Order

Michigan Supreme Court
Lansing, Michigan

July 25, 2017

Stephen J. Markman,
Chief Justice

151600

Brian K. Zahra
Bridget M. McCormack
David F. Viviano
Richard H. Bernstein
Joan L. Larsen
Kurtis T. Wilder,
Justices

CHANCE LOWERY,
Plaintiff-Appellee,

v

SC: 151600
COA: 319199
Calhoun CC: 2011-003414-NO

ENBRIDGE ENERGY LIMITED PARTNERSHIP and ENBRIDGE ENERGY PARTNERS LP,
Defendants-Appellants.

_____/

On order of the Court, leave to appeal having been granted, and the briefs and oral arguments of the parties having been considered by the Court, we REVERSE the April 2, 2015 judgment of the Court of Appeals and REINSTATE the November 8, 2013 order of the Calhoun Circuit Court granting the defendants' motion for summary disposition. A plaintiff may show "cause in fact" through circumstantial evidence and "reasonable inferences" therefrom, but not through "mere speculation" or "conjecture," *Skinner v Square D Co*, 445 Mich 153, 164 (1994), such as reasoning *post hoc ergo propter hoc*, see *Genesee Merchants Bank & Trust Co v Payne*, 381 Mich 234, 248 (1968) (opinion by KELLY, J.) ("But fact-finders, be they jury or court, may not indulge in conjecture. They are constrained to draw reasonable inferences from established facts. Reasoning '*post hoc ergo propter hoc*' does not meet this test.") (citation omitted). The plaintiff's expert opined that the defendants' oil spill was the cause in fact of the plaintiff's injury, reasoning that the plaintiff "wasn't having the problems before [the oil spill] and he was having the problems afterwards." Contrary to the Court of Appeals conclusion that the plaintiff's evidence reflects a "logical sequence of cause and effect," we conclude that the plaintiff's evidence reflects the logical fallacy of *post hoc* reasoning. Cf. *West v Gen Motors Corp*, 469 Mich 177, 186 n 12 (2003) ("Relying merely on a temporal relationship is a form of engaging in the logical fallacy of *post hoc ergo propter hoc* (after this, therefore in consequence of this) reasoning.") (quotation marks omitted). We, therefore, conclude that the plaintiff has failed to show a genuine dispute of material fact as to causation.

We do not retain jurisdiction.

MARKMAN, C.J. (*concurring*).

I concur in this Court's decision to reverse the judgment of the Court of Appeals and write separately to provide counsel to the bench and bar concerning toxic tort

litigation. This Court granted leave to appeal to consider: (a) the role of expert testimony in toxic tort cases; (b) the applicability of the general-and-specific-causation framework in toxic tort cases; and (c) the sufficiency of plaintiff's evidence of causation in the instant toxic tort case. *Lowery v Enbridge Energy Ltd Partnership*, 499 Mich 886 (2016). The importance of these issues is evinced, in part, by the fact that of the 54 cases heard by this Court during the present term, only 13 involved, as did this case, full grants. Today, the Court does not address these issues but instead resolves this case in an order of reversal. Uncertainty continues to characterize our toxic tort jurisprudence despite the fact that the general-and-specific-causation framework has proven uncontroversial in contemporary toxic tort law outside Michigan. Bernstein, *Getting to Causation in Toxic Tort Cases*, 74 Brook L Rev 51, 52 (2008) ("American courts have reached a broad consensus on what a plaintiff must show to prove causation in a toxic tort case. First, a plaintiff must show that the substance in question is capable of causing the injury in question. This is known as 'general causation.' Second, a plaintiff must show that this substance caused *his* injury. This is known as 'specific causation.' ") (citations omitted). I write separately only to provide some semblance of guidance to litigants in this and future cases-- to those pursuing and those defending toxic tort claims-- as well as similar guidance to the lower courts of our state in presiding over and in reviewing these claims. Such guidance is critical because in Michigan there is a paucity of law concerning toxic torts, much of what law exists is confusing and contradictory, and all this is occurring at a time when it appears that toxic tort litigation is on the upturn here as in other jurisdictions. I respectfully offer the following analysis to better clarify our toxic tort jurisprudence.

* * *

First, I would clarify that Michigan's long-held general rules regarding the necessity of expert testimony apply to toxic torts, i.e., expert testimony on causation is necessary in a toxic tort case when the legal proposition is beyond the common knowledge of an ordinary juror. Second, I agree with the vast majority of other jurisdictions that the general-and-specific-causation framework may be utilized to analyze the cause-in-fact element of a toxic tort claim. At a minimum, this framework should apply when a plaintiff seeks to prove factual causation employing group-based statistical evidence. In this case, plaintiff submitted such evidence to prove cause in fact. Accordingly, applying the framework, I would hold that plaintiff failed to present adequate evidence of cause in fact, specifically evidence establishing *either* general or specific causation. Therefore, I concur with the Court's reversal of the judgment of the Court of Appeals and remand to the trial court for reinstatement of its order granting summary disposition in defendants' favor.

A review of the facts that led to this litigation is helpful to understanding my analysis that follows. This case concerns a large and severe oil spill into a Michigan woodland and river. On July 26, 2010, a pipeline belonging to defendants, Enbridge

Energy Limited Partnership and Enbridge Energy Partners, LP, ruptured and released 840,000 gallons of crude oil into a woodland area. The oil eventually migrated into Talmadge Creek and the Kalamazoo River and further spread nearly 40 miles throughout Calhoun and Kalamazoo counties. The federal Environmental Protection Agency (EPA) eventually intervened, ordering a cleanup and conducting air monitoring and sampling to measure the level of volatile organic compounds (VOCs) in the air.[1] A voluntary evacuation was issued for the immediate geographic area of the spill.

Plaintiff, Chance Lowery, lived roughly 250 feet from the banks of the Kalamazoo River and approximately 11 to 13 miles downstream from the spill's source. He claimed to have smelled chemicals shortly after the spill and to have become sick as a result--coughing and vomiting for several days, and then proceeding to the hospital.[2] A scan performed at the hospital indicated that plaintiff had a stomach hemorrhage. Dr. John Koziarski, a general and vascular surgeon who is board certified in general surgery and vein diseases, performed a successful operation to repair the hemorrhage, which revealed that "a short gastric vessel midway down the stomach . . . had avulsed off of the spleen."

On the basis of these injuries and damage to his property, plaintiff filed a complaint alleging defendants' negligence. Regarding causation, the complaint alleged that plaintiff was exposed to "hazardous substances" that constituted "a proximate cause" of plaintiff's injuries. Those injuries included "nausea, a severe cough and violent vomiting, which caused a rupture of his short gastric artery, which required subsequent surgical repair and resulted in a disfiguring prominent surgical scar." Expert testimony on the matter of causation[3] consisted of deposition testimony by the treating physician, Dr. Koziarski, and deposition testimony and a report from Dr. Jerry Nosanchuk, a general physician who is board certified in family medicine. The former testified that plaintiff stated that he had taken Vicodin for a migraine, began vomiting, and then developed severe abdominal pain. Dr. Koziarski testified that Vicodin could cause vomiting but that

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[1] VOCs are an aggregation of chemicals that may be found in crude oil and can be harmful to humans at certain exposure levels. See generally Wallace, *Personal Exposures, Indoor and Outdoor Air Concentrations, and Exhaled Breath Concentrations of Selected Volatile Organic Compounds Measured for 600 Residents of New Jersey, North Dakota, North Carolina and California*, 12 Toxicological & Envtl Chemistry 215 (1986).

[2] Despite the uncertain timeline to which plaintiff testified-- describing symptoms manifested over the course of approximately five to seven days immediately following the spill-- his medical records show that he was admitted to the hospital on August 18, 2010, which was 23 days after the initial spill.

[3] The trial court granted summary judgment in plaintiff's favor regarding defendants' breach of duty-- i.e., that defendants acted negligently. That order has not been challenged here.

he had no medical opinion whether Vicodin was what specifically caused plaintiff's vomiting. He also had no opinion concerning whether plaintiff's anti-depression medication, Lamictal, could also cause migraines. Plaintiff never indicated to Dr. Koziarski that fumes wafting from the Kalamazoo River had caused or contributed to his vomiting or his headaches. Dr. Koziarski concluded that he could not opine as to whether plaintiff had exposure to the fumes or whether that exposure accounted for the "rupture or avulsion of the gastric artery[.]" He also could not determine the avulsion's medical cause.

Given that Dr. Koziarski did not opine as to whether the fumes caused plaintiff's condition, expert testimony on causation before the trial court was limited to Dr. Nosanchuk's testimony. He reviewed plaintiff's hospital records, a Michigan Department of Community Health document about the spill, a newspaper report concerning the spill, plaintiff's deposition testimony as well as his interrogatory answers, and photographs of plaintiff's backyard displaying its proximity to the river. He did not physically examine plaintiff. Dr. Nosanchuk was "of the opinion that the fumes from the oil spill caused [plaintiff] to have the migraine headaches, extreme coughing and nausea as well as vomiting. Ultimately, these problems caused a tear of the short gastric artery resulting in hemorrhage within the [stomach]."

Plaintiff also presented deposition testimony from his roommate, a neighbor, and a friend regarding the noticeable smell[4] near and within his apartment as well as information regarding VOCs exposure from the Centers for Disease Control and Prevention (CDC).[5] After discovery, defendants moved for partial summary disposition under MCR 2.116(C)(10). Following arguments, the trial court granted defendants' motion for summary disposition, limited only to plaintiff's ailments beyond vomiting and headaches. The court determined that there was nothing to link the cause of the ruptured artery to the oil spill. In response, plaintiff's counsel stated that he would rather the court grant summary disposition in its entirety because this "whole case is all about the surgery" and plaintiff would prefer to appeal the ruling immediately. The trial court concurred, and an order was entered by the court affirming its ruling on the record.

In a split opinion, the Court of Appeals reversed the grant of summary disposition and remanded for further proceedings. *Lowery v Enbridge Energy Ltd Partnership*,

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[4] Plaintiff's roommate compared the smell to "asphalt . . . like burning, rubber, tar . . . ." Plaintiff's friend explained, "[I]t smelled like you tipped over fuel oil in your driveway." Plaintiff's neighbor testified that the smell was "like rubber burning."

[5] Plaintiff attached the CDC documents to his response to defendants' motion for summary disposition. I make no determination as to whether these documents were properly in the record or would have been deemed admissible absent expert testimony; I assume they are properly part of plaintiff's proofs for purposes of my analysis.

unpublished per curiam opinion of the Court of Appeals, issued April 2, 2015 (Docket No. 319199), p 1. The majority held that expert testimony showing that the toxin, VOCs, was capable of causing the injuries alleged and that it actually did cause such injuries was not required in light of the Court's earlier decision in *Genna v Jackson*, 286 Mich App 413 (2009). *Lowery*, unpub op at 2-3. The Court found it sufficient that the circumstantial evidence plaintiff had presented established "a strong enough logical sequence of cause and effect for a jury to reasonably conclude that plaintiff's exposure to oil fumes caused his vomiting, which ultimately caused his short gastric artery to rupture." *Id*. at 3. In dissent, Judge JANSEN would have affirmed the trial court's grant of summary disposition. She reasoned that "[p]laintiff's theory of causation was attenuated" and that a jury comprised of lay people would be unable to determine whether the oil fumes could have caused vomiting and the resulting arterial tear absent the aid of expert testimony. *Id*. at 1 (JANSEN, J., dissenting). She further noted that Dr. Nosanchuk was unqualified to give such testimony and therefore that the jury was left on its own to speculate concerning the issue of causation. *Id*.

Defendants subsequently filed an application seeking leave to appeal in this Court, and we granted its application, requesting that the parties address "(1) whether the plaintiff in this toxic tort case sufficiently established causation to avoid summary disposition under MCR 2.116(C)(10); and (2) whether the plaintiff was required to present expert witness testimony regarding general and specific causation." *Lowery*, 499 Mich 886.

I. ANALYSIS

In a typical tort claim grounded in negligence, plaintiffs "must prove (1) that defendant owed them a duty of care, (2) that defendant breached that duty, (3) that plaintiffs were injured, and (4) that defendant's breach caused plaintiffs' injuries." *Henry v Dow Chemical Co*, 473 Mich 63, 71-72 (2005). "Proof of causation requires both cause in fact and legal, or proximate, cause." *Haliw v Sterling Heights*, 464 Mich 297, 310 (2001). "[L]egal cause or 'proximate cause' normally involves examining the foreseeability of consequences, and whether a defendant should be held legally responsible for such consequences." *Skinner v Square D Co*, 445 Mich 153, 163 (1994). "The cause in fact element generally requires showing that 'but for' the defendant's actions, the plaintiff's injury would not have occurred." *Id*. A plaintiff must demonstrate as a threshold matter that there is "more than a mere possibility" that the defendant caused the injury, *id*. at 166 (citation and quotation marks omitted), and must then present "substantial evidence" from which a jury could conclude that, more likely than not, "but for the defendant's conduct, the plaintiff's injuries would not have occurred," *Weymers v Khera*, 454 Mich 639, 647-648 (1997) (citation and quotation marks omitted). That substantial evidence "must exclude other reasonable hypotheses with a fair amount of certainty," *Skinner*, 445 Mich at 166 (citation and quotation marks omitted), because a jury cannot be permitted to merely guess about causation, *id*. at 174.

This Court has defined "an ordinary 'toxic tort' cause of action" as one in which "a plaintiff alleges he has developed a disease [or other injury] because of exposure to a toxic substance negligently released by the defendant." *Henry*, 473 Mich at 67. Toxic torts are thus a specific type of negligence claim. In order to establish a claim under a toxic tort theory, a plaintiff must prove an injury arising from exposure to a toxic substance. *Id*. at 72-73 (holding that plaintiffs could not maintain a toxic tort claim to recover damages for the cost of medical monitoring for potential *future* injuries), citing *Larson v Johns-Manville Sales Corp*, 427 Mich 301 (1986). This Court has not yet addressed whether the causation element of a toxic tort claim differs in any meaningful way from that of a traditional negligence claim. Indeed this case implicates several issues regarding causation in toxic tort cases in Michigan: namely, whether the cause-in-fact element of a toxic tort claim includes separate analyses of general and specific causation; if so, what evidence a plaintiff must provide on those issues to survive a summary disposition motion; and whether such evidence must include expert testimony. To address these questions, I begin with an analysis of the unique challenges posed by the cause-in-fact element of a toxic tort claim, i.e., those challenges that arise in addressing the general-and-specific-causation inquiries subsumed within.

A. CAUSE IN FACT

The great majority of jurisdictions have bifurcated the cause-in-fact element in toxic tort cases into separate and distinctive analyses of "general causation" and "specific causation."[6] This analytical approach for determining causation in toxic tort cases also

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[6] See, e.g., *CW ex rel Wood v Textron, Inc*, 807 F3d 827, 831 (CA 7, 2015) (applying Indiana substantive law, which required "evidence of general and specific causation"); *Knight v Kirby Inland Marine Inc*, 482 F3d 347, 351 (CA 5, 2007) ("General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury.") (citation and quotation marks omitted); *Mattis v Carlon Electrical Prod*, 295 F3d 856, 860 (CA 8, 2002) ("To prove causation in a toxic tort case, a plaintiff must show both that the alleged toxin is capable of causing injuries like that suffered by the plaintiff in human beings subjected to the same level of exposure as the plaintiff, and that the toxin was the cause of the plaintiff's injury.") (citation and quotation marks omitted); *Mitchell v Gencorp Inc*, 165 F3d 778, 781 (CA 10, 1999) ("[A] plaintiff must demonstrate the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover.") (citation and quotation marks omitted); *Ranes v Adams Laboratories, Inc*, 778 NW2d 677, 687-688 (Iowa, 2010) ("Courts have commonly bifurcated toxic-tort-causation analysis into two separate but related parts: general causation and specific causation. . . . The Third Restatement of Torts has recognized this relatively recent common practice as a device to organize a court's analysis and not as additional elements

finds support in the secondary literature.[7] Therefore, application of the general-and-specific-causation framework in toxic tort cases has been far from untested. The Restatement (Third) of Torts provides a lengthy discussion of the bifurcated general-and-specific-causation framework in its comments, noting in particular that

> [c]ases involving toxic substances often pose difficult problems of proof of factual causation. . . . Sometimes it is difficult to prove which defendant was connected to the toxic agent or whether an adequate warning would have prevented the plaintiff's harm. The special problem in these cases, however, is proving the connection between a substance and development of a specific disease. [Restatement Torts, 3d, Liability for Physical and Emotional Harm, § 28, comment *c*, p 402 (citations omitted).]

After noting that most causation issues in this context are resolved under the "but for" standard of factual causation, the Restatement provides that when a plaintiff presents "group-based statistical evidence" concerning a toxin, a plaintiff must prove that "the substance must be capable of causing the disease ('general causation') and that the substance must have caused the plaintiff's disease ('specific causation')." *Id*. at 404.

This is not a novel concept. General causation is implicit in all negligence claims, but in negligence claims that do not involve toxic torts, the plaintiff typically does not need to present separate proof of each type of causation because the relationship between general and specific causation is sufficiently direct and straightforward such that both types of causation are effectively proven together. By analogy, imagine a simple negligence claim in which the defendant drove his car over the plaintiff's foot, breaking it. Evidence proving that the car broke the plaintiff's foot practically proves both the "general" causation requirement of proof (that the car was *capable* of causing the injury) and the "specific" causation requirement (that the car *did in fact* break the foot). In other words, in a typical negligence claim, the same evidence will often prove that exposure to

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of the tort. The Restatement authors supplement their explanation by asserting factual causation is a necessary element in every tort case; the general and specific language has simply become more prevalent in toxic-tort cases. . . . This bifurcated analysis has not been explicitly used as the standard in Iowa. However, due to its general acceptance among scholars and courts of other jurisdictions, as well as the relative ease of application the analysis offers to courts examining complex issues of causation, we believe it is appropriate for courts to use the bifurcated causation language in toxic-tort cases.") (citations, quotation marks, and brackets omitted).

[7] See, e.g., Note, *Causation in Environmental Law: Lessons from Toxic Torts*, 128 Harv L Rev 2256, 2261-2262 (2015); Sanders, *The Controversial Comment* C*: Factual Causation in Toxic-Substance and Disease Cases*, 44 Wake Forest L Rev 1029, 1031 (2009); Stout & Valberg, *Bayes' Law, Sequential Uncertainties, and Evidence of Causation in Toxic Tort Cases*, 38 U Mich J L Reform 781, 784 (2005).

the harm produced by the defendant's negligence *could* and *did* cause the injury in dispute. See Restatement, § 28, comment *c*, p 402 ("In most traumatic-injury cases, the plaintiff can prove the causal role of the defendant's tortious conduct by observation, based upon reasonable inferences drawn from everyday experience and a close temporal and spatial connection between that conduct and the harm. Often, no other potential causes of injury exist. When a passenger in an automobile collision suffers a broken limb, potential causal explanations other than the collision are easily ruled out; common experience reveals that the forces generated in a serious automobile collision are capable of causing a fracture.").

But in a toxic tort claim, this relationship may be considerably less clear. See *Landrigan v Celotex Corp*, 127 NJ 404, 413 (1992) (noting that in the toxic tort context, "proof that a defendant's conduct caused decedent's injuries is more subtle and sophisticated than proof in cases concerned with more traditional torts"). For example, an injury such as cancer has many suspected causes, including exposure to various toxins in various quantities and durations. See Farber, *Toxic Causation*, 71 Minn L Rev 1219, 1227 (1987) ("One [issue] is the problem of establishing that the chemical involved is capable of causing the type of harm from which the plaintiff suffers. This is often difficult because the causation of diseases like cancer is so poorly understood."). In such cases, proof of cause in fact *may* need to take the form of separate proofs that the toxin *can* cause the harm and that it *did*. See, e.g., *Mattis v Carlon Electrical Prod*, 295 F3d 856, 860 (CA 8, 2002) ("To prove causation in a toxic tort case, a plaintiff must show both that the alleged toxin is capable of causing injuries like that suffered by the plaintiff in human beings subjected to the same level of exposure as the plaintiff, and that the toxin was the cause of the plaintiff's injury.") (citation and quotation marks omitted). Absent evidence regarding each inquiry, a jury could be left improperly to speculate as to the nature of the relationship between the toxin and the plaintiff's injury. *Skinner*, 445 Mich at 164 ("To be adequate, a plaintiff's circumstantial proof must facilitate reasonable inferences of causation, not mere speculation."). For these reasons, the general-and-specific-causation framework is helpful in toxic tort cases to ensure that the cause-in-fact element is properly proven; at a minimum, this is true when the plaintiff avails himself or herself of the framework by presenting group-based statistical evidence or similar scientific evidence[8] because such evidence[9] by its nature can only speak to whether the

[8] The application of the general-and-specific-causation framework should not be *limited* to instances in which the plaintiff presents group-based statistical evidence. As the Restatement further asserts: "In toxic-substances cases, the causal inquiry is modified by the limits of and available forms of scientific evidence. That inquiry *often* must address whether the [toxic] agent for which the actor is responsible is capable of causing the disease from which another suffers (known as general causation). In addition, the question whether the [toxic] agent caused the specific plaintiff's disease (known as specific causation) is confronted." Restatement, § 26, comment *g*, p 351 (emphasis added).

substance is *capable* of causing the alleged injury (general causation) and does not address whether the substance, in fact, caused the plaintiff's injury (specific causation).

In other instances, the general-and-specific-causation framework may be unnecessary to establish cause in fact, such as those instances in which the causal link between an injury and a toxin is as direct and apparent as it is in the case in which the car breaks the plaintiff's foot. Sometimes the "mechanism of causation is well understood . . . [or] the causal relationship is well established," such as when the resulting injury is immediate and traumatic rather than gradual and disease-based. Green et al, *Reference Guide on Epidemiology*, in Reference Manual on Scientific Evidence (3d ed), p 609 n 180. In such cases, the general-and-specific-causation framework might be unnecessary. Consider, for example, a plaintiff who suffers a severe chemical burn immediately after toxic acid has been spilled onto his skin. There might well be no need for the application of this analytical framework when the causal link is so clear and straightforward.[10]

Michigan has little authority on this topic, and this Court has yet to provide significant guidance. I take this opportunity to begin to rectify this.

B. GENERAL CAUSATION

General causation pertains to whether a toxin is capable of causing the harm alleged. A necessary predicate to this inquiry is identifying the asserted exposure level of the toxin. "A number of courts have required plaintiffs to prove the level of exposure (dose) in order to establish causation." *Goeb v Tharaldson*, 615 NW2d 800, 815 (Minn,

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[9] This less-direct evidence might not always consist of group-based statistical studies. See, e.g., *King v Burlington N Santa Fe R Co*, 277 Neb 203, 215-221 (2009) (explaining the value of epidemiological studies in evidencing general causation and providing that in the absence of epidemiological studies, an expert may refer alternatively to the United States Surgeon General's "Bradford Hill" factors for evidence of general causation).

[10] Because I recognize that not *every* toxic tort claim requires separate proof of general and specific causation in order to establish cause in fact, I also recognize that the absence of separate proofs regarding general and specific causation does not prevent a plaintiff from establishing a prima facie case of negligence in every toxic tort case. See *Christian v Gray*, 65 P3d 591, 604 (Okla, 2003) ("[G]eneral causation should be shown unless the particular controversy is inappropriate for general causation. We decline to list hypothetical controversies where general causation need not be shown."). It should be emphasized that these concepts "are not 'elements' of a plaintiff's cause of action" but rather "function as devices to *organize* a court's analysis . . . . So long as the plaintiff introduces admissible and sufficient evidence of factual causation, the burden of production is satisfied." Restatement, § 28, comment *c*, p 405 (emphasis added).

2000). "[T]he mere existence of a toxin in the environment is insufficient to establish causation without proof that the [particular] level of exposure could cause the plaintiff's symptoms." *Pluck v BP Oil Pipeline Co*, 640 F3d 671, 679 (CA 6, 2011). Put another way, causation "requires not simply proof of exposure to the substance, but proof of enough exposure to cause the plaintiff's specific illness." *McClain v Metabolife Int'l, Inc*, 401 F3d 1233, 1242 (CA 11, 2005).

Knowledge of the exposure level is crucial to determining whether the toxin can cause the harm because many substances are harmful in certain quantities but are safe at lower levels; carbon monoxide, for instance, is constantly in the air, but it only causes adverse health symptoms in certain higher concentrations. See *Zuchowicz v United States*, 140 F3d 381, 391 (CA 2, 1998) ("[A]ll drugs involve risks of untoward side effects. . . . At the approved dosages, the benefits of the particular drug have presumably been deemed worth the risks it entails. At greater than approved dosages, . . . the risks of tragic side effects (known and unknown) increase . . . ."); Eaton, *Scientific Judgment and Toxic Torts-- A Primer in Toxicology for Judges and Lawyers*, 12 J L & Pol'y 5, 11 (2003) (" 'All substances are poisonous—there is none which is not; the dose differentiates a poison from a remedy.' ") (citation and emphasis omitted). Moreover, a substance may cause different harmful effects in different doses. See Goldstein, *Toxic Torts: The Devil is in the Dose*, 16 J L & Pol'y 551, 554 (2008) ("Dose is defined as concentration multiplied by frequency or duration—it is not just the exposure level at any one point in time."). As a result, a substance may be harmful at a certain level of exposure but may not be sufficient to cause a particular adverse health effect. *In re Agent Orange*, 570 F Supp 693, 695 (ED NY, 1983) (stating that general causation "is addressed to the common question of whether exposure to [the toxin] *in the manner that it was used* in [plaintiff's location] could cause the kinds of injuries that plaintiffs claim to have suffered") (emphasis added). Accordingly, a plaintiff's evidence of general causation should be tailored to the *estimated* amount and duration of exposure at issue to enable the fact-finder to reasonably conclude that exposure to the defendant's toxin in the amount and duration alleged is capable of causing the alleged injury. See *Wright v Willamette Indus, Inc*, 91 F3d 1105, 1107 (CA 8, 1996) ("[T]here must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of that agent that are known to cause the kind of harm that the plaintiff claims to have suffered.").

C. SPECIFIC CAUSATION

Evidence of specific causation consists of proof that exposure to the toxin more likely than not caused *the plaintiff's* injury. Specific causation requires at minimum an approximate estimate of the plaintiff's exposure level as well as an evaluation and elimination of other reasonable potential causes. It is well accepted that "a plaintiff in a toxic tort case must prove . . . the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover." *Id*. at 1106. I recognize that "it is often . . . particularly difficult . . . to establish [exposure levels] in a [toxic] tort suit"

given "the adventitious, often accidental, and even unknown (at the time) exposures typical of toxic tort cases . . . ." Cranor, *Toxic Torts: Science, Law, and the Possibility of Justice* (New York: Cambridge University Press, 2d ed, 2016), at 252. Therefore, as in ordinary negligence claims, circumstantial evidence of causation *may* be sufficient to establish exposure adequate to prove specific causation. See *Skinner*, 445 Mich at 164 (stating that a plaintiff can satisfy his or her burden to prove causation in a negligence claim by providing circumstantial proof that facilitates reasonable inferences of causation). This position is also in accordance with that of other jurisdictions that have held that exposure levels in a toxic tort case can be "roughly established through reliable circumstantial evidence." See, e.g., *Blanchard v Goodyear Tire & Rubber Co*, 190 Vt 577, 578-579 (2011) (citation and quotation marks omitted). Federal courts likewise have concluded that "exact details pertaining to the plaintiff's exposure are beneficial" but "not always available, or necessary," *Westberry v Gislaved Gummi AB*, 178 F3d 257, 264 (CA 4, 1999), and that "precise data on the exact degree of exposure to each chemical" is not always required, see *Harper v Illinois Cent Gulf R*, 808 F2d 1139, 1141 (CA 5, 1987).

Nevertheless, to avoid leaving the jury to speculate, a plaintiff should set forth at least some evidence that he or she was exposed to the toxin at issue, including the estimated amount and duration of exposure. *Skinner*, 445 Mich at 164 ("To be adequate, a plaintiff's circumstantial proof must facilitate reasonable inferences of causation, not mere speculation."). While toxic tort plaintiffs are not required to provide "a mathematically precise table equating levels of exposure with levels of harm, . . . there must [nonetheless] be evidence from which a reasonable person could conclude that a defendant's emission has probably caused a particular plaintiff the kind of harm of which he or she complains before there can be a recovery." *Wright*, 91 F3d at 1107. A plaintiff should not rely "merely on a temporal relationship [to establish causation because this] is a form of engaging in 'the logical fallacy of post hoc ergo propter hoc (after this, therefore in consequence of this)' reasoning." See *West v Gen Motors Corp*, 469 Mich 177, 186 n 12 (2003) (citation omitted); *McClain*, 401 F3d at 1243 ("[S]imply because a person takes drugs and then suffers an injury does not show causation. Drawing such a conclusion from temporal relationships leads to the blunder of the *post hoc ergo propter hoc* fallacy.").

Instead, the plaintiff's exposure level should be shown, at minimum, by circumstantial evidence that facilitates reasonable inferences.[11] See *Mitchell v Gencorp*

[11] For instance, in *Curtis*, the Fifth Circuit held that the plaintiff's expert gave rise to a genuine issue of fact regarding specific causation when he opined that the plaintiff had been exposed to a toxin at levels of at least 200 to 300 parts per million based on results of lower level exposure tests, work practices at the exposure site, and the nature of his symptoms. *Curtis v M&S Petroleum, Inc*, 174 F3d 661, 671 (CA 5, 1999).

Inc, 165 F3d 778, 781 (CA 10, 1999) ("Guesses, even if educated, are insufficient to prove the level of exposure in a toxic tort case."). "In cases claiming personal injury from exposure to toxic substances, it is essential that the plaintiff demonstrate that she was, in fact, exposed to harmful levels of such substances." *Abuan v Gen Electric Co*, 3 F3d 329, 333 (CA 9, 1993) (citation, quotation marks, and emphasis omitted). Evidence of the plaintiff's exposure level should encompass proof that the plaintiff was *actually* exposed to the defendant's toxin as well as the *estimated* amount and duration of exposure. See *Allen v Pennsylvania Engineering Corp*, 102 F3d 194, 199 (CA 5, 1996) (stating that a toxic tort plaintiff must show that he or she was exposed to harmful quantities of a chemical to sustain his or her burden); *Wintz ex rel Wintz v Northrop Corp*, 110 F3d 508, 513 (CA 7, 1997) (holding that the plaintiff's expert testimony failed to establish exposure to a chemical when the expert did not address "how frequently, in what quantity, or in what form" the plaintiff was exposed to the chemical or the plaintiff's "specific dose").

Another significant component of specific causation in a toxic tort case pertains to the evaluation and elimination of other reasonably relevant potential causes of a plaintiff's symptoms.[12] In order to demonstrate specific causation, a plaintiff's " 'evidence must exclude other reasonable hypotheses with a fair amount of certainty.' " *Skinner*, 445 Mich at 166, quoting 57A Am Jur 2d, Negligence, § 461, p 442. One common method for excluding reasonably relevant potential causes of a plaintiff's injury may be a "differential etiology," sometimes characterized as a "differential diagnosis." *Myers v Illinois Cent R Co*, 629 F3d 639, 644 (CA 7, 2010) (explaining that the former term is the more accurate in referring to causation because it focuses on identifying the cause of the ailment from which plaintiff suffers, whereas the latter term focuses on the identification of that ailment). Differential etiology is "a method by which all [reasonably relevant] possible causes of a condition are listed and then the various causes are ruled out so as to leave the most likely cause or causes of a particular patient's problem." *Dengler v State Farm Mut Ins Co*, 135 Mich App 645, 649 (1984); see also *Attorney General v Beno*, 422 Mich 293, 312-313 (1985) (differential diagnosis describes "the process of elimination of other possible maladies" as the cause of a plaintiff's symptoms); *Westberry*, 178 F3d at 262 ("[D]ifferential etiology is a standard scientific technique of identifying the cause of a medical problem by eliminating the *likely* causes until the most probable one is isolated.") (emphasis added; punctuation omitted).

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[12] Plaintiff's evidence need not address every remote possible cause in the universe. See *Skinner*, 445 Mich at 166, quoting 57A Am Jur 2d, Negligence, § 461, p 442 (" 'The evidence need not negate all other possible causes . . . .' "); *Viterbo v Dow Chem Co*, 826 F2d 420, 424 (CA 5, 1987) (holding in a toxic tort case that the plaintiff's expert need not disprove or discredit every possible cause of the plaintiff's injury other than the one espoused by him, but must do more than simply pick the cause that is most advantageous to the plaintiff's claim).

Without the performance of a differential etiology,[13] " '[t]here may be 2 or more plausible explanations as to how an event happened or what produced it; yet, if the evidence is without selective application to any 1 of them, they remain conjectures only [and are insufficient to establish causation].' " *Skinner*, 445 Mich at 164, quoting *Kaminski v Grand Trunk W R Co*, 347 Mich 417, 422; 79 NW2d 899 (1956). As explained earlier, specific causation is subsumed within the cause-in-fact inquiry. In order to prove cause in fact, a plaintiff must demonstrate that there is more than an "evenly balanced" probability that the conduct of the defendant *was*, rather than was *not*, the cause in fact of the harm suffered. *Mulholland v DEC Int'l Corp*, 432 Mich 395, 416 n 18 (1989) (citation and quotation marks omitted); see also *Skinner*, 445 Mich at 164 ("Nor is it sufficient to submit a causation theory that, while factually supported, is, at best, just as possible as another theory."). A differential etiology is included in the specific-causation inquiry under this burden because a plaintiff that fails to perform a differential etiology or some equivalent will not be able to meet his or her overall burden as described in *Mulholland*, i.e., when various possible causes of an injury exist, and when the plaintiff has not identified the most probable of these, the probability that the defendant's conduct-- as opposed to some other potential cause-- constituted the cause in fact of the plaintiff's harm remains "evenly balanced." In such instances, the jury is left to infer causation from correlation, which it cannot do because "[i]t is axiomatic in logic and in science that correlation is not causation. This adage counsels that it is error to infer that A causes B from the mere fact that A and B occur together." See, e.g., *Craig v Oakwood Hosp*, 471 Mich 67, 93 (2004). As a result, specific causation includes the need by some reasonable means to evaluate and eliminate other reasonably relevant potential causes of the plaintiff's injury.

D. EXPERT TESTIMONY

Because of the complexity of the general-and-specific-causation inquiry in toxic tort cases, it may also be necessary for a plaintiff to present expert testimony.[14] Many

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[13] While a differential etiology is not specifically or necessarily required in every toxic tort case, a plaintiff should utilize *some* reliable method, or introduce *some* evidence, designed to exclude other reasonably relevant potential causes of his or her injury. For example, some courts rely on studies comparing the incidence of the disease in groups exposed to the toxin and groups not exposed. See Green et al, pp 611-612.

[14] I am cognizant that plaintiff in this case has presented expert testimony and thus whether he was required to do so is largely irrelevant to the resolution of his claim. I address this issue nonetheless because questions concerning the need for expert testimony will often be integral in cases of the instant sort-- toxic tort cases. It is altogether appropriate that this Court-- as the court of last resort of this state, as the court ultimately responsible for the fair and orderly development of our common law-- in reasonable

jurisdictions have held that expert testimony is generally necessary or else even suggest that it is always required in a toxic tort case.[15] That conclusion is reiterated in the secondary literature as well.[16] Michigan has yet to address this matter in a toxic tort case

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ways, set forth the law more clearly so that litigants can reasonably apprehend their respective legal obligations in initiating and defending against claims of the instant sort. As members of this Court have stated on innumerable occasions during oral argument in our courtroom, it is the Court's responsibility not only to address the case immediately before us in accordance with the law but also to afford guidance in the "next one-hundred" similar cases. The question whether expert testimony is required is critical in identifying the proofs that must be provided by a plaintiff to satisfy his or her evidentiary burden when the general-and-specific-causation framework applies. My analysis would afford little guidance to litigants concerning the application of the general-and-specific-causation framework were I to fail to address the logically related and recurrent question of the need for expert testimony in such cases. Future litigants are entitled to at least minimal guidance concerning what is required to survive summary disposition in toxic tort cases.

[15] See, e.g., *Milward v Rust-Oleum Corp*, 820 F3d 469, 476 (CA 1, 2016) (requiring expert testimony in a toxic tort case and stating that "[a]s is well-established under Massachusetts law, 'expert testimony is required to establish medical causation' ") (citation omitted); *Junk v Terminix Int'l Co*, 628 F3d 439, 450 (CA 8, 2010) ("In proving both types of causation, 'expert medical and toxicological testimony is unquestionably required to assist the jury.' ") (citation omitted); *Seaman v Seacor Marine LLC*, 326 F Appx 721, 723 (CA 5, 2009) ("A plaintiff in such a case [i.e., a case involving injuries from exposure to toxins] cannot expect lay fact-finders to understand medical causation; expert testimony is thus required to establish causation."); *Wills v Amerada Hess Corp*, 379 F3d 32, 46 (CA 2, 2004) ("In a case such as this [concerning exposure to a toxin], where an injury has multiple potential etiologies, expert testimony is necessary to establish causation . . . ."); *Redland Soccer Club, Inc v Dep't of Army of US*, 55 F3d 827, 852 (CA 3, 1995) (applying Pennsylvania law in a toxic tort case and stating that "[w]hen the complexities of the human body place questions as to the cause of pain or injury beyond the knowledge of the average layperson . . . the law requires that expert medical testimony be employed") (brackets, quotation marks, and citation omitted); *Harris v CSX Transp, Inc*, 232 W Va 617, 653 (2013) ("[T]he need for expert testimony to supply [the] critical causal connection is often the key to a plaintiff's toxic tort case . . . .").

[16] Gold, *The "Reshapement" of The False Negative Asymmetry in Toxic Tort Causation*, 37 Wm Mitchell L Rev 1507, 1536 (2011) ("[D]eciding a toxic causation dispute is inherently beyond the ken of lay people and therefore demands expert scientific testimony."); see also Comment, *Causation in Toxic Tort Litigation: "Which Way Do We Go, Judge?"*, 12 Vill Envtl LJ 33, 34-35 (2001) ("The existence of . . . unique causation problems that confront plaintiffs in toxic torts makes it necessary for parties to offer expert testimony.").

specifically, but the generally applicable rule in Michigan is that expert testimony is required when highly technical and scientific questions are at issue. *Elher v Misra*, 499 Mich 11, 21-22 (2016) (requiring expert testimony on negligence in a medical malpractice action unless the matter "is within the common knowledge and experience" of the average juror); *Teal v Prasad*, 283 Mich App 384, 394 (2009) (generally requiring expert testimony on causation in a medical malpractice action); see also *Amorello v Monsanto Corp*, 186 Mich App 324, 331 (1990) (affirming summary disposition when the plaintiffs' expert failed to establish the causation element of the plaintiffs' products-liability claim). This rule originates in our common law and is grounded in the notion that scientific questions should be addressed by those with the relevant professional skill and knowledge so as not to leave jurors to speculate regarding matters beyond their knowledge. See, e.g., *Miller v Toles*, 183 Mich 252, 258 (1914); *Spaulding v Bliss*, 83 Mich 311, 315 (1890); *Mayo v Wright*, 63 Mich 32, 40 (1886); *Wood v Barker*, 49 Mich 295, 298 (1882). Put another way, the generally applicable rule is not a separate or a distinctive rule at all, but rather is a part of the plaintiff's evidentiary burden to establish cause in fact. When the jury is able only to speculate concerning causation-- which is all jurors can do when a matter is scientific in character such that it is beyond their common knowledge-- the plaintiff has not satisfied his or her burden. Because the causation inquiry in toxic tort cases is often scientific in nature, a plaintiff will often be hard-pressed to satisfy that evidentiary burden absent expert testimony; absent such testimony, the jury will only be left to speculate. For this reason, I would apply our general rule and conclude that the need for expert testimony regarding causation in a toxic tort case is determined on the basis of whether the matter "is so obvious that it is within the common knowledge and experience of an ordinary layperson." *Elher*, 499 Mich at 21-22. If "the untrained layman would be qualified to determine intelligently and to the best possible degree [the elements of the claim] without enlightenment from those having a specialized understanding of the subject involved in the dispute," then expert testimony is unnecessary and indeed is inadmissible.[17] *People v Kowalski*, 492 Mich 106, 123 (2012) (opinion by MARY BETH KELLY, J.) (citation and quotation marks omitted); *Gilbert v DaimlerChrysler Corp*, 470 Mich 749, 790 (2004) (expert testimony is admissible to assist the trier of fact to understand a proposition that is " 'beyond the ken of common knowledge' ") (emphasis omitted), quoting *Zuzula v ABB Power T & D Co, Inc*, 267 F Supp 2d 703, 711 (ED Mich, 2003). Conversely, expert testimony may be required when

[17] Under MRE 702, expert testimony *cannot* be introduced at trial unless it assists the jury with a proposition beyond their common knowledge. As established by even our earliest medical malpractice jurisprudence, a party *must* introduce expert testimony at trial if the proposition is not within the common knowledge of the average juror. See, e.g., *Miller*, 183 Mich at 258. Both standards are helpful to the current inquiry due to their complementary character. Seemingly, when considered together, these rules suggest that expert testimony that is admissible is most often required, and expert testimony that is not required is most often inadmissible.

the causation inquiry "is scientific in nature," *Nelson v American Sterilizer Co (On Remand)*, 223 Mich App 485, 489 (1997), such that it is beyond "the common knowledge and experience of the jury," see *Bryant v Oakpointe Villa Nursing Ctr, Inc*, 471 Mich 411, 426 (2004) (holding that a claim sounds in medical malpractice and thus requires expert testimony when the questions at issue are not "within the common knowledge and experience of the jury").[18]

II. APPLICATION

As explained earlier, in raising a toxic tort claim, a plaintiff is required to provide proof of cause in fact. In the present case, plaintiff has relied on group-based statistical evidence or similar scientific proof. Therefore, the general-and-specific-causation framework would apply. Accordingly, the Court should examine plaintiff's evidence to determine whether he has sufficiently shown general and specific causation, that is, whether the pertinent toxin (VOCs) is capable of causing the alleged injury and whether plaintiff here was actually exposed to that toxin at a level sufficient to cause the severe coughing and vomiting that, in turn, would cause his gastric artery to avulse. Given that the final step of this inquiry is clearly beyond "the common knowledge and experience of a jury," *Dorris v Detroit Osteopathic Hosp Corp*, 460 Mich 26, 47 (1999), plaintiff was required to present expert testimony to that effect. Plaintiff also needed to reasonably evaluate and eliminate other reasonable potential causes of his injuries.

A. GENERAL CAUSATION

Plaintiff here failed to establish that a causal link generally exists between the toxin released by the negligent act (VOCs) and the asserted harm (coughing, vomiting, and avulsion). Judge JANSEN correctly recognized that the harm suffered was "attenuated" from the negligent act, meaning that it required two findings to establish *general* causation: (1) VOCs in the level and duration at issue are capable of causing the degree and duration of coughing and vomiting at issue; and (2) coughing and vomiting in the degree and duration caused by the VOCs are capable of causing a gastric artery to avulse. Plaintiff's proofs-- that is, the CDC documents-- contain the generally recommended exposure limits and the permissible exposure limits for three of the main chemicals found in VOCs-- Toluene, Benzene, and m-Xylene-- as well as state that overexposure to these chemicals can cause some amount of nausea or headaches. Indeed, plaintiff's toxic tort claim did not fail with respect to damages for his coughing and nausea; the trial court denied defendants' summary disposition motion regarding those injuries. Plaintiff also presented some evidence indicating that coughing and vomiting

[18] To the extent that the Court of Appeals' opinion in *Genna*, 286 Mich App 413, has been understood *never* to require expert testimony in toxic tort cases, I believe that this is in error, and therefore I would explicitly reject this understanding.

can cause a gastric artery to avulse, albeit only rarely.

Nevertheless, plaintiff's general-causation evidence falls short because it fails to show what *level* and *duration* of exposure to VOCs can cause the *severity* and *duration* of coughing and vomiting that is necessary to cause a gastric artery to avulse. After all, not every person suffering stomach flu also suffers an avulsion of a gastric artery. When plaintiff's expert was asked if he knew "what specific levels of exposure are required to cause any of [the] symptoms [plaintiff suffered]," he declined to provide an opinion: he did not "think that's a question that could be answered unless you are speaking of a specific person and you would have to -- you would have to gauge that in retrospect because everybody is different, I think. I believe that to be true." The remainder of plaintiff's proofs also fail to address this critical point-- i.e., whether plaintiff's level of VOCs exposure was capable of causing the level of vomiting necessary to cause a gastric artery to avulse. While plaintiff was not required to reference specific data and could have established general causation by alternative methods,[19] plaintiff presented *no* evidence regarding the exposure level necessary to cause his particular injuries and has failed to sustain his burden to prove general causation as a result.

B. SPECIFIC CAUSATION

Plaintiff failed to establish specific causation. Again, plaintiff's harm was attenuated from defendants' action and required two findings to establish *specific* causation-- plaintiff's exposure to VOCs more likely than not caused him to cough and vomit, and such coughing and vomiting more likely than not caused his gastric artery to avulse. Plaintiff lacked evidence of specific causation on numerous grounds. First, he did not show that he was exposed to any VOCs, let alone exposure of the magnitude necessary to cause his particular symptoms. Second, he failed to reasonably consider and eliminate other potential causes of his symptoms. Third and last, he failed to provide adequate evidence concerning the causal link between his coughing and vomiting and the avulsion of his gastric artery-- a determination that is certainly beyond the common knowledge of the average juror and thus that required sufficient evidence in the form of expert testimony.[20]

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[19] See, e.g., *King*, 277 Neb at 215-221 (discussing the "Bradford Hill" factors for evidence of general causation in the absence of epidemiological studies).

[20] I would presume for the purposes of my analysis that plaintiff's expert testimony was admissible, but I would find that it was nevertheless insufficient. See *Conde v Velsicol Chem Corp*, 24 F3d 809, 813 (CA 6, 1994) ("Accordingly, we turn to the question of whether the [plaintiffs'] expert testimony, assuming that it is admissible, is sufficient to withstand summary judgment for [the defendant] on the issue of medical causation."); *Elkins v Richardson-Merrell, Inc*, 8 F3d 1068, 1071 (CA 6, 1993) (affirming summary judgment in the defendant's favor and concluding that precedent establishes that the court

I examine each of these shortcomings in turn. Plaintiff's first failure was to overlook the matter of personal exposure. When plaintiff's expert, Dr. Nosanchuk, was questioned regarding his conclusion that plaintiff was exposed to VOCs that made him cough and vomit, Dr. Nosanchuk admitted that he did not know where the oil spill started or how far the release site was located from plaintiff's home. When asked what chemicals were in the oil, he responded: "I think it was benzyl, toluylene, xylene. Maybe there was something else too." He obtained this knowledge online and admitted that he lacked specific knowledge of which chemical constituents were present or in what quantities. Nor did he have any information concerning the emission or dispersion rates of VOCs. "[O]n a personal level" his understanding of VOCs effects was based on pumping gasoline into his own car: "[T]hey're an irritant. I don't really understand the toxicology. I know that they're irritants and I know that they're capable of causing cough, nausea, vomiting, irritation of the eyes and any other mucous membranes." All that this testimony would reasonably demonstrate to the fact-finder is that VOCs contained in gasoline pumped into a car can, under *some* circumstances, act as an irritant. By itself, however, this fact neither evidences that plaintiff inhaled or was otherwise exposed to the VOCs contained in defendant's oil nor that such VOCs acted as an irritant under *these* circumstances.

Plaintiff himself only alleged that he smelled oil fumes "really strong" for several days. But, "[i]t is important to understand that these VOCs can be smelled at levels well below those that would cause health problems." EPA, *Enbridge Oil Spill: How is Air Quality Affected?*, p 1 (emphasis omitted), available at <https://www.epa.gov/sites/production/files/2016-06/documents/enbridge_fs_airquality_20100802.pdf> (accessed May 31, 2017) [https://perma.cc/5FU8-DKGD]. Despite knowledge here of the oil release site's location, the amount spilled, and the duration of the incident, plaintiff did not provide any scientific information regarding VOCs, such as the conditions under which VOCs evaporate into the air, how quickly they do so and in what concentrations, the amount of surface oil necessary to produce a toxic level of VOCs in the air, how VOCs disperse in the air, and how long VOCs remain in the air.[21] This list is only

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"treat[s] the plaintiff's expert opinion indicating a basis of support for the plaintiffs' [sic] theories . . . to be admissible but 'simply inadequate . . . [to] permit a jury to conclude that [the toxin] more probably than not causes [the type of injury the plaintiff suffered]' ") (citation omitted; second alteration in original).

[21] Plaintiff did not even use the EPA air monitoring sampling data results, which provided the amounts of various VOCs in parts per billion by volume of the highest peak readings in locations near the oil spill. See, e.g., EPA, *Enbridge Oil Spill: Human Health Air Screening Levels*, available at <https://archive.epa.gov/region5/enbridgespill/data/web/pdf/enbridge_voc_screening_levels_20100813.pdf> (accessed May 31, 2017)

illustrative because plaintiff was not necessarily required to provide evidence on *all* these issues or to provide detailed chemical testing, modeling, and case studies to prove his claim. But he had to, at least approximately, establish his own level of exposure. *Blanchard*, 190 Vt at 579 ("[W]hile 'it is not always necessary for a plaintiff to quantify exposure levels precisely,' courts generally preclude experts from testifying 'as to specific causation without having any measurements of a plaintiff's exposure to the allegedly harmful substance.' "), quoting *Henricksen v ConocoPhillips Co*, 605 F Supp 2d 1142, 1157 (ED Wash, 2009). Plaintiff here provided *no* information whatsoever regarding his potential exposure to VOCs. Absent evidence of his exposure level, plaintiff could not establish specific causation and therefore failed to show the cause-in-fact element of his toxic tort claim. See *Henry*, 473 Mich at 67 ("In an ordinary 'toxic tort' cause of action, a plaintiff alleges he has developed a disease *because of* exposure to a toxic substance negligently released by the defendant.") (emphasis added).[22]

Second, plaintiff failed to adequately consider and eliminate other factors that *reasonably* could have caused his injuries. Two days after his surgery, plaintiff was still in the hospital and informed Dr. Koziarski that another migraine headache was forming. Plaintiff reported to Dr. Koziarski that he was "reluctant to take Norco or Vicodin as this is what made him throw up the first time" and that he also thought Lamictal, the medication used to treat his depression, "may be causing his migraines." Plaintiff's medical history indicated that he "[g]ets migraines when stressed" and "has nausea and dry heaves[;] however it only occurs if he smokes or is around smoke." The same record stated that plaintiff smokes. Further, plaintiff had visited the hospital in January 2008, complaining of headaches and nausea, which he then attributed to his recently increased dosage of Lamictal. Plaintiff was required to exclude these reasonably relevant potential causes of his injuries with a reasonable amount of certainty, but as shown in the record, his evidence to this effect was insufficient.

[https://perma.cc/MF78-3UQU]. Plaintiff argues that defendants relied on information outside the record in providing the EPA's sampling data to this Court, but defendants correctly argue that they were allowed to observe that such data existed and was publicly available in making the argument that plaintiff failed to demonstrate his exposure to harmful levels of VOCs. Such information is judicially noticeable. See MRE 201(b).

[22] Even assuming arguendo that plaintiff's exposure could be garnered from reasonable inferences given the high number of other individuals in plaintiff's area that developed symptoms consistent with VOCs exposure and plaintiff's evidence to this effect, see *Curtis*, 174 F3d at 671-672 (holding that it was acceptable that the plaintiffs' expert established exposure *in part* by considering the fact that several refinery workers developed the same cluster of symptoms consistent with benzene exposure shortly after the chemical was introduced to the refinery), plaintiff failed to establish specific causation for the additional reasons that follow.

Initially when plaintiff's expert, Dr. Nosanchuk, was asked whether he had ruled out other relevant potential causes of plaintiff's injuries, he essentially denied having performed a differential etiology of any kind, believing "that other potential causes were very unlikely." But he later recalled not having been asked to consider alternative causes and stated: "I'm sure I considered a lot of them. I don't remember any one that sticks in my mind." When asked how he was able to rule out these other causes, his *full* response was, "Thought about it." Then when he was pressed on his evaluation and elimination of alternative causes of plaintiff's symptoms, Dr. Nosanchuk several times resorted to a well-known and insufficient manner of causal reasoning-- mistaking correlation for causation. See, e.g., *Craig*, 471 Mich at 93 ("It is axiomatic in logic and in science that correlation is not causation.").

For instance, when Dr. Nosanchuk was questioned on his claim that the oil leak was the *sole* cause of plaintiff's symptoms, he stated that he meant "as far as I was concerned that is what was causing it. He wasn't having the problems before and he was having the problems afterwards. The oil spill and the problems associated with the oil spill are capable of doing that and I think they did do that and that is my clinical judgment based on what I knew." As the majority's order correctly notes, Dr. Nosanchuk's reasoning is also an example of the *post hoc ergo propter hoc* fallacy. See *State of Ohio v US Dep't of the Interior*, 279 US App DC 109, 150 (1989) (explaining the fallacy in an oil spill case and stating that it is "the fallacy of assuming that, simply because a biological injury occurred after a spill, it must have been caused by the spill"). Dr. Nosanchuk's report, in which he opined that Lamictal was not related to plaintiff's health problems, contained the same flawed analysis. In explaining the basis for his opinion, Dr. Nosanchuk cited the Michigan Department of Community Health report that stated that the chemicals released in the spill could cause headaches and nausea. Because plaintiff was "squarely within the parameters [i.e., the location of the affected area] as outlined in the report," Dr. Nosanchuk believed the spill had to have caused plaintiff's symptoms and that Lamictal had nothing to do with these symptoms. Demonstrating that the VOCs *could* have caused headaches and nausea fails to establish that Lamictal-- or any other potential cause-- *did not* cause plaintiff's headaches and vomiting. The fact-finder thus is left with no evidence ruling out other causes or even tending to show that other causes are less likely the cause of the injury than are the VOCs.

Additionally, when Dr. Nosanchuk was asked if smoking could have been related to the nausea and vomiting, his response was simply that it could not have been. Later, however, he acknowledged that Vicodin could cause nausea and that smoking could cause coughing, but he did not believe that smoking was related to plaintiff's problems: "Now, whether or not the smoking played any part at all [in plaintiff's symptoms], I don't know. All I know is my understanding is . . . he didn't complain of significant cough before the fumes." This type of correlative reasoning is not enough to reasonably eliminate an alternative cause. It does not provide a fact-finder any rationale for concluding that the VOCs are more likely the cause of plaintiff's maladies than smoking.

In sum, plaintiff's expert only provided conclusory conjecture based on correlative reasoning, and therefore his testimony was insufficient to reasonably eliminate other reasonably relevant potential causes of plaintiff's injuries as is required to establish specific causation. *Skinner*, 445 Mich at 164 (holding that impermissible conjectures do not amount to reasonable causal inferences).

Third and finally, plaintiff did not provide adequate evidence concerning the causal link between his coughing and vomiting and the avulsion of his gastric artery. The average juror cannot be expected to know the internal bodily reactions necessary to cause a gastric artery to avulse off of the spleen, and plaintiff did not provide adequate medical expert testimony on this topic. Plaintiff's expert provided " 'an explanation consistent with [the] known facts or conditions, but not deducible from them as a reasonable inference,' " otherwise known as a conjecture, which explanation is insufficient to establish causation. *Skinner*, 445 Mich at 164, quoting *Kaminski*, 347 Mich at 422.

When questioned about the duration of VOCs exposure necessary to trigger an avulsion, Dr. Nosanchuk speculated that the injury could have occurred suddenly or "[m]aybe" "minor micro injuries" occurred over time, but he acknowledged, "I don't know what happened," followed by: "I can't really comment on the-- I felt this is what did it and it happened. As far as why it took that long, I don't know." When asked if anything in the medical literature supported his testimony, he stated, "Not as much as my experience and my clinical judgment." Even if Dr. Nosanchuk's experience was relevant, he still failed to offer the fact-finder any rationale for his conclusions and, in fact, likely undercut those conclusions by repeatedly saying that he did not "know" why he reached them.

Regarding what may have caused the avulsion of plaintiff's gastric artery, Dr. Nosanchuk had to "look this up" because he was "not an anatomist"-- an expert in the structure or internal workings of the human body. He reviewed the abstracts of three articles that he considered to be relevant. The abstracts list several potential causes of gastric artery tears and note that "*rarely* [is] vomiting" a predisposing condition. (Emphasis added.) Dr. Nosanchuk made no attempt to explain why plaintiff's avulsion was among those rare cases in which coughing or vomiting caused the injury as opposed to other possibilities.

In explaining what he relied on to form his ultimate opinion concerning the cause of plaintiff's avulsion, he testified: "[T]here was an oil spill. That's a known fact. There [were] fumes. That was a known fact. People got sick and some of them coughed, had nausea, and vomiting. It was the-- without anything specific, the total of that is what I based my opinion on. . . . I'm a very simple guy. Spill, fumes, sick people, to me they're related based on 40 years doing this for a living." In other words, the oil spill caused the injuries because it occurred before the injuries. A fact-finder could not rely on this rationale to reach a verdict favoring plaintiff. See *West*, 469 Mich at 186 n 12.

Overall, Dr. Nosanchuk provided only correlative reasoning based on his "clinical judgment." For purposes of this Court's review, the problems associated with his testimony are not ones of reliability or soundness of methodology, but rather speak to whether he produced any evidence tending to show that defendants' oil fumes more likely than not caused the avulsion of plaintiff's gastric artery.[23] I, in agreement with the majority, conclude that he did not. *Wright*, 91 F3d at 1107 (stating that it is "not enough for a plaintiff to show that a certain chemical agent sometimes causes the kind of harm that he or she is complaining of"). Plaintiff's expert did not show that a causal relationship between defendants' VOCs and plaintiff's arterial tear was more probable than not, and plaintiff thereby failed to create a genuine issue of fact as to specific causation. *Skinner*, 445 Mich at 174 ("Because the experts' conclusions regarding causation are premised on mere suppositions, they did not establish an authentic issue of causation."). Therefore, the trial court properly granted summary disposition in defendants' favor.

III. CONCLUSION

A toxic tort is no different than any other negligence claim in that a plaintiff must present evidence establishing factual or "but for" causation. Where, as here, a plaintiff presents evidence in the form of group-based statistical studies or similar proof, the general-and-specific-causation framework would apply. Evidence of general causation must include proof that the toxin in the alleged exposure level can cause the alleged harm. Evidence of specific causation must include proof that the plaintiff was actually exposed to the relevant toxin as well as a rough estimation of his or her exposure level. Specific causation additionally requires a plaintiff to evaluate and eliminate to a reasonable extent other reasonably relevant potential causes of his or her injuries. Furthermore, if the issue or proposition in a toxic tort case is beyond the common

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[23] When considering scientific evidence of specific causation in a toxic tort case at the summary disposition stage, "the question is not whether there is some dispute about the validity or force of a given study, but rather, whether it would be unreasonable for a rational jury to rely on that study to find causation by a preponderance of the evidence." *In re Joint Eastern & Southern Dist Asbestos Litigation*, 52 F3d 1124, 1133 (CA 2, 1995).

knowledge of an ordinary juror, expert witness testimony is required. Because plaintiff here failed to present evidence establishing either general or specific causation, I concur in the Court's reversal of the judgment of the Court of Appeals and remand to the trial court for reinstatement of its order granting summary disposition in defendants' favor.

ZAHRA, J., and WILDER, J., join the statement of MARKMAN, C.J.