hoffman, and Hoffman, *Wegener's granulomatosis: role of environmental exposures*, 16 *Clinical and Experimental Rheumatology* (November-December 1998), p 669, the authors stated that while the etiology of WG remained unknown, “[t]he predominant involvement of the airways and the presence of neutrophilic alveolitis at disease onset have led us to postulate that an inhaled agent may trigger the onset of WG.” In their study, the authors found that, as between WG and certain control groups, “[s]tatistically significant differences occurred [greater in the WG group] in regard to . . . vocational exposure to fumes or particulate materials . . . , residential exposure to particulate materials from construction . . . , and occupational exposure to pesticides[.]” *Id.* The authors also noted that one of several indicators reflecting the onset of WG is sinusitis of more than two months' duration. *Id.* at 670.

With respect to the apparent link between pesticides and WG, plaintiffs submitted scientific articles and literature regarding phosphorous and pesticides, showing that various forms of phosphorous are common components of pesticides. Centers for Disease Control and Prevention, *Organophosphorus Insecticides: Dialkyl Phosphate Metabolites*, Biomonitoring Summary, National Biomonitoring Program (July 2013), p 1 (“Organophosphorus insecticides . . . have accounted for a large share of all insecticides used in the United States.”); GoodGuide, *Organophosphate Pesticides: Dialkyl Phosphate Metabolites*, Scorecard (2011), p 1 (“Organophosphate pesticides account for about half of the insecticides used in the United States.”); Betteridge, Thompson, Baker, and Kemp, *Photoelectron Spectra of Phosphorus Halides, Alkyl Phosphites and Phosphates, Organo-Phosphorus Pesticides, and Related Compounds*, 44 *Analytical Chemistry* #12 (October 1972), p 2005.

We now turn to Dr. Gershwin's deposition testimony. He testified that “we know that autoimmunity, including [WG], occurs from a combination of genetic susceptibility and environmental factors.” When Dr. Gershwin was asked about environmental factors or chemicals in relation to autoimmunity, he observed that “[i]f we're referring to [WG], for example, we know that materials that alter the mucosal airway, whether it's superantigens, whether it's silica exposure, whether it's chemicals and certain hydrocarbons and solvents and pesticides[,] have been shown to be associated with patients who develop [WG].” Dr. Gershwin conceded that there was no literature that specifically indicated that phosphoric acid causes or contributes to WG. But he noted that one must look to “mechanisms of action” and “depend by analogy on the science which has already been done and peer reviewed on environmental agents, including the epidemiological data on solvents, hydrocarbons, agricultural products, [and] silica

in ANCA-positive patients.”<sup>3</sup> He also stated a few times that there would be no studies in which a person was experimentally exposed to phosphoric acid, as to conduct such testing would be unethical.

Dr. Gershwin testified that many solvents and pesticides contain phosphorus or phosphates, and he accurately noted a couple of times that silicon and phosphorus are next to each other on periodic table of elements (numbers 14 and 15 respectively), although he did not elaborate on the relevancy of this fact, simply implying a similarity in properties.<sup>4</sup> Dr. Gershwin then testified:

Well, many of the hydrocarbons contain phosphates as well. I already explained to you what the periodic table is. And incidentally, it's not really the phosphates that are the basis of my opinion. It's the phosphoric acid and what happens when it goes in solution, and I think that's what's critical. . . . Mrs. Walters presented with an acute onset, . . . with a very high-titer ANCA . . . . We had an advantage on her in understanding etiology that we won't have in people that have a more chronic exposure. Because, in fact, the silica exposure data shows it's not necessarily the duration of exposure as it is the intensity of an exposure. And of course, in the eight hours she had a very intense exposure.

. . .

So Mrs. Walters was exposed [o]n . . . February 11th, thereabouts. Her first sort of signs are about three or four weeks later. Even though temporal association by itself should not be the only criteria in this case, it is the perfect criteria for a primary immune response. Meaning if I were to immunize you with an antigen and ask how long it would take you to make a primary IGG [immunoglobulin] response, the time period in which she manifested the sinus symptoms is absolutely out of a textbook of immunology.

And because her disease began with her sinus symptoms and then became this incredible immunological storm that crescendoed into her upper airway issues, her failures, her respiratory distress . . . .

. . .

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<sup>3</sup> The literature indicated that not all patients with active WG have ANCAs present. *Wegener's granulomatosis: role of environmental exposures*, 16 *Clinical and Experimental Rheumatology* at 673. However, with respect to Walters, Dr. Gershwin testified that she “had a very high-titer ANCA.”

<sup>4</sup> Dr. Gershwin did indicate that “they have a number of interesting properties, including isoelectric focusing and so forth.” We appreciate that the particular arrangement of the elements on the periodic table is based on various properties of the elements.

So you then go back to the primary immune response, you have phosphoric acid, an[] intense exposure, incredible electrophilic agent that is not supposed to be exposed to water. She puts it in her mouth, and she leaves it there. And now you present when she finally is evaluated with a very high-titer ANCA. You don't usually have that scenario.

...

[W]hen I look at the potency and the chemistry of the phosphoric acid in the etching solution, it's just above and beyond anything that I can even compare it to. I mean, this was an incredible inflammatory insult. . . . [When] we look at Mrs. Walters and we look at the chemistry of what she was exposed to, the time period of her clinical presentation, the safety data sheets [for the etching solution], the data that's already available on environmental factors, this is a very important case for the medical literature.

...

[We] really have a plausible scientifically acceptable mechanism of action.

...

So I think what happened is phosphoric acid hits water, disassociates, produces an incredible inflammatory response, including neutrophil. That's why she had the symptoms she had. The neutrophils enter the area, they marginate acutely as they are supposed to, they degrade, the myeloperoxidase neutrophilic antigens get released, she mounts an immune response, she boosts it, and that's why she has . . . WG.

Dr. Gershwin further testified that sinusitis does not cause WG; rather, sinusitis is a manifestation, sign, and symptom of WG. He opined that Walters "would not have got [WG] had it not been for the etching solution." Dr. Gershwin clarified that he could not state that she would not have suffered WG in the future, given her predisposition to WG and the possibility of another type of environmental exposure; however, he emphasized that Walters would not have suffered WG when she did but for the exposure to the etching solution. Dr. Gershwin opined that WG would reduce Walters' life span by ten 10 years.

Defendants filed a motion in limine to preclude Dr. Gershwin's testimony. They argued that there was no scientific support for his conclusion that the etching solution is an environmental catalyst that, along with Walters' alleged predisposition to WG, caused the onset of her WG. Defendants additionally contended that Dr. Gershwin's testimony regarding a causal connection between the etching solution / phosphoric acid and WG failed to meet the test for reliability under MCL 600.2955. The trial court granted the motion, ruling that Dr. Gershwin's testimony was not reliable under MRE 702 and not supported by the scientific and medical data. The trial court noted a few times that the articles and literature expressed that the etiology or cause of WG remained unclear and unknown. The trial court also concluded that the scientific and medical data relied on by plaintiffs failed to show a connection between phosphorus, phosphates, or phosphoric acid and the chemicals and environmental factors that had been

identified in literature as being associated with WG; and even that association was tenuous. Plaintiffs appeal by leave granted.

In *Chapin v A & L Parts, Inc*, 274 Mich App 122, 126-127; 732 NW2d 578 (2007), this Court set forth the applicable standard of review and the general principles that are implicated when examining the admissibility of expert testimony:

This Court reviews for an abuse of discretion a trial court's determination of the qualifications of a proposed expert witness. This Court likewise reviews for an abuse of discretion a trial court's decision whether to admit evidence, although admission of legally inadmissible evidence is necessarily an abuse of discretion. The interpretation of an evidentiary rule is reviewed de novo in the same manner as the examination of the meaning of a court rule or a statute. Rules of evidence are construed in the same way as statutes.

Before a trial court may admit any expert testimony, the trial court is required by MRE 702<sup>[5]</sup> to ensure that each aspect of an expert witness's proffered testimony—including the data underlying the expert's theories and the methodology by which the expert draws conclusions from that data—is reliable. While the exercise of this gatekeeper role is within a court's discretion, a trial judge may neither abandon this obligation nor perform the function inadequately. The plain language of MCL 600.2955(1)<sup>[6]</sup> establishes the Legislature's intent to

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<sup>5</sup> MRE 702 provides:

If the court determines that scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education may testify thereto in the form of an opinion or otherwise if (1) the testimony is based on sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

<sup>6</sup> MCL 600.2955(1) provides:

In an action for the death of a person or for injury to a person or property, a scientific opinion rendered by an otherwise qualified expert is not admissible unless the court determines that the opinion is reliable and will assist the trier of fact. In making that determination, the court shall examine the opinion and the basis for the opinion, which basis includes the facts, technique, methodology, and reasoning relied on by the expert, and shall consider all of the following factors:

(a) Whether the opinion and its basis have been subjected to scientific testing and replication.

assign the *trial court* the role of determining . . . whether proposed scientific opinion is sufficiently reliable for jury consideration. The United States Supreme Court emphasized that the inquiry is flexible and focused solely on principles and methodology rather than ultimate conclusions, and its overarching subject is the scientific validity—and thus the evidentiary relevance and reliability—of the principles that underlie a proposed submission. [Citations, quotation marks, and alteration brackets omitted.]

In *Chapin*, this Court recognized the “Sir Bradford Hill” (SBH) methodology for examining causation, which methodology goes beyond just using epidemiological data and is primarily intended to determine cause and effect. *Id.* at 133. The SBH methodology contains nine criteria: strength of association; temporality (cause must precede effect); biologic or response gradient (basic toxicological knowledge); consistency; specificity, biological plausibility; coherence; experimental evidence; and analogy. *Id.* at 133-134.

Here, Dr. Gershwin’s opinion that Walters’ exposure to the phosphoric acid in the etching solution caused her WG was predicated on: (1) the caustic nature of phosphoric acid; (2) phosphoric acid being a WG-triggering environmental factor or chemical; (3) the intensity and duration of the phosphoric acid exposure (overnight while Walters slept); (4) the area of exposure (inside the mouth where moisture exists); (5) the textbook timing of the presentation of an immune response in relationship to the date of exposure and the overall chronology of events; (6) the incredible extent of the immune response (a very high-titer ANCA); (7) the manifestation

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(b) Whether the opinion and its basis have been subjected to peer review publication.

(c) The existence and maintenance of generally accepted standards governing the application and interpretation of a methodology or technique and whether the opinion and its basis are consistent with those standards.

(d) The known or potential error rate of the opinion and its basis.

(e) The degree to which the opinion and its basis are generally accepted within the relevant expert community. As used in this subdivision, “relevant expert community” means individuals who are knowledgeable in the field of study and are gainfully employed applying that knowledge on the free market.

(f) Whether the basis for the opinion is reliable and whether experts in that field would rely on the same basis to reach the type of opinion being proffered.

(g) Whether the opinion or methodology is relied upon by experts outside of the context of litigation.

and duration of a classic WG symptom, sinusitis; (8) Walters' predisposition to WG; and (9) the medical and scientific literature, directly or by analogy. Dr. Gershwin's analysis employed many of the criteria associated with the SBH methodology; it was a cause and effect methodology.

With respect to the caustic nature of phosphoric acid, this was established by the product safety data sheet regarding the etching solution, which indicated that it is corrosive, can cause chemical burns, permanent tissue damage, swallowing difficulties, vomiting, diarrhea, and possible shock, is harmful if swallowed, can irritate the respiratory system, and that it should not be exposed to moist air or water. Given that Walters slept overnight with a dental tray in her mouth that was filled with the etching solution, there is no dispute regarding the intensity, duration, and location of the exposure. There also does not appear to be any dispute concerning the claimed textbook timing or chronology of the presentation of an immune response in relationship to the date of exposure, nor as to Walters' very high-titer ANCA. Next, the medical and scientific literature cited above supports the proposition that sinusitis can be a manifestation of the onset of WG, especially where it is of long duration, as was the case with Walters.

On the issue of the role of environmental factors and genetic influences relative to causation, as reflected above, the medical and scientific literature did indeed indicate that the etiology of WG remains unclear and unknown. However, when read in context, this proposition was clearly meant to indicate that a "definitive" or "absolutely certain" etiology is unclear and unknown. In *Staphylococcus aureus, T-cell repertoire, and Wegener's granulomatosis*, 68 Joint Bone Spine at 373, the authors stated that "[t]he pathogenesis of WG and other vasculitides associated with ANCAs . . . probably involves an interaction between a genetic susceptibility and environmental factors." (Emphasis added.) In *Epidemiology of Wegener's granulomatosis: Lessons from descriptive studies and analyses of genetic and environmental risk determinants*, 24 Clinical and Experimental Rheumatology at S-82, S-85 to S-87, the authors, while acknowledging that the "etiology of WG remains unknown," nonetheless stated that "[b]ased on a growing number of epidemiologic investigations carried out during the last 15 years, current understanding is that of a complex disease resulting from the interplay among multiple genetic and environmental risk factors." (Emphasis added.) Those authors also indicated that "studies are consistent in finding positive associations between crystalline silica exposure and risk of WG." (Emphasis added.) In *The environment, geoepidemiology and ANCA-associated vasculitides*, 9 Autoimmunity Reviews at A293-A294, the authors stated that "environmental factors have been considered important in the development of ANCA." (Emphasis added.) And in *Wegener's granulomatosis: role of environmental exposures*, 16 Clinical and Experimental Rheumatology at 669, the authors noted that while the etiology of WG remained unknown, "[t]he predominant involvement of the airways and the presence of neutrophilic alveolitis at disease onset have led us to postulate that an inhaled agent may trigger the onset of WG."

Accordingly, while not definitive, there is medical and scientific literature indicating that the *probable or likely cause* of WG is a combination of environmental and genetic factors. We observe that plaintiffs, in order to succeed, are not required to *definitively* establish a causal link between the exposure and WG.<sup>7</sup> Moreover, the inquiry regarding whether proposed scientific

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<sup>7</sup> "To establish a prima facie case of negligence, a plaintiff must prove four elements: (1) a duty owed by the defendant to the plaintiff, (2) a breach of that duty, (3) causation, and (4) damages."

opinion is sufficiently reliable for a jury to consider “is flexible and focused solely on principles and methodology *rather than ultimate conclusions*[.]” *Chapin*, 274 Mich App at 126-127 (emphasis added). The trial court here made an error of law in analyzing the question of the admissibility of Dr. Gershwin’s testimony by effectively requiring plaintiffs to establish causation and their case prior to trial and to do so definitively. And the trial court further erred by failing to take into consideration Dr. Gershwin’s legitimate reliance on the nature, duration, intensity, and location of the exposure, the temporal proximity of the immune response to the date of exposure, and the duration and nature of an expected manifestation of WG, i.e., a lengthy battle with sinusitis, which all played a role in Dr. Gershwin’s overall analysis.

With respect to the lack of medical or scientific literature specifically connecting an exposure to phosphoric acid with WG, there was literature, cited above, reflecting the prevalence of phosphates in pesticides and showing a statistically significant association between pesticides and the development of WG. *Organophosphorus Insecticides: Dialkyl Phosphate Metabolites*, Biomonitoring Summary, National Biomonitoring Program, at 1; *Are Environmental Factors Important in Primary Systemic Vasculitis?*, 48 *Arthritis & Rheumatism* at 814-815; *Wegener’s granulomatosis: role of environmental exposures*, 16 *Clinical and Experimental Rheumatology* at 669. Also, while not expressly stated, it appears that Dr. Gershwin’s view was that phosphoric acid is comparable to silica, which has been linked to WG, in the context of environmental factors that cause WG. Regardless, the lack of a specific study looking at direct exposures to phosphoric acid is understandable considering the ethical dilemma posed by conducting such a study. And we find our situation analogous to that in *Chapin*, where “a number of epidemiological studies had analyzed mesothelioma among automobile brake mechanics and failed to show an association between asbestos-based automobile brake products and mesothelioma.” *Chapin*, 274 Mich App at 135. This Court, nonetheless, allowed the plaintiffs’ expert to testify about a causal link between exposure to asbestos-based automobile brake products and mesothelioma, considering all of the SBH criteria supporting causation. *Id.* at 140. The *Chapin* panel ruled:

This case does *not* present a situation involving questionable or absent epidemiological evidence coupled with questionable or absent *other* evidence, or

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*Case v Consumers Power Co*, 463 Mich 1, 6; 615 NW2d 17 (2000). The causation element encompasses both cause in fact and proximate or legal cause. *Id.* at n 6. “The cause in fact element generally requires showing that ‘but for’ the defendant’s actions, the plaintiff’s injury would not have occurred.” *Skinner v Square D Co*, 445 Mich 153, 163; 516 NW2d 475 (1994). It is not sufficient to proffer “a causation theory that, while factually supported, is, at best, just as possible as another theory.” *Id.* at 164. A “plaintiff must present substantial evidence from which a jury may conclude that *more likely than not*, but for the defendant’s conduct, the plaintiff’s injuries would not have occurred.” *Id.* at 164-165 (emphasis added). But “litigants do not have any right to submit an evidentiary record to the jury that would allow the jury to do nothing more than guess.” *Id.* at 174. The *Skinner* Court further observed that “[t]he evidence need not negate all other possible causes” and absolute certainty on causation is not required. *Id.* at 166, quoting 57A Am Jur 2d, Negligence, § 461, p 442.

coupled with an “expert” who actually lacks the requisite qualifications.<sup>[8]</sup> Rather, this is a case involving strong and undisputed support for . . . [plaintiffs’ expert’s] position, coupled with fairly consistent yet potentially questionable contradictory evidence, depending on which expert is to be believed. Although clearly not universally accepted, and although unsupported by epidemiological studies that may or may not be flawed, . . . [his] opinion is certainly objective, rational, and based on sound and trustworthy scientific literature. [*Id.*]

As indicated, in *Chapin*, there were studies that *actually showed no correlation* between exposure to asbestos-based automobile brake products and mesothelioma, yet the expert was allowed to testify, and here there is no indication that there exist any studies showing that phosphoric acid does not cause WG. Furthermore, as in *Chapin*, there was other unquestionable cause-and-effect evidence showing a link between the exposure and WG; plus, there are epidemiological studies generally supporting Dr. Gershwin’s opinion. We also note that defendants do not counter Dr. Gershwin’s assertion that when phosphoric acid comes into contact with moisture, such as it did inside Walters’ mouth, it produces an incredible inflammatory response. Again, the product safety data sheet regarding the etching solution provided that it should not be exposed to moist air or water, is harmful if swallowed, and can cause respiratory distress. We are not prepared to preclude Dr. Gershwin’s testimony simply because there is not a specific study showing that exposure to phosphoric acid causes WG.

In sum, Dr. Gershwin’s testimony was based on sufficient facts or data, it was the product of reliable principles and methods, and Dr. Gershwin applied the principles and methods reliably to the facts of the case, MRE 702. The trial court erred in granting defendants’ motion in limine. The trier of fact will ultimately be free to weigh Dr. Gershwin’s opinion on causation and accept or reject it. See *Chapin*, 274 Mich App at 140 (“[D]eciding this case at an evidentiary hearing, depriving the jury of the opportunity to fulfill its proper role as fact-finder, would be inappropriate.”).

Reversed and remanded for further proceedings consistent with this opinion. We do not retain jurisdiction. Having fully prevailed on appeal, plaintiffs are awarded taxable costs under MCR 7.219.

/s/ William B. Murphy  
/s/ Deborah A. Servitto

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<sup>8</sup> There is no dispute that, as found by the trial court, Dr. Gershwin is highly qualified to give an opinion in this area of medicine; his CV is extremely impressive.