

In the Supreme Court
Appeal from the Court of Appeals
Jansen, P.J., and Meter and Beckering, JJ.

CHANCE LOWERY,

Plaintiff-Appellee,

Docket No. 151600

v.

ENBRIDGE ENERGY, LIMITED
PARTNERSHIP and ENBRIDGE ENERGY
PARTNERS, L.P.,

Defendants-Appellants.

**BRIEF ON APPEAL – DEFENDANTS-APPELLANTS ENBRIDGE ENERGY,
LIMITED PARTNERSHIP AND ENBRIDGE ENERGY PARTNERS, L.P.**

ORAL ARGUMENT REQUESTED

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STATEMENT OF THE BASIS OF THE COURT’S JURISDICTION

On November 8, 2013, the Calhoun County Circuit Court issued its “Order Granting Defendants’ Motion for Summary Disposition.” (App 246a). Pursuant to MCR 7.203(A) and MCR 7.204(A), Plaintiff filed a timely claim of appeal to the Court of Appeals, which reversed the trial court’s decision in a 2-1 opinion issued on April 2, 2015. (App 249a).

Defendants Enbridge Energy, Limited Partnership and Enbridge Energy Partners, L.P.’s (“Enbridge”) timely applied for leave to appeal on May 14, 2015, and this Court granted Enbridge’s application for leave to appeal on March 30, 2016. (App 254a). This Court has jurisdiction over Enbridge’s appeal pursuant to MCR 7.303(B)(1).

STATEMENT OF QUESTIONS INVOLVED

In granting leave to appeal, the Court directed the parties to 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. See *Genna v Jackson*, 286 Mich App 413 (2009).” These questions encompass the heart of this case, and Enbridge will address them in the following order:

(1) “[W]hether the plaintiff was required to present expert witness testimony regarding general and specific causation.”

In a 2-1 decision, the Court of Appeals majority answered: No.

The Court of Appeals dissent answered: Yes.

The trial court would answer: Yes.

Enbridge answers: Yes.

(2) “[W]hether the plaintiff in this toxic tort case sufficiently established causation to avoid summary disposition under MCR 2.116(C)(10).”

In a 2-1 decision, the Court of Appeals majority answered: Yes.

The Court of Appeals dissent answered: No.

The trial court would answer: No.

Enbridge answers: No.

I. INTRODUCTION

The necessity for and sufficiency of expert testimony on causation, both in this case and in toxic tort cases in general, requires definitive guidance from this Court. While the Court of Appeals in *Genna v Jackson*, 286 Mich App 413; 781 NW2d 124 (2009), “decline[d] to adopt” a blanket requirement of the need for expert testimony, *id.* at 418, *Genna* involved a unique set of facts. In *Genna*, the plaintiffs’ children became seriously ill after being exposed to “massively high levels” of mold toxins inside their home, and quickly recovered as soon they moved out. Under those particular circumstances, *Genna* held that causation could be reasonably inferred without the assistance of an expert.

But *Genna* is not the typical toxic tort case. And the *Genna* reasoning and holding are not controlling in cases like this one. This case, like most toxic tort cases, does not involve a plaintiff’s acknowledged exposure to “massively high levels” of a toxic substance in a confined space, resulting in immediate symptoms that were relieved once the plaintiff was no longer exposed. Here, there was a release of crude oil from Enbridge’s Line 6B oil pipeline into a vacant woodland area located near Marshall, Michigan. That crude oil eventually migrated into the Talmadge Creek and then the Kalamazoo River. Because of existing flood conditions, the oil traveled rapidly down the river for approximately 35 miles, releasing volatile organic compounds (“VOCs”) into the air along the way. At no time was there a mandatory evacuation of the area (including near the release site) due to health concerns or dangers.

At the time of the incident, Plaintiff Chance Lowery *lived more than ten miles away* from the release site. Yet Lowery claims that *more than three weeks* after the incident, and *more than one week* after the smell of oil went away, the VOCs from the oil caused him to experience headaches, nausea, and vomiting that was so severe that it led to the “avulsion” (or tearing away) of his gastric artery.

In support of his claim, Lowery relied on the testimony of a family medicine doctor (and his attorney's lifelong friend), Jerry Nosanchuk, D.O. Dr. Nosanchuk acknowledged that he had no training or experience in either toxicology or vascular medicine, that he did not have "any idea" about the levels of VOC exposure necessary to cause the symptoms that Lowery alleged, and did not know or attempt to ascertain his actual level of exposure, if any, to toxic chemicals. Dr. Nosanchuk failed to review any of the thousands of air monitoring and sampling results taken under the direction of the United States Environmental Protection Agency ("EPA") (the federal agency in charge of directing and monitoring the clean-up of the oil release), which measured the VOCs throughout the area for months after the release. Instead, Dr. Nosanchuk *assumed* that exposure to oil fumes caused Lowery's symptoms because "[h]e wasn't having the problems before and he was having the problems after."

Dr. Nosanchuk reached that conclusion without examining Lowery and despite the fact that: (1) Lowery has a history of migraine headaches and nausea that he and his doctors have long attributed to his use of the antidepressant drug Lamictal; (2) Lowery told emergency room doctors and the surgeon who repaired his artery that he had taken a Vicodin just before the vomiting began, and that he thought the Vicodin had caused it;¹ and (3) vomiting is a recognized side effect of Vicodin. Despite those potential alternative causes for Lowery's headaches, nausea, and vomiting, Dr. Nosanchuk testified in his deposition that he could not remember considering "anything specifically," and that he rejected other potential causes based solely on his "clinical judgment."

The trial court concluded that Lowery could not "link up the etiology of the ruptured aorta [sic]" and granted summary disposition in Enbridge's favor, but the Court of Appeals

¹ As a result, Lowery was even reluctant to take Vicodin after his surgery.

reversed in a 2-1 opinion. (App 249a). The Court of Appeals majority acknowledged Enbridge's position that Dr. Nosanchuk's testimony was "inadequate" to establish causation, but concluded that expert testimony *was not even necessary* because there was 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." The Court of Appeals dissent argued that Lowery's "theory of causation was attenuated," and that "without sufficient expert testimony on the issue of causation, [Lowery] could not establish a genuine issue of material fact concerning whether the Kalamazoo River oil spill proximately caused his ruptured artery and internal bleeding."

The Court of Appeals dissent had it exactly right. Although this Court has not yet addressed the issue, courts from other jurisdictions have widely recognized that in order to establish causation in a toxic tort case, a plaintiff is required to provide evidence of exposure to toxic chemicals at a level that was harmful and known to cause the symptoms being alleged (general causation), and that the plaintiff's exposure was in fact the cause of his or her symptoms (specific causation). As the Sixth Circuit has explained, causation in toxic tort cases involves "scientific assessments that must be established through the testimony of a medical expert." *Pluck v BP Oil Pipeline Co*, 640 F3d 671, 677 (CA 6, 2011).

Although there may be unique cases where expert testimony is not needed to establish this causal link, those are the exception, not the rule. And this case is not an exception to the rule. Lowery, who lived miles downriver from the release site, claims that the headaches, nausea, and vomiting that led to the rupture of his gastric artery occurred more than three weeks after the oil leak and more than a week after he acknowledged the odor from the release was gone. As the Court of Appeals dissent observed, Dr. Nosanchuk conceded that he did not know

“the medical effects of exposure to toxic chemicals and volatile organic compounds, and that he had never treated a patient with a ruptured abdominal artery resulting in internal bleeding.” More importantly, Dr. Nosanchuk conceded that he did not know or attempt to ascertain Lowery’s actual level of exposure to VOCs, despite the availability of thousands of individual air sampling and air monitoring results taken throughout the area.

Instead, Lowery’s expert simply ignored all of that data, and merely assumed that exposure to oil fumes must have caused Lowery’s alleged symptoms since (1) there was an oil release and (2) Lowery claimed to have experienced symptoms (even though his symptoms were not new to him and were readily explained by other potential causes). That is a classic example of the logical fallacy *post hoc ergo propter hoc*, i.e., the assertion of a cause and effect relationship simply because one event follows the other in time. This Court, and others around the country, have consistently rejected such an approach to causation. See, e.g., *Craig v Oakwood Hosp*, 471 Mich 67, 93; 684 NW2d 296 (2004) (“[I]t is error to infer that A causes B from the mere fact that A and B occur together.”); *Higgins v Koch Development Corp*, 794 F3d 697, 703-704 (CA 7, 2015) (rejecting “the fallacy of saying that because effect A happened at some point after alleged cause B, the alleged cause was the actual cause”) (citation and internal quotation marks omitted).

As a result, the Court of Appeals majority erred in relying on *Genna* to find “genuine issues of material fact to be resolved by the jury.” Not only must *Genna* be read consistently with *Craig*, but *Genna* does not suggest that causation can *ever* be established in the absence of *any* evidence of exposure. Instead, *Genna* simply stands for the unremarkable proposition that in limited circumstances where there is sufficient circumstantial evidence of exposure allowing a reasonable inference of causation, expert testimony may not be necessary. That simply is not the

case here given that, unlike in *Genna*, there is no evidence that Lowery was *ever* exposed to *any* VOCs, let alone at a level that could cause the bout of vomiting that Lowery claims to have experienced three weeks after the oil leak and that he blames for the rupture of his gastric artery. As the Court of Appeals dissent correctly recognized, expert testimony was necessary to establish both (1) that “fumes from the oil spill caused plaintiff’s vomiting, and (2) that “plaintiff’s vomiting caused his resulting vascular injury.”

Because Lowery failed to provide sufficient evidence of causation to create a genuine issue of material fact for trial, Enbridge requests that the Court reverse the Court of Appeals’ decision and reinstate the trial court’s decision granting summary disposition to Enbridge.

II. FACTUAL AND PROCEDURAL BACKGROUND

A. A release from Enbridge’s Line 6B oil pipeline resulted in a discharge of crude oil that eventually migrated into the Kalamazoo River.

On July 26, 2010, Enbridge Energy, Limited Partnership reported a release on its Line 6B oil pipeline, approximately one mile downstream of Enbridge’s pumping station in Marshall, Michigan. The crude oil was initially released into a vacant woodland area, then flowed into Talmadge Creek and eventually migrated to the Kalamazoo River. (See Defs’ Mot for Summ Disp at 4-5, App 16-17a). Because the river was in flood stage at the time, the oil was carried more than 35 miles downstream through Calhoun and Kalamazoo counties, to where the Kalamazoo River meets Morrow Lake.² As the crude oil was traveling downstream, volatile

² The Court can appropriately take judicial notice of this fact, which is “not subject to reasonable dispute” in that it is “capable of accurate and ready determination by resort to sources whose accuracy cannot reasonably be questioned.” MRE 201(b)(2). This and other background information concerning the Line 6B incident is widely available from reputable sources, including a section of the EPA’s website dedicated to providing information about the incident. See *EPA Response to Enbridge Oil Spill*, <<https://www3.epa.gov/region5/enbridgespill/>> (accessed May 1, 2016) (“Heavy rains caused the river to overtop existing dams and carried oil 35 miles downstream on the Kalamazoo River”); *In re Application of Indiana Michigan Power*

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organic compounds (“VOCs”) were emitted from the crude oil and dissipated into the air. It was the alleged exposure to these VOCs that Plaintiff claims caused his symptoms.

Within hours of the release, air sampling and monitoring was commenced under the control and direction of the EPA to assess potential health hazards and to determine whether an evacuation of the area was warranted. Although the Calhoun County Public Health Department issued voluntary evacuation notices for residents living in the immediate vicinity of the release site, there was no mandatory evacuation for any resident. There also was no evacuation notice or order of any kind (voluntary or mandatory) near where Lowery resided. The air sampling and monitoring continued for months and resulted in thousands of data points measuring the VOCs that had been released from the crude oil into the air.³

B. Plaintiff Chance Lowery sued claiming that he suffered headaches, nausea, and vomiting as a result of exposure to toxic fumes, and that a fit of vomiting led to the rupture of his gastric artery.

At the time of the incident, Lowery lived at 279 Silver Street in Battle Creek (Defs’ Mot for Summ Disp at 5, App 17a), which is near the Kalamazoo River but about thirteen miles downstream from the release site. Lowery testified at his deposition that he smelled oil “within 12 hours” of the July 26, 2010 pipeline leak, that the smell was strong for “three to five days,” and that it went away after “about five to seven days.” (Deposition of Chance Lowery at 42-43,

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Co, 275 Mich App 369, 371 n 2; 738 NW2d 289 (2007) (taking judicial notice of statements contained on the U.S. Department of Energy’s website).

³ Once again, the Court can take judicial notice of this EPA-directed sampling and monitoring, as well as the issuance of voluntary evacuation notices. See *EPA Response to Enbridge Oil Spill – Data*, <<https://www3.epa.gov/region5/enbridgespill/data/index.html>> (accessed April 28, 2016) (containing air quality and sampling data collected along the Talmadge Creek, Kalamazoo River, and Morrow Lake); *EPA Response to Enbridge Oil Spill – Questions and Answers* <<https://www3.epa.gov/region5/enbridgespill/qanda.html>> (accessed May 1, 2016) (“In some areas affected by the spill, Calhoun County Public Health Department issued voluntary evacuation notices based on the level of benzene measured in the air.”).

App 32a).⁴ On the second day, Lowery claimed that he started getting migraine headaches that were so severe he was “bedridden” ten hours a day. (*Id.* at 46-47, 49, App 33-34a).⁵ Lowery testified that the migraines lasted for “five or seven days,” and that he was also “vomiting non stop practically the whole five to seven days.” (*Id.* at 46, 49, App 33-34a).

On either August 17 or 18, 2010 (more than three weeks after the oil release), Lowery apparently had another migraine and took a Vicodin that he “got off a friend.” (*Id.* at 47-49, App 33-34a).⁶ After vomiting again, he felt severe abdominal pain. (*Id.* at 47-49, App 33-34a). Lowery drove himself to Bronson Battle Creek Hospital, where he had emergency surgery on August 18, 2010 to repair a ruptured gastric artery that was causing internal bleeding. (See Bronson Hospital medical records, App 41a).⁷

⁴ In response to Enbridge’s motion for summary disposition, Lowery submitted an affidavit stating that the smell of oil was “almost unbearable” for “two weeks.” (App 176a). It is well established, however, that a party may not “contrive factual issues” by contradicting his or her own deposition testimony in a later-filed affidavit. *Kaufman & Payton, PC v Nikkila*, 200 Mich App 250, 257; 503 NW2d 728 (1993).

⁵ This testimony was contradicted by that of Lowery’s friend, Michael Condon, who said that during the three weeks between the oil incident and Lowery’s surgery, he and Lowery met up every couple of days because Lowery did not want to be at home near the smell of oil. (Michael Condon Dep at 8-10, App 43-45a). Condon further testified that during the time period when Lowery claims to have been bedridden all day, they met to play disc golf, basketball, watch movies, or hang out at Condon’s house. (*Id.* at 9, 25, App 44a, 50a).

⁶ We know that this occurred on one of these two dates because, as discussed below, Lowery went to the hospital on August 18, 2010. In his deposition, Lowery said that he could not remember whether he took the Vicodin “the same day or the day before.” (*Id.* at 48, App 33a). But he told his surgeon that he took the Vicodin earlier that day, and that it was what caused him to vomit. (Deposition of John Koziarski, M.D., at 16-17, App 58-59a).

⁷ In its opinion, the Court of Appeals majority suggested that a “reasonable reading” of Lowery’s deposition testimony was that “he had an approximately weeklong spell of severe migraines that started the day after the spill and then, approximately a week after that, he experienced a several-days-long bout of vomiting.” (COA Op at 3, App 251a). Although Lowery’s account of his alleged migraines and vomiting – *allegedly occurring for five to seven days after he first smelled oil* – clearly does not match up with the date of his hospital admission *two weeks* later, no

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Lowery's surgeon, John Koziarski, M.D., who is board-certified in both surgery and phlebology (i.e., vein diseases), testified that he could not determine the cause of Lowery's ruptured gastric artery:

Q. And in this instance, you testified earlier that you could not determine the actual medical cause of Mr. Lowery's torn artery or avulsed artery; is that correct?

A. That is correct. [Deposition of John Koziarski, M.D., at 8, 36-37, App 70-71a.]

C. Lowery has a history of migraine headaches and nausea.

During discovery, it was revealed that Lowery actually has a history of migraines and nausea. Lowery has long suffered from depression and bipolar disorder, and was being treated with an antidepressant drug called Lamictal. (Lowery Dep, 34-35, 37, App 30-31a; see also Summit Pointe medical records, App 36-40a). Lowery's medical records from Summit Pointe Community Mental Health in Battle Creek contain various references to his complaints about getting headaches, nausea, and "dry heaves" from his Lamictal, especially when he smoked cigarettes or was around smoke, as well as migraines when "stressed" and from "impacted wisdom teeth":⁸

November 29, 2007 "Medication Review"

I believe I have him diagnosed as a bipolar disorder and started him on a trial of Lamictal. He comes in today saying that Lamictal at 100 mg a day is helpful to him. He says in the past where he would have gone off or been upset he is calmed by the medication. However he has nausea and dry heaves [sic] however it only occurs if he smokes or is around smoke.

* * *

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"reasonable reading" of Lowery's testimony suggests that he had an entirely separate "several-days-long bout of vomiting" leading up to the rupture of his artery on August 18, 2010.

⁸ Lowery smoked medical marijuana and was a regular cigarette smoker. (Lowery Dep at 39-40, 57- App 31a, 35a).

I discussed with the patient that the medication appears helpful that it should not be stopped, that he should stop smoking and to continue to take the Lamictal

January 16, 2008 “Medication Check”

Chief complaints:

Morning headache/nausea with Lamictal increased from 75 to 100 mg. daily

February 11, 2010 “Summit Pointe Assessment”

Gets migraines when stressed . . .

* * *

. . . He has . . . “something like migraines” which he attributes to impacted wisdom teeth. . . .

D. Lowery told emergency room doctors and his surgeon that an antidepressant drug was causing migraines, and that he vomited after taking Vicodin to relieve one of them; he never mentioned the oil leak.

Lowery’s history of migraines and difficulties with Lamictal are also documented in his Bronson Hospital records, and were confirmed by his surgeon. Lowery told Dr. Koziarski that he believed the Lamictal was causing his migraines, and that he became nauseous and vomited after taking a Vicodin:

Q. And what did you learn from Mr. Lowery regarding his abdominal pain?

A. That it came on suddenly. He had taken some Vicodin earlier that day for a migraine headache and then started vomiting, and then following that became dizzy and lightheaded and then developed this severe abdominal pain.

* * *

Q. . . . Did Mr. Lowery indicate to you what he thought caused him to vomit?

A. He said that he thought it was from the Vicodin.

* * *

Q. Okay. This record also indicates “He is going to restart on his Lamictal that he was taking as an outpatient for bipolar disorder.” Do you see that?

A. Yes.

Q. And “He is wondering about changes to his Lamictal, as this may be causing his migraines.” Do you see that as well?

A. Yes.

Q. And is this something that was reported by Mr. Lowery to you?

A. Yes. [See Koziarski Dep at 16-17, 22, App 58-59a, 63a; Bronson Hospital records, “8/20/10 Progress Notes,” App 41a.]

In fact, Lowery was so convinced that the Vicodin caused his vomiting that he was reluctant to take it after his surgery when he complained of yet another migraine. As reported in a progress note two days after Lowery’s surgery:

He is starting to get a migraine again. He is reluctant to take Norco or Vicodin as this is what made him throw up the first time. . . .⁹

Based on Lowery’s reports that his Lamictal was causing his migraines, Dr. Koziarski requested a psychiatric consult with Dr. Anoop Thakur to determine if the medication could be changed. (Koziarski Dep at 22-23, App 63-64a). Lowery’s hospital records make no mention of the oil leak, and Dr. Koziarski testified in his deposition that when Lowery came to the hospital on August 18, 2010, he did not say anything about exposure to oil causing any of his symptoms:

Q. At any time did Mr. Lowery indicate that the odor or the fumes in the Kalamazoo River caused his migraine headache?

A. No.

Q. Did Mr. Lowery at any time indicate to you during your treatment of him that odor and fumes from oil in the Kalamazoo River caused his vomiting?

A. No.

Q. Did Mr. Lowery ever indicate to you, during his hospital admission, that he had severe coughing?

A. No.

⁹ See Bronson Hospital records, “8/20/10 Progress Notes,” App 41a; Koziarski Dep at 17, 20-21, App 59a, 61-62a.

- Q. Okay. And did Mr. Lowery at any time indicate that odor or fumes from the oil in the Kalamazoo River caused severe coughing?
- A. No. [Koziarski Dep at 24-25, App 65-66a.]
- E. The trial court granted summary disposition to Enbridge, concluding that Lowery failed to present expert testimony establishing a causal connection between the oil leak and the rupture of his gastric artery.**

On September 30, 2013, Enbridge filed a motion for summary disposition arguing that Lowery did not have sufficient evidence to establish a causal link between the oil leak and either his alleged headaches and vomiting or the rupture of his gastric artery. (See Defs' Mot for Summ Disp, App 11a).¹⁰

In response, Lowery relied on the testimony of his medical expert, Jerry Nosanchuk, D.O., who opined that "fumes from the oil spill caused Chance Lowery 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 abdominal cavity." (See Jerry L. Nosanchuk, M.D. "Record Review and Opinion," p 3, App 150a; see also Deposition of Jerry Nosanchuk, M.D., at 48, App 93a).¹¹ Lowery also relied on (1) general information from a Centers for Disease Control "Pocket Guide" about the VOCs found in crude oil (including benzene, xylene, and toluene), (2) testimony from one of his neighbors in a different case about a "bad smell" after the oil leak, (3) testimony from a friend, David Condon, that he could smell oil at Lowery's house, and (4) the fact that Lowery and his girlfriend, Ashlee Green, saw cleanup

¹⁰ On October 7, 2013, the trial court granted partial summary disposition to Lowery as to the duty and breach elements of his negligence claim, leaving only proximate cause and damages issues.

¹¹ At the time Enbridge filed its motion for summary disposition, only the "rough" transcript of Dr. Nosanchuk's deposition was available. As a result, the page numbering of that transcript is slightly different than for the additional excerpts attached to Plaintiff's answer to Enbridge's motion and to Enbridge's reply brief.

workers in “hazmat suits.” (See Plaintiff’s Ans to Defs’ Mot for Summ Disp, App 131a, and Supp to Pls’ Reply Br to Defs’ Mot for Summ Disp, App 223a).

In its reply brief, Enbridge argued that none of Lowery’s evidence was sufficient to create a genuine issue of material fact regarding causation, as there was no evidence that Lowery had been exposed to VOCs at a level sufficient to cause the symptoms he was alleging. (See Reply Br in Supp of Defs’ Mot for Summ Disp, App 180a). As for Lowery’s reliance on Dr. Nosanchuk’s testimony, Enbridge argued that he lacked training or experience in either toxicology or vascular surgery, and that his causation opinion was speculative because he did not know anything about Lowery’s actual exposure to VOCs, if any, and failed to properly rule out alternative causes for his headaches and vomiting, such as Lowery’s use of Lamictal and Vicodin. (*Id.*).

On November 4, 2013, the trial court held a hearing on Enbridge’s motion for summary disposition. (See November 4, 2013 Hrg Tr, App 230a). The trial court was satisfied that Lowery had sufficient evidence to connect his “vomiting and headaches” to the oil leak, but that Lowery had failed to “link up the etiology of [his] ruptured aorta [sic]”:

THE COURT: But in the Defendant’s brief they say medical records from Mr. Lowery’s hospitalization which contain contemporaneous statements of his condition indicate that he never even mentioned to any of his doctors that the fumes from the oil, were allegedly causing him so much discomfort and illness. Instead he told doctors that he thought the migraines were caused by his bipolar medication, and that the nausea and vomiting was caused by Vicodin he had been taking. Since Mr. Lowery has no evidence of causation either specific, specific or general, Enbridge is entitled to summary disposition of his negligence claim.

I will acknowledge that in other cases I have made the determination based upon the witnesses in those cases that exposure can cause headaches and general discomfort, causing people to go to their doctor. That first chasm has been bridged, but to go from that point to surgery, how do I get there . . . ?

* * *

What I'm going to do, gentlemen, I will grant partial summary disposition as it relates to any ailment or physical problem that Mr. Lowery had beyond the vomiting and headaches. I just don't have anything, Mr. Bloom, to link up the etiology of ruptured aorta [sic]. [*Id.* at 12-15, App 241-244a.]¹²

Although the trial court initially intended to grant partial summary disposition only as to Lowery's claim relating to his ruptured gastric artery, Mr. Lowery's counsel requested that the trial court grant summary disposition in its entirety because "we never really made a claim for the nausea and headaches . . . this whole case is all about the surgery, so if you are going to grant the motion, grant it totally, so that I can then appeal it." (*Id.* at 14, App 246a). The trial court thus granted summary disposition as to all of Lowery's claims, and a final order was entered on November 8, 2013. (See Order Granting Defs' Mot for Summ Disp, App 243a).

F. In a 2-1 decision, the Court of Appeals majority reversed, holding that Lowery did not need expert testimony and that his claim "goes beyond mere speculation."

On April, 2, 2015, the Court of Appeals issued a 2-1 opinion reversing the trial court's decision and remanding the case for further proceedings. (COA Op, App 249a). The Court of Appeals majority, citing *Genna v Jackson*, 286 Mich App 413; 781 NW2d 124 (2009), concluded that Lowery did not need "direct expert testimony" to prove causation because "there was 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 2, App 250a). Though the Court of Appeals majority acknowledged "that there are other plausible explanations for plaintiff's injury," the majority reasoned that "this

¹² Enbridge of course disagrees with the trial court's suggestion that a causal connection between VOC exposure and Lowery's alleged headaches, nausea, and vomiting could be established simply by pointing to claims being made by residents in other cases. Not only is there no record evidence concerning those residents' claimed exposures, but as discussed further below, Lowery was required to provide evidence that *he* was exposed to VOCs at a level sufficient to cause *his* alleged symptoms.

only serves to highlight that there are genuine issues of material fact to be resolved by a jury.” (*Id.* at 3, App 251a).

Judge Kathleen Jansen dissented, arguing that Lowery needed expert testimony because “whether the fumes released by the oil spill caused plaintiff’s vomiting, and whether plaintiff’s vomiting in turn caused his abdominal artery to rupture, are not matters within the common understanding of average jurors.” (COA Dissent at 1, App 252a). The dissent further argued that Dr. Nosanchuk was not qualified to opine on causation, and that “[w]ithout sufficient expert testimony,” a jury could only “speculate on the issue of causation.” (*Id.* at 2, App 253a).

III. SUMMARY OF ARGUMENT

Despite the Court of Appeals majority’s assertion, Lowery was required to provide reliable expert testimony in support of his claims. Both this Court and the Court of Appeals have recognized the importance of expert testimony in resolving questions that are beyond the “common understanding” of jurors. Although this Court has not had occasion to address the extent to which expert testimony must be provided in toxic tort cases, courts in other jurisdictions consistently recognize that expert testimony is crucial in resolving the complex causation questions that arise in such cases. This case exemplifies the need for expert testimony. As the Court of Appeals dissent understood, whether VOCs from the oil caused Lowery to suffer headaches, nausea, and vomiting more than three weeks after the oil leak and more than a week after Lowery said the smell of oil went away, and whether Lowery’s vomiting led to the rupture of his gastric artery, “are not matters within the common understanding of average jurors.” (COA Dissent at 1, App 252a). Instead, they require the “specialized knowledge” of a qualified expert. (*Id.*).

Lowery’s failure to present reliable expert evidence is fatal to his claims. Although Lowery’s medical expert, Dr. Nosanchuk, asserted a causal connection between the oil leak and

Lowery's alleged headaches, nausea, and vomiting, it was pure speculation on Dr. Nosanchuk's part. Dr. Nosanchuk conceded that he did not even attempt to ascertain whether Lowery was exposed to VOCs at a level sufficient to cause his symptoms. Instead, Dr. Nosanchuk assumed that Lowery must have been exposed because he claimed to have exhibited symptoms consistent with exposure and generally defaulted to his "clinical judgment" to disregard alternative causes. Courts, however, have consistently rejected the notion that a mere *correlation* between exposure to a toxic substance and the onset of symptoms is sufficient to demonstrate a *causal connection*. This is especially true here, as the temporal connection is weak and there are other equally (if not more) plausible and intervening explanations for the alleged symptoms. Lowery has a history of migraine headaches and nausea when taking the antidepressant drug Lamictal, and he told his doctors that the vomiting he experienced on the day his gastric artery ruptured was caused by a Vicodin he had just taken. Lowery's expert simply ignored all of this evidence and summarily concluded that in his "clinical judgment," the oil must have caused the symptoms.

And even if it could be said that there was sufficient evidence to create a genuine issue of material fact as to whether VOC exposure caused Lowery's alleged headaches, nausea, and vomiting, a jury could only speculate as to whether it had anything to do with the avulsion of his gastric artery. Lowery's own surgeon did not determine the cause of the rupture, and while Dr. Nosanchuk once again claims to have relied on his "clinical judgment" in opining that it was caused by Lowery's alleged vomiting, he provided no support whatsoever. Without reliable expert testimony, Lowery failed to create a genuine issue of material fact as to whether the oil leak caused his injuries. Thus, the trial court properly granted summary disposition to Enbridge.

IV. ARGUMENT

A. Standard of Review

This Court reviews de novo a trial court's decision on a motion for summary disposition under MCR 2.116(C)(10). *Coblentz v City of Novi*, 475 Mich 558, 567; 719 NW2d 73 (2006). "In reviewing a ruling made under this court rule, a court tests the factual support [for a plaintiff's claim] by reviewing the documentary evidence submitted by the parties." *Id.* The Court "review[s] the evidence and all legitimate inferences in the light most favorable to the nonmoving party." *Id.* at 567-568. "Where the proffered evidence fails to establish a genuine issue regarding any material fact, the moving party is entitled to judgment as a matter of law." *Id.* (citations and internal quotation marks omitted).

B. Lowery was required to present qualified expert testimony regarding general and specific causation.

This Court has never specifically addressed the requirements for establishing causation in a toxic tort case, but many other courts have. It is widely recognized that in order to demonstrate causation in such a case, "the plaintiff must establish both general and specific causation through proof that the toxic substance is capable of causing, and did cause, the plaintiff's alleged injury." *Pluck v BP Oil Pipeline Co*, 640 F3d 671, 677 (CA 6, 2011). Although it applied Ohio law, the Sixth Circuit's decision in *Pluck* is representative of the general approach to causation "in a toxic tort case." See *In re Dow Corning Corp*, 541 BR 643, 654 (ED Mich, 2015) (citing *Pluck* as persuasive authority in the absence of controlling Michigan case law on the issue).¹³

¹³ See also *Golden v CH2M Hill Hanford Group, Inc*, 528 F3d 681, 683 (CA 9, 2008) ("To survive summary judgment on a toxic tort claim for physical injuries, Golden had to show that he was exposed to chemicals that could have caused the physical injuries he complains about (general causation), and that his exposure did in fact result in those injuries (specific causation)."); *Bonner v ISP Techs Inc*, 259 F3d 924, 928 (CA 8, 2001) ("[T]o 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

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As *Pluck* further explained, “[b]oth causation inquiries involve scientific assessments that *must* be established through the testimony of a medical expert.” *Pluck*, 640 F.3d at 677 (emphasis added). “Without this testimony, ‘a plaintiff’s toxic tort claim will fail.’” *Id.* (citation omitted). Other federal and state courts have similarly recognized the need for expert testimony to establish that a plaintiff’s alleged injuries were caused by exposure to a toxic substance. For example, in *Higgins v Koch Development Corp*, 794 F3d 697 (CA 7, 2015), the plaintiff was exposed to a “cloud of chlorine gas” after an equipment malfunction at an outdoor waterpark. *Id.* at 700. He immediately experienced “chest tightness, burning eyes, shortness of breath, and nausea,” and was diagnosed at the hospital with “mild chemical exposure.” *Id.* More than a year later, the plaintiff visited a pulmonologist and was diagnosed with reactive airways dysfunction and chronic asthma. *Id.* In holding that the plaintiff needed expert testimony to support his claim that these long-term conditions were “a consequence of inhaling chlorine gas” at the waterpark, the Seventh Circuit observed that “a typical layperson does not possess the requisite knowledge to draw a causative line, without the assistance of a medical expert, between a brief encounter with chlorine gas and the onset of either RADS (a disease with which, we are confident, most lay people have no familiarity) or asthma.” *Id.* at 702.

The Second Circuit reached the same conclusion in *Wills v Amerada Hess Corp*, 379 F3d 32 (CA 2, 2004), finding “the causal link between exposure to toxins and other behavior and squamous cell carcinoma” to be “sufficiently beyond the knowledge of the lay juror that expert testimony is required to establish causation” in cases brought under the federal Jones Act. *Id.* at 46. As the Eleventh Circuit observed in *Rink v Cheminova, Inc*, 400 F3d 1286 (CA 11, 2005)

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plaintiff, and that the toxin was the cause of the plaintiff’s injury.”); *Neal v Dow Agrosciences LLC*, 74 SW3d 468, 472 (Tex App, 2002) (“In toxic tort litigation, causation is often discussed in terms of general and specific causation.”).

(applying Florida law), “toxic tort cases . . . are won or lost on the strength of the scientific evidence presented to prove causation.” *Id.* at 1297 (citation omitted).¹⁴

This is entirely consistent with this Court’s own precedents in the medical malpractice context. In *Woodard v Custer*, 473 Mich 1; 702 NW2d 525 (2005), the plaintiff’s infant son was admitted to the Pediatric Intensive Care Unit at the University of Michigan Hospital for treatment for a respiratory problem. “When the infant was moved to the general hospital ward, physicians in that ward discovered that both of the infant’s legs were fractured.” *Id.* at 3. The plaintiff sued the hospital and the treating physician, alleging that the fractures were the result of the “the improper placement of an arterial line in the femoral vein of the infant’s right leg and the improper placement of a venous catheter in the infant’s left leg.” *Id.* Reversing the Court of Appeals’ decision that expert testimony was not necessary, this Court held that “whether a leg may be fractured in the absence of negligence when placing an arterial line . . . in a newborn’s leg is not within the common understanding of the jury.” *Id.* at 9.

Outside of the medical malpractice context, the Court of Appeals has likewise observed that because causation inquiries in toxic tort cases are “scientific in nature . . . it is to the scientific community that the law must look for the answer.” *Nelson v American Sterilizer Co*, 223 Mich App 485, 489; 566 NW2d 671 (1996) (finding expert witnesses to be “indispensable” in a case involving “whether chronic inhalation exposure to EtO causes steatohepatitis in humans”). And this Court has long recognized that determining the cause of any “physical

¹⁴ See also *In re Paoli RR Yard PCB Litigation*, 916 F2d 829, 838 (CA 3, 1990) (“Plaintiffs set out to prove that their personal injuries were proximately caused by their exposure [to PCBs] Their case depends upon expert testimony pertaining to exposure and causation.”); *Schnexnyder v Exxon Pipeline Co*, 815 So2d 156, 160 (La App, 2005) (upholding the dismissal of the plaintiffs’ claim that “exposure to crude oil vapors caused a number of physical injuries” in part because they “did not provide any expert testimony on their behalf which establish medical causation between the oil spill and alleged physical effects from the spill”).

ailment” typically calls for the assistance of experts. See *Lindley v City of Detroit*, 131 Mich 8, 10; 90 NW 665 (1902) (“Ordinarily, the testimony of experts is required to determine the cause of physical ailments.”).

In light of this Court’s own precedents concerning when expert testimony is needed, as well as established case law from other jurisdictions stressing the importance of expert testimony to establish causation in toxic tort cases, the Court of Appeals majority erred in holding that Lowery did not need expert testimony to survive summary disposition in this case. Although the majority relied on *Genna v Jackson*, 286 Mich App 413; 781 NW2d 124 (2009) in reaching that conclusion, *Genna* involved a unique set of facts that are easily distinguishable. In *Genna*, there was ample evidence of exposure such that the jury could reasonably infer causation without expert assistance. In *Genna*, the plaintiffs and the defendant lived in neighboring condominiums that “shared a foundation, walls, an attic, and a plumbing stack.” *Id.* at 415. While the defendant was away from home for several months visiting her brother in Florida, her water heater ruptured, resulting in an infestation of toxic mold. As the *Genna* Court described it:

There were patches of mold of all different colors all over the walls and ceilings in her kitchen, family room, and dining area. The hot water tank was spewing water a few feet from the shared foundation wall and there were several inches of standing water on the floor and surface mold throughout the entire basement. [*Id.*]

As a result, the defendant’s condominium was “so grossly contaminated” that “[m]old experts concluded that the interior of defendant’s condominium . . . needed to be demolished.” *Id.* at 416. The plaintiffs also presented evidence from a microbial expert who analyzed mold samples taken from *both the plaintiffs’ and the defendant’s condominiums*. The expert identified two different molds – penicillium and aspergillus – that are highly toxic, and concluded that “the levels of these two molds were unusually high, to the extent that both plaintiffs’ and defendant’s condominiums would not be healthy environments in which to live.” *Id.*

In affirming a jury verdict in the plaintiffs' favor, *Genna* found that “[w]hile no doctor was able to testify specifically that the [plaintiffs’] children were ill because of their exposure to toxic mold,” there was sufficient evidence of causation because (1) “all the microbial evidence showed massively high levels of surface and airborne mold toxins in both plaintiffs’ and defendant's condominiums,” (2) there was evidence “that the molds in the units were toxic and are known to be toxic to humans and that they can cause toxic reactions in people,” and (3) the children’s allergy doctor concluded that the mold was a “probable confounding factor,” particularly because “the children had been otherwise healthy before their mold exposure and their symptoms resolved after they moved from their home.” *Id.* at 420-421. In light of this evidence, *Genna* concluded that “[i]t does not take an expert to conclude that . . . [the defendant] more likely than not [is] responsible for [the plaintiffs’] injuries.” *Id.* at 421 (citation omitted).

In support of its decision, *Genna* cited the Sixth Circuit’s decision in *Gass v Marriott Hotel Services, Inc*, 558 F3d 419 (CA 6, 2009), a similarly unique and distinguishable case in which the plaintiffs claimed that during their stay at the defendant’s hotel, the defendant’s employees “sprayed their belongings with an unknown pesticide and filled their hotel room with toxic vapors, causing [them] to become ill.” *Id.* at 422. While the defendants were spraying the pesticides, one of the plaintiffs walked into the hotel room. That plaintiff testified that there was a “thick, horrid, acrid, putrid, odor” in the room and that the “haze of chemicals in the room was so thick that she could ‘see it, smell it, taste it, [and] feel it.’” *Id.* The other plaintiff immediately returned to the room and each plaintiff remained in the room to remove their belongings. Both of the plaintiffs began to feel sick immediately after their exposure and later endured symptoms that were consistent with exposure to the neurotoxins in pesticides. *Id.* at 422-424. Given the

unique circumstances of the plaintiffs' exposure, the Sixth Circuit found that expert testimony regarding specific causation was not necessary. *Id.* at 433.

This case is nothing like *Genna* and *Gass*. The plaintiffs in *Genna* presented evidence, including expert testimony, that their home was infested with mold to the extent that it was considered unsafe to live, while the plaintiffs in *Gass* presented evidence that they both became sick after walking into a room that had just been sprayed with pesticides containing neurotoxins known to cause the very symptoms the plaintiffs experienced within *fifteen minutes* of being exposed. *Gass*, 558 F3d at 423-424. Here, on the other hand, Lowery does not have any information regarding the levels of VOCs (if any) to which he might have been exposed in the days and weeks following the oil leak. Moreover, Lowery lived more than ten miles away from a release of oil into a rapidly flowing river (in contrast with the confined areas involved in *Genna* and *Gass*), and the vomiting that Lowery claims led to the rupture of his gastric artery occurred more than *three weeks* after the oil leak and more than a week after Lowery said the smell of oil went away. See, e.g., *Higgins*, 794 F3d at 703 (distinguishing *Gass* because “[t]he connection between the inhalation of harmful pesticides—exposure to which occurred in a confined hotel room—and [the alleged symptoms] is fairly obvious,” whereas the “causative connection” between the plaintiff’s “permanent and debilitating lung dysfunction” and exposure “to an undetermined quantity of airborne chlorine, inhaled outdoors after some unspecified interval of time following the release of the gas – is far less apparent”).

Finally, whereas there was no other plausible explanation for the plaintiffs' symptoms in *Genna* and *Gass*, here Lowery's alleged headaches, nausea, and vomiting are just as readily explained (if not more so) by his use of Lamictal and the Vicodin that Lowery himself believed was the cause of his vomiting. As other courts have recognized, expert testimony is especially

critical to establishing causation “where an injury has multiple potential etiologies.” *Wills*, 379 F3d at 46. See also *Brown v Burlington Northern Santa Fe Railway Co*, 765 F3d 765, 771 (CA 7, 2014) (“[W]hen there is no obvious origin to an injury and it has multiple potential etiologies, expert testimony is necessary to establish causation.”); *Howell v Centric Group, LLC*, 508 Fed Appx 834, 837 (CA 10, 2013) (same).

Despite the Court of Appeals majority’s assertion, Lowery’s testimony concerning his alleged exposure was not sufficient to permit a jury to make “reasonable inferences” of causation. *Genna* and *Gass* illustrate how there may be particularly compelling circumstances under which causation may be inferred without the assistance of an expert, but established case law demonstrates that this is not the norm, and it certainly is not the case here. As the Court of Appeals dissent explained, Lowery’s “theory of causation was attenuated. It required both (1) proof that the fumes from the oil spill caused plaintiff’s vomiting, and (2) proof that plaintiff’s vomiting caused his resulting vascular injury.” (COA Dissent at 1, App 251a). Those “are not matters within the common understanding of average jurors.” (*Id.*). “Because an untrained layperson would not be qualified to intelligently resolve these particular issues without enlightenment from someone with specialized knowledge of the subject, expert testimony was necessary.” (*Id.*).

C. Lowery did not sufficiently establish causation to avoid summary disposition under MCR 2.116(C)(10).

While Lowery relied on Dr. Nosanchuk in an effort to bridge the gap between the oil incident and his injury, Dr. Nosanchuk’s testimony and opinions could not do so. As further discussed below, given the lack of evidence that Lowery was actually exposed to any VOCs, as well as Dr. Nosanchuk’s failure to properly consider and rule out other potential causes for Lowery’s his alleged headaches, nausea, and vomiting, Dr. Nosanchuk’s causation theory is

entirely speculative. The same goes for the claimed causal link between Lowery's alleged vomiting and the rupture of his gastric artery. Lowery's *own surgeon* was not willing to speculate about the cause of that injury, and Dr. Nosanchuk once again offered nothing but conclusory and unsupported assertions.

1. A plaintiff in a toxic tort case must present evidence of exposure at levels sufficient to cause the plaintiff's alleged symptoms.

Courts in other jurisdictions have stressed that an expert seeking to opine on causation in a toxic tort case must have a least some evidence of the plaintiff's level of exposure. As the Sixth Circuit put it in *Pluck*, “[t]he plaintiff must show that [he or she] was exposed to the toxic substance and that the level of exposure was sufficient to induce the complained-of medical condition (commonly called a ‘dose-response relationship’).” *Pluck*, 640 F3d at 677. In making that showing, it is not enough for a plaintiff or his expert to assert the “mere existence of a toxin in the environment.” *Id.* at 679. Instead, there must be proof of actual exposure, and that “the level of exposure could cause plaintiff's symptoms.” *Pluck*, 640 F3d at 679. The Eighth Circuit aptly summarized this proof-of-exposure requirement in *Wright v Willamette Industries, Inc.*, 91 F3d 1105 (CA 8, 1996):

[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. At a minimum, we think that there 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. We do not require a mathematically precise table equating levels of exposure with levels of harm, but there must 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. [*Id.* at 1107.]¹⁵

¹⁵ See also *McClain v Metabolife Int'l Inc.*, 401 F3d 1233, 1242 (CA 11, 2005) (observing that causation “requires not simply proof of exposure to the substance, but proof of enough exposure to cause the plaintiff's specific illness”); *Mitchell v Gencorp, Inc.*, 165 F3d 778, 781 (CA 10, 1999) (“It is well established that a plaintiff in a toxic tort case must prove that he or she was

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Applying these principles, courts have consistently held that causation cannot be established without evidence demonstrating a plaintiff's exposure to potentially harmful chemicals. For example, in *Wright*, the plaintiffs claimed that they suffered "headaches, sore throats, watery eyes, running noses, dizziness, and shortness of breath" as a result of their exposure to formaldehyde emissions from the defendant's nearby plant. The Eighth Circuit reversed a jury verdict in favor of the plaintiffs because there was no evidence that they were exposed to a "hazardous level" of emissions. Thus, the jury could "only have speculated about whether the amount of formaldehyde from Willamette's plant to which each plaintiff was exposed was sufficient to cause their injuries or, indeed, any injuries at all." *Id.* at 1108.

The Fifth Circuit used a similar analysis in rejecting a plaintiff's toxic tort claim in *Moore v Ashland Chemical Inc*, 151 F3d 269 (CA 5, 1998). In that case, the plaintiff's expert opined that the plaintiff's pulmonary illness resulted from his temporary exposure to industrial chemicals while cleaning up a spill. Shortly afterward, the plaintiff began experiencing "dizziness, watery eyes, and difficulty in breathing." *Id.* at 272. A pulmonary specialist eventually diagnosed him with reactive airways dysfunction syndrome. *Id.* In support of his opinion, the plaintiff's expert relied on "the relatively short time between [the plaintiff's] exposure to the chemicals and the onset of his breathing difficulty," *id.* at 278, along with

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exposed to and injured by a harmful substance In order to carry this burden, 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.'"); *Allen v Pennsylvania Engineering Corp*, 102 F3d 194, 199 (CA 5, 1996) ("Scientific knowledge of the harmful level of exposure, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case."); *Zellers v NexTech Northeast, LLC*, 895 F Supp 2d 734, 741 (ED Va, 2012), *aff'd* 533 Fed Appx 192 (CA 4, 2013) (citing the "rule observed among several circuit courts that the plaintiff in a toxic tort case bears the burden of demonstrating her 'actual level of exposure' to the alleged toxin"); *Blanchard v Goodyear Tire & Rubber*, 190 Vt 577, 578; 30 A3d 1271 (2011) ("[P]laintiffs in toxic exposure cases must demonstrate specific causation by submitting evidence concerning 'the amount, duration, intensity, and frequency of exposure.'").

information from a material safety data sheet indicating that the chemicals “were irritating to the lungs at some level of exposure.” *Id.* Critically, however, the expert did not know the “level of exposure necessary for a person to sustain the injuries about which the MSDS warned.” *Id.* He also did not know the level of the plaintiff’s exposure to the fumes. *Id.* at 278. The Fifth Circuit affirmed the district court’s finding that the expert’s testimony was not sufficient to demonstrate causation. Because the expert “had no accurate information on the level of [the plaintiff’s] exposure to the fumes,” he “necessarily had no support for the theory that the level of chemicals to which [the plaintiff] was exposed caused [his illness].” *Id.*¹⁶

The New York Court of Appeals’ recent decision in *Sean R ex rel Debra R v BMW of North America, LLC*, 26 NY3d 801; 2016 NY Slip Op 01000; ___ NE3d ___ (2016), provides another useful example. There, the plaintiff alleged that he was born with “severe mental and physical disabilities” as a result of his “*in utero* exposure to unleaded gasoline vapor caused by a defective fuel hose in his mother’s BMW.” *Id.* at 805. In support of that claim, the plaintiff’s experts opined that he was “exposed to a sufficient amount of gasoline vapor to have caused his injuries based on the reports by plaintiff’s mother and grandmother that the smell of gasoline occasionally caused them nausea, dizziness, headaches and throat irritation.” *Id.* at 809. In upholding the lower court’s decision to exclude the plaintiff’s experts’ testimony, the New York Court of Appeals rejected their attempt to “wor[k] backwards from reported symptoms to divine an otherwise unknown concentration of gasoline vapor.” *Id.* at 810. The court acknowledged that “it is sometimes difficult, if not impossible, to quantify a plaintiff’s past exposure to a

¹⁶ See also *Johnson v Arkema, Inc.*, 685 F3d 452, 472 (CA 5, 2012) (relying on *Moore* to reject the plaintiff’s toxic tort claim because “[the plaintiff] [did] not offer any evidence that the actual amounts of tin oxide to which he was exposed were of a sufficient concentration level to cause his restrictive lung disease and pulmonary fibrosis”).

substance,” but stressed that it had “not dispensed with the requirement that a causation expert in a toxic tort case show, through generally accepted methodologies, that a plaintiff was exposed to a sufficient amount of a toxin to have caused his injuries.” *Id.* at 812.

2. Lowery and his expert failed to provide evidence of actual exposure to VOCs.

As these and other similar cases show,¹⁷ a plaintiff in a toxic tort case cannot demonstrate specific causation without at least some evidence of exposure to chemicals at a level sufficient to cause the plaintiff’s alleged symptoms. Here, there simply was no such evidence presented. Indeed, Lowery’s expert did not even try to ascertain the level of exposure. Despite opining that “the fumes from the oil spill were the sole cause of Chance Lowery’s migraine, extreme coughing, nausea and vomiting,” Dr. Nosanchuk admittedly has no knowledge of Lowery’s *actual exposure* to VOCs, if any, let alone whether he was exposed to VOCs at a level considered to be capable of causing the sorts of symptoms Lowery alleges. That is because Dr. Nosanchuk did not review *any* of the available air monitoring results or sampling data gathered

¹⁷ See also *Wills*, 379 F3d at 49 (rejecting the plaintiff’s expert’s causation opinion and affirming summary judgment because there was no evidence that the decedent “had been exposed to a harmful amount of toxins”); *Nelson v Tennessee Gas Pipeline Co*, 243 F3d 244, 252-253 (CA 6, 2001) (holding that even levels of PCBs “in excess of allowable limits” could not establish causation absent evidence that the plaintiffs were “exposed at a level that could cause neurological and lung impairments,” and observing that the plaintiffs’ expert “admitted no knowledge concerning the actual exposure of the seven plaintiffs to PCBs”); *Heller v Shaw Indus, Inc*, 167 F3d 146 (CA 3, 1999) (affirming grant of summary judgment in part because there was no evidence that carpet installed in the plaintiff’s home emitted VOCs at a level sufficient to cause her alleged respiratory symptoms); *Conde v Velsicol Chem Corp*, 24 F3d 809 (CA 6, 1994) (upholding exclusion of expert testimony and grant of summary judgment to the defendant because there was no evidence that the plaintiffs had been exposed to chlordane found in termiticide used in their home at levels sufficient to cause their alleged headaches, nausea, diarrhea, and abdominal pain); *Norfolk S Ry Co v Rogers*, 270 Va 468, 486-487; 621 SE2d 59 (2005) (“Norfolk Southern cannot be held liable on a theory of exposure to excessive amounts of silica dust when there was no evidence of exposure to silica dust beyond exposure to a dust cloud of unknown content.”).

after the Line 6B incident, including samples taken from the vicinity of Lowery's home. (Nosanchuk Dep at 30-31, App 158a).¹⁸ While the materials from the Centers for Disease Control and Prevention (CDC) that Dr. Nosanchuk referenced in his deposition indicate that certain symptoms *may* occur after exposure *at certain levels* to the chemicals found in crude oil, that information is meaningless without evidence as to whether Lowery was actually exposed to those chemicals, and whether he was exposed at sufficient levels (and for sufficient time) to cause his alleged symptoms.

Moreover, this is not a case where there was no data available, or where it was impossible to at least estimate Lowery's potential VOC exposure. Courts emphasizing the need for evidence of a plaintiff's actual exposure to toxic chemicals have recognized that while proof of precise levels of exposure is not necessarily required, there must at least be some evidence from which exposure can reasonably be inferred. See, e.g., *Zellers v NexTech Northeast, LLC*, 533 Fed Appx 192, 198 (CA 4, 2013) ("While it is true, as Ms. Zellars argues, that precise information regarding a plaintiff's level of exposure 'is not always available, or necessary[,] it is also true that 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.'" (citation omitted); *Wright*, 91 F3d at 1107 ("We do not require a mathematically precise table equating levels of exposure with levels of harm, but there must be evidence from which a reasonable person could conclude

¹⁸ Not only did Lowery fail to request during discovery any of the extensive air monitoring or air sampling data, but, as discussed, most of the data was publically available on the EPA's website. See <<http://www.epa.gov/enbridgespill/data/index.html>> (accessed May 1, 2016).

that [exposure to a toxic chemical] has probably caused a particular plaintiff the kind of harm of which he or she complains before there can be a recovery.”¹⁹

For example, in *Curtis v M & S Petroleum, Inc*, 174 F3d 661 (CA 5, 1999), the plaintiffs’ industrial hygienist relied on several factors to conclude that the plaintiff refinery workers “were exposed to levels of benzene that were several hundred times above the permissible exposure level of 1 ppm.” *Id.* at 671. First, the plaintiffs experienced a cluster of “well-known symptoms of overexposure to benzene” soon after the refinery began processing a new product containing high levels of the chemical. *Id.* Second, several “Draeger tube tests” performed by refinery workers indicated that the workers “were exposed to at least 100 ppm.” *Id.* Finally, the expert relied on the refinery’s work practices and design, which made exposure to high benzene levels likely. *Id.* at 671-672. The Fifth Circuit found that such evidence “amply support[ed] [the expert’s] finding that the refinery workers were exposed to benzene at levels several hundred times higher than 1 ppm.” *Id.* at 672.

¹⁹ See also *Sean R*, 26 NY3d 801 at 808-809 (“Although it is ‘not always necessary for a plaintiff to quantify exposure levels precisely[,] we have never ‘dispensed with a plaintiff’s burden to establish sufficient exposure to a substance to cause the claimed adverse health effect[.]’ ‘At a minimum . . . there must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of th[e] agent that are known to cause the kind of harm that the plaintiff claims to have suffered.’”) (citations omitted); *Blanchard v Goodyear Tire & Rubber*, 190 Vt 577, 578-579; 30 A3d 1271 (2011) (“Of course, in many, if not most, toxic tort cases it is impossible ‘to quantify with hard proof—such as the presence of the alleged toxic substance in the plaintiff’s blood or tissue—the precise amount of the toxic substance to which an individual plaintiff was exposed.’ . . . But, while ‘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.’”) (citations omitted); *Abraham v Union Pac R Co*, 233 SW 3d 13, 22 (Tex App, 2007) (“Appellants need not produce a mathematically precise table equating levels of exposure with levels of harm to show that they were exposed to toxic levels of creosote, but they must produce evidence from which a reasonable person could conclude that their exposure probably caused their injuries.”).

Contrast the facts of *Curtis* with this case. Within hours of the discovery of the release, air monitoring and air sampling was commenced and proceeded under the direction of the EPA and other governmental agencies, and continued for months throughout Calhoun County. Literally thousands of data points were available that showed the levels of VOCs from the time of the release until months afterwards. Dr. Nosanchuk did not review *any* of this data to determine if there was exposure. Instead, when asked to supply the basis for his belief that Lowery had been exposed to VOCs in the air surrounding his home, Dr. Nosanchuk responded only that he understood that there were VOCs “in the water” and that Lowery smelled oil. (*Id.* at 30-31, App 158a). Yet Dr. Nosanchuk did not know (1) where the release site was, (2) the emission rates of the VOCs found in crude oil, (3) that Lowery lived more than ten miles downstream from the release site, or (4) what being able to “smell” oil says about a person’s exposure to VOCs, if anything. (*Id.* at 27-30, App 157-158a).

To simply ignore the scientific data available and instead assume that there must have been exposure is the worst form of “junk science,” and is precisely why reliable expert testimony is necessary to establish causation. In moving for summary disposition, Enbridge challenged the reliability of Dr. Nosanchuk’s opinion and Lowery’s ability to rely on it in opposing summary disposition. (See Enbridge’s Mot for Summ Disp at 11-14, App 23-26a; Reply Br in Supp of Enbridge’s Mot for Summ Disp at 6-7, App 186-187a). While neither the trial court nor the Court of Appeals majority specifically addressed this issue, it is apparent that Dr. Nosanchuk’s testimony regarding causation is insufficient to support Lowery’s claim.

MRE 702 requires trial courts 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.” *Gilbert v DaimlerChrysler Corp*, 470

Mich 749, 779; 685 NW2d 391 (2004). In cases involving “injury to a person or property,” MCL 600.2955(1) underscores this reliability requirement by providing that 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.” See also *Elher v Misra*, 499 Mich 11; ___ NW2d ___; 2016 WL 483425, *5 (2016) (explaining that MCL 600.2955(1) requires the trial court to “examin[e] the opinion and its basis, including the facts, technique, methodology, and reasoning relied on by the expert, and by considering several factors,” including “whether the basis for the opinion is reliable” and whether expert’s methodology “is relied upon by experts outside of the context of litigation”). “Careful vetting of all aspects of expert testimony is especially important when an expert provides testimony about causation.” *Gilbert*, 470 Mich at 782.

Although such assessments are usually made in the trial context, it is also appropriate to consider the reliability of an expert’s causation opinion at the summary disposition stage. See, e.g., *Skinner v Square D Co*, 445 Mich 153, 173-174; 516 NW2d 475 (1994) (rejecting expert conclusions as insufficient to establish causation for purposes of deciding the defendant’s motion for disposition because they were “premised on mere suppositions”); *Amorello v Monsanto Corp*, 186 Mich App 324, 331-332; 463 NW2d 487 (1990) (“Summary disposition is not precluded simply because a party has produced an expert to support its position. The expert’s opinion must be admissible [under MRE 702, which provides that] [t]he facts and data upon which the expert relies in formulating an opinion must be reliable.”) (some citations omitted).²⁰

²⁰ See also *Karbel v Comerica Bank*, 247 Mich App 90, 104; 635 NW2d 69 (2001) (rejecting expert opinion and affirming summary disposition in the defendant’s favor because the opinion was based on “impermissible conjecture, not reasonable inferences”), citing *Skinner*.

Dr. Nosanchuk's testimony does not come close to meeting the reliability requirements of MRE 702 and MCL 600.2955. As an initial matter, the Court of Appeals dissent correctly noted that Dr. Nosanchuk acknowledged that his practice "was limited to the treatment of routine medical conditions, [and] that he had no expertise regarding the medical effects of exposure to toxic chemicals and volatile organic compounds." (COA Dissent at 1, App 252a).²¹ Thus, Dr. Nosanchuk is simply not qualified to opine on the causation issue. See *Higgins*, 794 F3d at 705 ("[A]lthough a doctor may have 'experience diagnosing and treating asthma . . . that does not make him qualified to 'assess its genesis.' . . . [The plaintiff] put forth no evidence that [his expert] has ever treated another patient for chlorine gas exposure or has any training in toxicology."); *Plourde v Gladstone*, 190 F Supp 2d 708, 719-720 (D Vt, 2002), aff'd 69 Fed Appx 483 (CA 2, 2003) (finding that the plaintiff's toxicologist was not qualified to testify "that the herbicides sprayed by [the defendant] caused the injuries experienced by the [plaintiffs] and their livestock" because he professed "no experience or training in diagnosing and treating patients").²²

Even assuming he is qualified to opine on the causal connection between VOC exposure and the symptoms Lowery claims to have experienced, Dr. Nosanchuk freely acknowledged that he did not know *anything* about Lowery's potential exposure to VOCs, let alone whether he was exposed to levels sufficient to cause his alleged symptoms. (See Nosanchuk Dep at 27-31, App

²¹ During his deposition, Dr. Nosanchuk testified that he did not "really understand the toxicology" and "wouldn't have any idea" of the levels of VOC exposure necessary to cause the symptoms Lowery claimed to have experienced. (See Nosanchuk Dep at 26-27, 157a).

²² See also *Sutera v Perrier Group of Am*, 986 F Supp 655, 667 (D Mass, 1997) (finding that the plaintiff's oncology/hematology expert was not qualified to testify that exposure to benzene caused the plaintiff's leukemia, where the doctor had no special expertise in benzene exposure and, while qualified to diagnose leukemia, was not qualified to render an opinion as to its specific cause).

157-158a). Without that information, Dr. Nosanchuk's testimony regarding the cause of Lowery's alleged symptoms is speculative and unreliable, and thus insufficient to create a genuine issue of material fact for trial. Consistent with the federal case law previously discussed, that deficiency alone warrants summary disposition in Enbridge's favor. See, e.g., *Pluck*, 640 F3d at 679 (rejecting the plaintiffs' expert's causation opinion and affirming summary judgment because he "did not ascertain Mrs. Pluck's level of benzene exposure, nor did he determine whether she was exposed to quantities of benzene exceeding the EPA's safety regulations"); *Nelson*, 243 F3d at 252 (observing that the plaintiffs' expert "made no attempt to determine what amount of PCB exposure the . . . subjects had received and simply assumed that it was sufficient to make them ill," and affirming the lower court's exclusion of the expert's causation opinion for summary judgment purposes).

3. Mere correlation between alleged exposure and onset of symptoms is not enough.

In nevertheless finding there to be a genuine issue of material fact with respect to causation, the Court of Appeals majority reasoned that "there was 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." (COA Op at 3, App 251a). In support, the majority cited the fact that Lowery "lived in the vicinity of the oil spill," was "aware of an overpowering odor," and claimed to have experienced symptoms consistent with exposure to VOCs around the time of the incident. (*Id.*). But in reaching this conclusion, the Court of Appeals majority disregarded the undisputed evidence concerning the distance between Lowery's home and the release site, as well as the two-week time difference between Lowery's own report of smelling oil and the alleged vomiting that Lowery claims caused the rupture of his gastric artery. As opposed to evidence establishing a "logical sequence

of cause and effect,” the Court of Appeals majority instead relied, as did Dr. Nosanchuk, on nothing more than the fact that Lowery’s alleged symptoms coincided with the oil leak.

That is not enough. As federal courts have held, a plaintiff in a toxic tort case must do more than present evidence of the potential existence of a toxin in the environment, followed by the onset of alleged symptoms consistent with exposure. See, e.g., *Barrett v Rhodia, Inc*, 606 F3d 975, 984 (CA 8, 2010) (applying Nebraska law to hold that evidence of symptoms “consistent with” chemical exposure is insufficient to establish causation); *Nelson*, 243 F3d at 254 (rejecting the “circular reasoning that the plaintiffs must have been exposed to PCBs because PCBs were present in the environment and plaintiffs showed symptoms”); *Conde v Velsicol Chemical Corp*, 24 F3d 809 (CA 6, 1994) (holding that the plaintiffs’ experts’ testimony “that chlordane exposure ‘is consistent with’ [the plaintiffs’] observed symptoms” was “insufficient to permit a jury to conclude, by a preponderance of the evidence, that chlordane exposure caused the [plaintiffs’] health problems”).

This reasoning is in line with this Court’s own precedents. While the Court of Appeals majority correctly observed that “[a] plaintiff is permitted to prove his case through circumstantial evidence and reasonable inferences” (see COA Op at 3, App 251a), that is not what we have here. In *Skinner v Square D Co*, 445 Mich 153; 516 NW2d 475 (1994), this Court explained the role of circumstantial evidence in demonstrating causation and “the basic legal distinction between a reasonable inference and impermissible conjecture”:

As a theory of causation, a conjecture is simply an explanation consistent with known facts or conditions, but not deducible from them as a reasonable inference. There 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. On the other hand, if there is evidence which points to any 1 theory of causation, indicating a logical sequence of cause and effect, then there is a juridical basis for such a determination, notwithstanding the existence of other plausible theories with or without support in the evidence. [*Id.*]

at 164, quoting *Kaminski v Grand Trunk W R Co*, 347 Mich 417, 422; 79 NW2d 899 (1956).]

Wishing to “make clear what it means to provide circumstantial evidence that permits a reasonable inference of causation,” *Skinner* emphasized that “at a minimum, a causation theory must have some basis in established fact. However, a basis in only slight evidence is not enough. Nor is it sufficient to submit a causation theory that, while factually supported, is, at best, just as possible as another theory. Rather, the 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), citing *Kaminski*. While “the evidence need not negate all other possible causes,” it must “exclude other reasonable hypotheses with a fair amount of certainty.” *Craig v Oakwood Hosp*, 471 Mich 67, 87-88; 684 NW2d 296 (2004) (citation and internal quotation marks omitted). In sum, there must be “more than a mere possibility or a plausible explanation.” *Id.* at 87.

While not a toxic tort case, *Craig* provides a useful example of these principles. The plaintiff in *Craig* was born with cerebral palsy, which his expert opined was attributable to traumatic injury to the plaintiff’s brain during his mother’s labor and delivery. *Id.* at 91. As support, the expert relied on an MRI image showing the plaintiff’s “brain tissue had developed asymmetrically.” *Id.* at 92. The expert, however, never explained “how exactly the mechanisms he described led to cerebral palsy (as opposed to any other neurological impairment) and how they were connected to the asymmetric brain development depicted in [the] plaintiff’s MRI.” *Id.* This Court held that without evidence supplying this connection, the jury could only engage in improper speculation based on the mere *correlation* between the plaintiff’s alleged head injury and his cerebral palsy:

It 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. Given the absence of testimony on causation supplied by Dr. Gabriel, the jury could have found for plaintiff only if it indulged in this logical error-concluding, in effect, that evidence that plaintiff may have sustained a head injury, combined with evidence that plaintiff now has cerebral palsy, leads to the conclusion that the conduct that caused plaintiff's head injury also caused his cerebral palsy.

Such indulgence is prohibited by our jurisprudence on causation. . . . [*Id.* at 93.]

Consistent with *Craig*, it is not enough to simply show a *correlation* between alleged chemical exposure and symptoms; in other words, just because there was an event in proximity to the alleged injuries, causation has not been established. As *Craig* observed, “[i]t is axiomatic in logic and in science that correlation is not causation,” and thus “*it is error to infer that A causes B from the mere fact that A and B occur together.*” *Craig*, 471 Mich at 93 (emphasis added). Yet this is *precisely* what the Court of Appeals majority has permitted in this case. When asked to explain the basis for his causation opinion, Dr. Nosanchuk responded that it was based on the fact that Lowery “wasn’t having the problems before and he was having the problems afterwards and the oil spill”:

Q. . . . You indicate [in your written opinion] that Mr. Lowery’s migraine[,] extreme coughing and vomiting [--] or the oil spill was the sole cause of Mr. Lowery’s symptoms; is that correct?

A. That’s correct.

Q. What do you mean by sole cause?

A. It means 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 and 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. [Nosanchuk Dep at 48-49, App 93-94a (emphasis added).]

This is exactly the sort of *post hoc ergo propter hoc* reasoning that this Court rejected in *Craig*, and that federal courts have said is insufficient to establish causation in toxic tort cases.

See, e.g., *Higgins*, 794 F3d at 703-704 (rejecting notion that onset of respiratory symptoms after exposure to chlorine established causation because it would invoke “the fallacy of saying that because effect A happened at some point after alleged cause B, the alleged cause was the actual cause”) (citation and internal quotation marks omitted); *McLain*, 401 F3d at 1243 (“[P]roving a temporal relationship between [exposure to a substance] and the onset of symptoms does not establish a causal relationship. . . . Drawing such a conclusion from temporal relationships leads to the blunder of the *post hoc ergo propter hoc* fallacy.”); *Young v Burton*, 567 F Supp 2d 121, 140 (D DC, 2008) (rejecting expert testimony in toxic mold exposure case: “Drawing conclusions about causation from temporality is a common logical fallacy known as *post hoc ergo propter hoc* (after the fact, therefore because of the fact), and is as unpersuasive in the courts as it is in the scientific community.”).

Although Lowery claims to have experienced nausea, coughing, and vomiting following the Line 6B incident, there is no evidence that it was *caused* by exposure VOCs – and certainly not *three weeks after the oil leak and more than a week after Lowery said the smell of oil went away*, which is when Lowery claims that vomiting led to the rupture of his gastric artery. In order to demonstrate a causal connection, Lowery was required to present at least *some* evidence that he was actually exposed to benzene or other VOCs at the time of his reported symptoms, and at a level sufficient to cause his alleged symptoms. See, e.g., *Wright*, 91 F3d at 1107 (“At a minimum, we think that there 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.”).

Here, there is no such evidence, as Dr. Nosanchuk admitted that he did not review any of the available air monitoring results or sampling data gathered after the Line 6B incident. As the

Court of Appeals dissent properly recognized, Lowery's "evidence" thus demonstrates nothing more than a "mere possibility of causation," which is not enough to survive a motion for summary disposition. (See COA Dissent at 2, App 253a, citing *Badalamenti v William Beaumont Hospital-Troy*, 237 Mich App 278, 285-286; 602 NW2d 854 (1999)).

4. Lowery's expert claimed to perform a "differential diagnosis," but never examined Lowery and failed to account for alternative causes for his alleged symptoms.

Further exposing the speculative and unreliable nature of Dr. Nosanchuk's causation testimony is his failure to account for Lowery's use of Lamictal and Vicodin as potential alternative causes of the alleged headaches and vomiting that he claims led to the rupture of his gastric artery. As previously discussed, Lowery was not an otherwise healthy individual who suddenly developed his alleged symptoms for the first time after the oil leak. Instead, Lowery's medical records reflect a history of migraine headaches and nausea that he has long attributed to the Lamictal he was taking for his depression. Lowery even complained of a migraine the day *after* his surgery, and once again blamed it on the Lamictal, leading his surgeon to seek a psychiatric consult. And Lowery was resistant to taking Vicodin to treat his migraine because he thought it was the Vicodin that caused him to start vomiting the day his gastric artery ruptured.

Although the Court of Appeals majority acknowledged those other "plausible explanations" for Lowery's alleged symptoms, it dismissed them as merely serving "to highlight that there are genuine issues of material fact to be resolved by a jury." (COA Op at 3, App 251a). But such reasoning defies this Court's precedents. As *Skinner* explained, "[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.*" *Skinner*, 445 Mich at 164 (citation omitted; emphasis added). This means that "if [the] evidence lends equal support to inconsistent conclusions or is equally consistent with contradictory

hypotheses, negligence is not established.” *Id.* at 166-67 (citation omitted). In other words, courts “cannot permit the jury to guess.” *Id.* at 166 (citation omitted; emphasis added).

As the Sixth Circuit explained in *Pluck*, experts commonly use “differential diagnosis” to assist in determining the likely cause for an illness. In a toxic tort case, the two critical steps in any differential diagnosis are (1) the “ruling in” of chemical exposure, which requires evidence of the dose of chemicals to which the plaintiff was exposed, and (2) the “ruling out” of alternative causes “based on a physical examination, clinical tests, and a thorough case history.” *Pluck*, 640 F3d at 678 (citation omitted).²³ In performing a proper differential diagnosis, the physician “should seek more than a patient’s self-report of symptoms or illness and . . . should . . . determine that a patient is ill and what illness the patient has contracted.” *Best v Lowe’s Home Centers, Inc.*, 563 F3d 171, 179 (CA 6, 2009) (citation omitted).

In *Pluck*, for example, the Sixth Circuit held that the plaintiff’s expert did not perform a proper differential diagnosis in reaching his opinion that benzene exposure caused the plaintiff’s non-Hodgkin’s lymphoma (“NHL”) when an underground pipeline leaked gasoline into the surrounding groundwater. The plaintiffs’ expert simply concluded that “chronic low-level exposure can and does cause NHL” and that “[t]here is no safe level for benzene in terms of causing cancer.” *Id.* The Sixth Circuit rejected that analysis, finding the plaintiff’s mere exposure to be insufficient and observing that “it is well-settled that the mere existence of a toxin in the environment is insufficient to establish causation without proof that the level of exposure could cause the plaintiff’s symptoms.” *Id.* The expert also failed to “rule out” alternative causes,

²³ See also *Dengler v State Farm Mut Ins Co*, 135 Mich App 645, 649; 354 NW2d 294 (1984) (“[D]ifferential diagnosis . . . is simply a method by which all 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.”).

such as the fact that the plaintiff had an “extensive smoking habit,” and had potentially been exposed to unidentified “solvents.” *Id.* at 680.

This is in contrast to the expert’s causation analysis in *Best*, 563 F3d 171, in which the Sixth Circuit held that the plaintiff’s treating physician properly used a differential diagnosis in reaching his opinion that the plaintiff’s loss of smell (anosmia) resulted from his head and face being accidentally doused with pool cleaning chemicals while shopping at a Lowe’s Home Center store. First, the expert objectively obtained the nature of the plaintiff’s injury by personally administering a well-recognized test “to confirm Best’s complaint that he could not smell.” *Id.* at 180. Second, the expert “compiled a list of possible causes for the injury, including virus, accident, brain tumor, brain surgery, exposure to chemicals, medications, or an id[i]opathic (unknown) cause.” *Id.* at 180-181. Although there was no published material confirming that inhalation of the chemical could cause anosmia, the court found that the expert properly relied on the product’s Material Safety Data Sheet (MSDS) and “his own knowledge of medicine and chemistry that the chemical can cause damage to the nasal and sinus mucosa upon inhalation,” and observed that the expert had “treated other patients who developed anosmic symptoms after inhaling chlorine derivatives.” *Id.* at 181.

Finally, the expert ruled out other causes, focusing on “chemicals, medications, or id[i]opathic causes” since there was “no evidence that virus, accident, brain tumor, or brain surgery were applicable.” *Id.* The expert concluded that an idiopathic cause would likely “not appear over such a short duration of time,” and “eliminated nine of ten of [the plaintiff’s] medications.” *Id.* Although there was one medication that the expert did not specifically rule out, the court noted that there was no evidence that it might cause anosmia. *Id.* Because the expert employed “the same level of intellectual rigor that characterizes the practice of an expert

in the relevant field,” the court held that the expert’s differential diagnosis was admissible. *Id.* at 181-182, quoting *Kumho Tire*, 526 US at 152.

While Dr. Nosanchuk claimed to have performed a differential diagnosis, his testimony suffers from the same flaws as in *Pluck*, and is nothing like the expert’s careful analysis in *Best*. First, Dr. Nosanchuk failed to “rule in” VOC exposure as the cause of Lowery’s alleged symptoms because – as discussed – he did not have any evidence of actual exposure. Second, he failed to “rule out” alternative causes. As an initial matter, Dr. Nosanchuk did not examine Lowery or conduct any tests, a fact that the Court of Appeals majority acknowledged. (COA Op at 2, App 250a). In fact, Dr. Nosanchuk never even spoke with him. (Nosanchuk Dep at 45, App 91a). Despite that fact, Dr. Nosanchuk dismissed, without any explanation, the possibility that Lowery’s alleged symptoms could have been explained by his past medical history. When asked about his differential diagnosis, Dr. Nosanchuk initially suggested that he *did not even consider* other potential causes:

Q. So did you rule out other potential causes when you made that determination?

A. I think that other potential causes were very unlikely.

Q. And what is that based on?

A. My clinical judgment.

Q. *Did you consider other potential causes?*

A. *I was not given any other potential causes to consider.*

Q. *So the answer is no?*

A. *No. Well, I take that back. I mean, as a physician in my own practice, I have to be very careful because – I try to be very careful. There’s a lot of always possible factors in everything, but you always have to consider the most likely cause and the most relevant exciting factor and the most – in my view, that is what it was. When I think about things with patients, I think about a lot of things. Do I remember thinking about anything*

specifically, no. This was my clinical judgment. [Nosanchuk Dep at 49, App 94a (emphasis added).]

While Dr. Nosanchuk eventually got around to saying he “considered” and “thought about” ruling out other possible causes (*id.* at 79, App 103a), it most certainly was not based on “a physical examination, clinical tests, and a thorough case history.” *Pluck*, 640 F3d at 678 (citation omitted). Nor did he provide “a reasonable explanation as to why ‘he . . . concluded that [any alternative cause suggested by the defense] was not the sole cause.’” *Best*, 563 F3d at 179. For instance, in the face of medical reports that Lowery had previously experienced headaches and nausea from his use of the antidepressant drug Lamictal, especially when he smoked or was around smoke,²⁴ Dr. Nosanchuk summarily excluded Lowery’s use of Lamictal from his differential diagnosis without any knowledge of the dose Lowery was taking, whether he was increasing or decreasing his dosage at the time, or whether he was consistently taking Lamictal as it was prescribed. (See Nosanchuk Dep at 53-55, App 159a).²⁵ Moreover, Dr. Nosanchuk did not know the frequency of Lowery’s marijuana use, and did not even know that he was a cigarette smoker. (*Id.* at 80, App 103-104a). Yet, Dr. Nosanchuk rejected the possibility of an interaction between Lowery’s medication and smoking by simply defaulting to the generic explanation that he was relying on his “clinical judgment.” (*Id.* at 86, App 105-106a).

²⁴ The Court can take judicial notice that the U.S. Food and Drug Administration-approved “medication guide” for Lamictal lists both “nausea” and “vomiting” as “common side effects.” See <<http://www.fda.gov/downloads/Drugs/DrugSafety/UCM152835.pdf>> (accessed May 1, 2016); *Chapman v Abbott Labs*, 930 F Supp 2d 1321, 1323 (MD Fla, 2013) (taking judicial notice of FDA-approved label).

²⁵ Dr. Nosanchuk acknowledged that Lowery’s prior reported headaches and nausea while taking Lamictal “may have been something when he was increasing his dose before he was acclimated to the drug.” (Nosanchuk Dep at 54, App 159a). But, he did not explain why he did not consider that factor in his differential diagnosis when he admitted that he did not know Lowery’s dose of Lamictal or whether he was taking it consistently as prescribed.

Dr. Nosanchuk also dismissed Vicodin as a potential cause of Lowery's vomiting without any analysis, based only on his predetermined conclusion that exposure to VOCs was the cause:

Q. When you were evaluating Mr. Lowery's symptoms of headache, nausea, coughing and vomiting, did you consider at all that he may have been having side effects from taking Vicodin?

A. I did not consider that to be a reasonable conclusion.

Q. Why is that?

A. For the reasons I outlined earlier. I think he was exposed to a toxin. I think that is what caused his symptoms and caused him to vomit violently.
...

* * *

Q. So it's your opinion that Mr. Lowery's use of Vicodin in this instance did not cause any of his symptoms?

A. I don't believe so.

Q. And that's just – and it's just because you have ruled it out based on your clinical knowledge?

A. That's my medical judgment. [*Id.* at 85-87, App 201a.]

As this exchange demonstrates, Dr. Nosanchuk did not properly rule out Vicodin as a potential cause. Rather, because he had already assumed that exposure to the fumes from the oil were the cause of Lowery's symptoms, Dr. Nosanchuk likewise assumed that nothing else could have been the cause.

As mentioned, Lowery told his surgeon that he believed Vicodin caused his vomiting, and Dr. Nosanchuk confirmed that "Vicodin can certainly cause nausea in susceptible people" (*Id.* at 82, App 200a). Dr. Nosanchuk also pointed out that "[g]enerally the patients I have that take Vicodin and vomit don't take it again" (*Id.*), yet based on his "medical judgment" he dismissed the fact that Lowery refused Vicodin at the hospital following his surgery for fear he would vomit again. (*Id.* at 86-87, App 201a).

Dr. Nosanchuk similarly failed to provide any explanation or support for disregarding Lowery's history of migraines. Dr. Nosanchuk asserted that it did not change his opinion because Lowery's prior headaches were less severe. But when asked about the basis for that conclusion, Dr. Nosanchuk responded only that the "basis is the patient's story, as far as I know." (*Id.* at 76, App 197-198a). When further pressed to point out exactly what evidence he relied upon, Dr. Nosanchuk could only say that he was "sure it was a communication of some kind, but I don't recall exactly." (*Id.* at 77, App 198a). This was despite the fact that Dr. Nosanchuk admittedly never even spoke to Lowery. (*Id.*). Far from "highlight[ing] that there are genuine issues of material fact to be resolved by the jury," Dr. Nosanchuk's failure to account for alternative causes of Lowery's alleged symptoms further demonstrates that his causation theory is nothing more than pure speculation. See also *Higgins*, 794 F3d at 705 (rejecting expert's opinion because she "essentially diagnosed [the plaintiff] after listening to his own description of his symptoms and the events at [the waterpark]—some fourteen months after the fact—and after looking at the results (though not the underlying data) of the pulmonary function study conducted by another doctor the year before. But the record is silent on whether [she] considered other possible causes of [the plaintiff's] ailments and, if so, how and why she ruled them out.").

5. Even if exposure to oil fumes caused Lowery's alleged nausea and vomiting, a jury could only speculate as to what caused Lowery's gastric artery to rupture.

Finally, even if a jury could reasonably find that Lowery's alleged nausea, vomiting, and coughing were caused by exposure to oil fumes, Lowery still failed to present evidence that this is what caused the avulsion of his short gastric artery. As mentioned, the Court of Appeals majority asserted that expert testimony is not needed because there was a "strong enough logical sequence of cause and effect." (COA Op at 3, App 251a). But as the Court of Appeals dissent

explained, “whether plaintiff’s vomiting . . . caused his abdominal artery to rupture [is not a matter] within the common understanding of average jurors.” (COA Dissent at 1, App 252a). Indeed, *not even Lowery’s own surgeon could say that there was a causal connection.* (Koziarski Dep at 36-37, App 70-71a) (“Q. And in this instance, you testified earlier that you could not determine the actual medical cause of Mr. Lowery’s torn artery or avulsed artery; is that correct? A. That is correct.”)). To suggest, as the Court of Appeals majority did, that a lay jury could reasonably infer causation without the assistance of an expert defies both common sense and this Court’s decision in *Craig*, which made it abundantly clear that a jury cannot be permitted to “indulg[e]” in the “logical error” that “A causes B from the mere fact that A and B occur together.” *Craig*, 471 Mich at 93.

Lowery was instead required to present testimony from a qualified expert in order to create a genuine issue of material fact as to causation, and failed to do so. As an initial matter, the Court of Appeals dissent was right when it found Dr. Nosanchuk to be unqualified to opine on the purported causal connection between Lowery’s alleged coughing and vomiting and the rupture of his artery:

Plaintiff’s proffered expert, Jerry Nosanchuk, D.O., was a family-medicine doctor without experience or training in toxicology or vascular surgery. Nosanchuk testified at his deposition that his practice was limited to the treatment of routine medical conditions, that he had no expertise regarding the medical effects of exposure to toxic chemicals and volatile organic compounds, and that he had never treated a patient with a ruptured abdominal artery resulting in internal bleeding. I simply cannot conclude that Nosanchuk was qualified to opine on the causation of plaintiff’s injury or that his testimony would have assisted the trier of fact in any way. [COA Dissent at 1-2, App 252-253a.]

Indeed, Dr. Nosanchuk even admitted that he had to “look this up, I’m not an anatomist.” (See Nosanchuk Dep at 68, App 98a). As this Court held in *Gilbert*, 470 Mich 749, courts must be vigilant in enforcing the limits of an expert’s purported expertise. *Id.* at 787-788 (finding an

expert witness to be unqualified to provide medical causation testimony that was beyond the scope of the witness's expertise as a social worker).

But even if Dr. Nosanchuk were qualified to opine as to what caused Lowery's gastric artery to avulse, his causation theory is speculative and lacking support in any medical literature. Dr. Nosanchuk acknowledged that gastric artery rupture is a "relatively rare condition" (Nosanchuk Dep at 70, App 99a), and the article abstracts he cited involving artery rupture following vomiting merely confirmed its rarity. (See Exhibits A and B). Neither article purports to establish a causal connection. Instead, they merely address the need for emergency room doctors to recognize the possibility of abdominal bleeding – *regardless of the cause* – when presented with a patient experiencing severe abdominal pain.

As this Court has instructed time and again, it is not enough for an expert simply to cite a study and assert that it supports the expert's opinion. For example, in *Craig*, 471 Mich at 80-83, the Court observed that although the plaintiff had "produced several articles and authorities" in response to the defendants' *Daubert*²⁶ challenge, the plaintiff failed to provide a "single authority that truly supported" the plaintiff's expert's causation theory:

. . . Dr. Gabriel's etiological theory, as summarized by defendant in arguing its motion [in limine], was that "hyperstimulation" of the uterus caused the head of the fetus (plaintiff) to pound against his mother's pelvic anatomy, thereby producing permanent brain damage. . . .

In response to this motion, plaintiff's attorney produced several articles and authorities that were meant to demonstrate a link between the use of Pitocin and the type of injury sustained by plaintiff. But while some of these articles described a correlation between the use of Pitocin and generalized brain injury, none of these authorities supported the theory of causation actually put forth by Dr. Gabriel. . . .

* * *

Plaintiff failed to introduce a single authority that truly supported Dr. Gabriel's theory in response to defendant's motion. Instead, plaintiff repeatedly

²⁶ *Daubert v Merrell Dow Pharm, Inc*, 509 US 579; 113 S Ct 2786; 125 L Ed 2d 469 (1993).

stressed that medical literature amply supported the proposition that Pitocin could cause brain damage--a proposition defendant did not contest--and supplied the court with literature to that effect. But this literature had little to do with Dr. Gabriel's causal theory

Citing *Craig*, the Court in *Edry v Adelman*, 486 Mich 634; 786 NW2d 567 (2010), explained that although "a lack of supporting literature" is "not dispositive," it is an "important factor" to determining the reliability of an expert's testimony. *Id.* at 640. In *Edry*, the plaintiff's expert opined that a delay in diagnosing the plaintiff's breast cancer reduced her chances of surviving five years from 95 percent to 20 percent. *Id.* at 637. However, the expert's opinion was contradicted by the opinions of other experts in the case, as well as "published literature on the subject." *Id.* at 640. This Court found the expert's opinion to be "unreliable and inadmissible" in the absence of either supporting literature or "some other form of support":

[N]o literature was admitted into evidence that supported Dr. Singer's testimony. Although he made general references to textbooks and journals during his deposition, plaintiff failed to produce that literature, even after the court provided plaintiff a sufficient opportunity to do so. Plaintiff eventually provided some literature in support of Dr. Singer's opinion in her motion to set aside the trial court's order, but the material consisted only of printouts from publicly accessible websites that provided general statistics about survival rates of breast cancer patients. The fact that material is publicly available on the Internet is not, alone, an indication that it is unreliable, but these materials were not peer-reviewed and did not directly support Dr. Singer's testimony. Moreover, plaintiff never provided an affidavit explaining how Dr. Singer used the information from the websites to formulate his opinion or whether Dr. Singer ever even reviewed the articles.

Plaintiff failed to provide any support for Dr. Singer's opinion that would demonstrate that it has some basis in fact, that it is the result of reliable principles or methods, or that Dr. Singer applied his methods to the facts of the case in a reliable manner, as required by MRE 702. While peer-reviewed, published literature is not always a necessary or sufficient method of meeting the requirements of MRE 702, in this case the lack of supporting literature, combined with the lack of any other form of support for Dr. Singer's opinion, renders his opinion unreliable and inadmissible under MRE 702. Under MRE 702, it is generally not sufficient to simply point to an expert's experience and background to argue that the expert's opinion is reliable and, therefore, admissible. Plaintiff has failed to satisfy her burden regarding the admissibility of Dr. Singer's

opinion; therefore, the trial court did not abuse its discretion by excluding Dr. Singer's testimony as unreliable under MRE 702. [*Id.* at 640-642.]²⁷

While *Craig* and *Edry* were medical malpractice cases, the Court of Appeals has recognized that it is just as important to require supporting literature for an expert's opinion that exposure to a chemical caused a plaintiff's injury. In *Amorello*, 186 Mich App 324, the plaintiffs alleged that they suffered "a variety of medical problems as a result of exposure to polychlorinated biphenyls (PCBs) which allegedly leaked from a Detroit Edison electrical transformer located in their backyard." *Id.* at 326. The manufacturer of the transformer, Monsanto Corporation, moved for summary disposition arguing that "even if plaintiffs had been exposed to PCBs, plaintiffs could not establish a causal relationship between the exposure and their health problems." *Id.* at 328.

In affirming the trial court's decision to grant Monsanto's motion, the Court of Appeals observed that although the plaintiffs had supplied expert testimony in support of their claim, including testimony from two physicians and a toxicologist, it was not sufficient to overcome summary disposition because the experts' opinions had no support in "scientific and medical literature" and otherwise lacked a "reasonable medical or reliable scientific basis":

Plaintiffs also argue that testimony establishes that plaintiffs' health problems were caused by PCB exposure. Plaintiffs rely on the testimony of the examining physicians, Drs. Feldman and Grundland, and of Norman Zimmerman, a toxicologist.

Summary disposition is not precluded simply because a party has produced an expert to support its position. The expert's opinion must be admissible. MRE 403; MRE 702. . . . The facts and data upon which the expert relies in formulating an opinion must be reliable. . . . Plaintiffs offer no evidence

²⁷ See also *Elher*, 499 Mich 11; __ NW2d __; 2016 WL 483425, *7 ("While peer-reviewed, published literature is not always necessary or sufficient to meet the requirements of MRE 702, the lack of supporting literature, combined with the lack of any other form of support, rendered [the plaintiff's expert's] opinion unreliable and inadmissible under MRE 702."); *Tondreau v Hans*, 495 Mich 860; 836 NW2d 691 (2013) (relying on *Edry* to reject unsupported causation opinions).

to rebut defendants' claim that the testimony of plaintiffs' experts did not have a reasonable medical or reliable scientific basis and is not supported by scientific and medical literature. Hence, plaintiffs did not show that the opinion testimony was admissible. Summary disposition was appropriate. [*Id.* at 331-332.]

Federal courts have likewise excluded expert testimony in toxic tort cases when the studies cited by the expert do not support the expert's opinion. See, e.g., *Baker v Chevron USA, Inc*, 680 F Supp 2d 865, 877 (SD Ohio, 2010), *aff'd* 533 Fed Appx 509 (CA 6, 2013) (rejecting expert's opinion that the plaintiffs' injuries resulted from "cumulative exposure to benzene" because none of the studies the expert cited "support[ed] an opinion that benzene can cause the illnesses from which [the] [plaintiffs] suffer[ed] at the extremely low doses or exposures [the plaintiffs] experienced"); *LeBlanc v Chevron USA*, 396 Fed Appx 94, 98-100 (CA 5, 2010) (finding that the studies cited by the plaintiffs' expert as support for his opinion that exposure to benzene at an oil refinery caused their family member's rare bone marrow disease did not provide a sufficiently reliable basis for the expert's conclusion, in part because they did not directly assess the relationship between benzene exposure and the disease at issue, and actually "disclaim[ed] the causal connection" being drawn by the expert).

Here, Dr. Nosanchuk provided no support whatsoever for his opinion that exposure to oil fumes three weeks after the Line 6B incident, and more than ten miles away from the release site, suddenly caused Lowery to vomit so severely that it resulted in the rupture of his gastric artery. While Dr. Nosanchuk claimed to have "looked at some articles on short gastric artery rupture," he only reviewed the abstracts, and he had no idea whether they were peer-reviewed. (Nosanchuk Dep at 21, App 191a).²⁸ When he was asked why he selected those particular

²⁸ Dr. Nosanchuk explained that obtaining the full articles "would have cost me money, so I didn't." (Nosanchuk Dep at 22, App 191a).

abstracts, Dr. Nosanchuk responded that it was because they were in “understandable English” and “were more believable to me.” (*Id.*).

Even taking the abstracts at face value, they do not support Dr. Nosanchuk’s opinion that Lowery’s “vomiting and retching . . . caused the tear.” (*Id.* at 69, App 99a). The first abstract reported a “very unusual case” of a patient that suffered a spontaneous tear of his short gastric artery after forceful gagging during teeth brushing, and noted “[s]everal factors such as pregnancy, hypertension and atherosclerosis have been described in association with abdominal apoplexy. Blunt trauma, inflammatory conditions, aneurysm rupture[,] and *rarely vomiting* are some predisposing conditions.” (See Exhibit A) (emphasis added).

The second abstract is even more attenuated. (See Exhibit B). It merely reported a patient who presented with “watery diarrhea and abdominal fullness followed by vomiting after the ingestion of alcohol but was later diagnosed with [abdominal bleeding].” (*Id.*). The abstract states that “abdominal apoplexy [hemorrhaging] *should be considered in the differential diagnosis* of unexplained hemorrhagic shock with an abrupt onset of severe abdominal pain associated with vomiting.” (*Id.*) (emphasis added). Neither abstract suggests, let alone establishes, a specific causal connection between vomiting and a tear in the short gastric artery.

A review of Dr. Nosanchuk’s deposition testimony reveals that he simply *assumed* causation based solely on Lowery’s claim that he was vomiting at the time he experienced severe abdominal pain. But once again, *correlation* is not sufficient to establish *causation*. See *Craig*, 471 Mich at 93. Nor is it enough for Dr. Nosanchuk to rely on his “clinical judgment.” Without “supporting literature” or “any other form of support,” Dr. Nosanchuk’s causation opinion is speculative and unreliable. And as the Court of Appeals dissent properly recognized, “[w]ithout sufficient expert testimony on the issue of causation, [Lowery] could not establish a genuine

issue of material fact concerning whether the Kalamazoo River Oil spill proximately caused his ruptured artery and internal bleeding.” (COA Dissent at 2, App 253a). Because the Court of Appeals majority’s contrary decision permits Lowery’s case to proceed to trial on the basis of conjecture instead of reasonable inferences of causation, it should be reversed.

V. RELIEF REQUESTED

For the reasons discussed, the Court should hold that Lowery did not sufficiently establish causation to avoid summary disposition under MCR 2.116(C)(10), and that he was instead required to present expert testimony regarding general and specific causation. The Court should reverse the Court of Appeals’ contrary decision and reinstate the trial court’s order granting Enbridge’s motion for summary disposition.

Respectfully submitted,

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/s/ Phillip J. DeRosier

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Dated: May 25, 2016

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DETROIT 40856-38 1385165v10

In the Supreme Court
Appeal from the Court of Appeals
Jansen, P.J., and Meter and Beckering, JJ.

CHANCE LOWERY,

Plaintiff-Appellee,

Docket No. 151600

v.

ENBRIDGE ENERGY, LIMITED
PARTNERSHIP and ENBRIDGE ENERGY
PARTNERS, L.P.,

Defendants-Appellants.

EXHIBITS TO DEFENDANTS-APPELLANTS' BRIEF ON APPEAL

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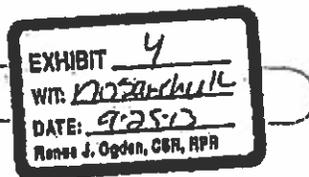
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- Exhibit A Abstract, “Short gastric artery apoplexy after gagging”
- Exhibit B Abstract, “Spontaneous rupture of the short gastric artery after vomiting”

Exhibit A

Search term



Short gastric artery apoplexy after gagging.

Jabr FI, et al. [Show all](#)

J Med Liban. 2012 Jul-Sep;60(3):173-5.

Department of Internal Medicine, Horizon Medical Center, Dickson TN 37055, USA. fijabr@gmail.com

Abstract

Abdominal apoplexy, the spontaneous hemorrhage into the peritoneal cavity, is usually caused by a rupture of visceral vessels such as short gastric arteries. Several factors such as pregnancy, hypertension and atherosclerosis have been described in association with abdominal apoplexy. Blunt trauma, inflammatory conditions, aneurysm rupture and rarely vomiting are some predisposing conditions. We report a very unusual case of a patient who had a spontaneous short gastric artery acute hemoperitoneum caused by forceful gagging during teeth brushing. The patient was treated with blood transfusion, fluids resuscitation, laparotomy, and suture ligation of the bleeder vessel. We also review the medical literature of abdominal apoplexy, a rare etiology of acute abdomen that should be recognized early in the Emergency Department.

PMID 23198460 [PubMed - indexed for MEDLINE]

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Gastric artery apoplexy presenting as an acute abdomen in an adolescent.

Spontaneous rupture of the short gastric artery after vomiting.

Abdominal apoplexy: a case study of the spontaneous rupture of the gastroepiploic artery.

Massive and acute hemoperitoneum due to rupture of the uterine artery by erosion from an endometriotic lesion.

Spontaneous colonic mesenteric hemorrhage: report of an unusual case of abdominal apoplexy.

PubMed

EXHIBIT 5
WIT: *D. S. Skeik*
DATE: 9-25-12
Renee J. Ogden, CSR, RPR

Display Settings: Abstract

Performing your original search, ***short gastric artery***, in PubMed will retrieve **283** records.

J Med Liban. 2012 Jul-Sep;60(3):173-5.

Short gastric artery apoplexy after gagging.

Jabr FI, Skeik N.

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Abstract

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PMID: 23198460 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms

LinkOut - more resources

Exhibit B

PubMed

Display Settings: Abstract

ELSEVIER
Full Text Article

Am J Emerg Med. 2012 Mar;30(3):513.e1-3. doi: 10.1016/j.ajem.2011.01.001. Epub 2011 Feb 26.

Spontaneous rupture of the short gastric artery after vomiting.

Ho MP, Chang CJ, Huang CY, Yu CJ, Tsai KC, Chen HA, Chaung WK.

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Abstract

Spontaneous rupture of the short gastric artery is an extremely rare event that can cause abdominal apoplexy or spontaneous hemoperitoneum. For the emergency physician, simultaneous restoration of circulatory volume and a rapid diagnosis remain central to a successful outcome in such critical cases. We reported a 21-year-old man who initially presented with watery diarrhea and abdominal fullness followed by vomiting after the ingestion of alcohol but was later diagnosed with hemoperitoneum, resulting in hemorrhagic shock due to spontaneous rupture of the small branches of the short gastric artery. The patient underwent emergency exploratory laparotomy with a good outcome. Abdominal apoplexy should be considered in the differential diagnosis of unexplained hemorrhagic shock with an abrupt onset of severe abdominal pain associated with vomiting.

PMID: 21354760 [PubMed - Indexed for MEDLINE]

Publication Types, MeSH Terms

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