

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.

**REPLY BRIEF OF DEFENDANTS-APPELLANTS ENBRIDGE ENERGY,
LIMITED PARTNERSHIP AND ENBRIDGE ENERGY PARTNERS, L.P.**

DICKINSON WRIGHT PLLC
Kathleen A. Lang (P34695)
Michael G. Vartanian (P23024)
Phillip J. DeRosier (P55595)
Kelley M. Haladyna (P63337)
500 Woodward Avenue, Suite 4000
Detroit, MI 48226
(313) 223-3500

Attorneys for Defendants-Appellants

TABLE OF CONTENTS

INDEX OF AUTHORITIES..... ii

I. INTRODUCTION1

II. ARGUMENT2

 A. Lowery misstates the appropriate standard for reviewing Enbridge’s motion for summary disposition.2

 B. The need for expert testimony in order to establish causation in toxic tort cases is well recognized.3

 C. A jury can only speculate as to whether Lowery’s alleged injuries were caused by exposure to oil vapors.5

III. RELIEF REQUESTED.....10

INDEX OF AUTHORITIES

Cases

Brown v Burlington Northern Santa Fe Railway Co, 765 F3d 765 (CA 7, 2014)..... 5

Bryant v Oakpointe Villa Nursing Centre, 471 Mich 411; 684 NW2d 864 (2004)..... 3

Burton v CSX Transp, Inc, 269 SW3d 1 (Ky, 2008)..... 8

Craig v Oakwood Hosp, 471 Mich 67; 684 NW2d 296 (2004)..... 3

Elher v Misra, 499 Mich 1; 878 NW2d 790 (2016) 7

Gass v Marriott Hotel Services, Inc, 558 F3d 419 (CA 6, 2009) 1

Genna v Jackson, 286 Mich App 413; 781 NW2d 124 (2009) 1, 4

Higdon v Kelly, 371 Mich 238; 123 NW2d 780 (1963) 4

Lindley v City of Detroit, 131 Mich 8; 90 NW 665 (1902) 3

Pluck v BP Oil Pipeline Co, 640 F3d 671 (CA 6, 2011) 3, 5, 6, 9

Savage v Peterson Distributing Co, Inc, 379 Mich 197; 150 NW2d 804 (1967)..... 8

Skinner v Square D Co, 445 Mich 153; 516 NW2d 475 (1994)..... 3

Smith v Globe Life Ins Co, 460 Mich 446; 597 NW2d 28 (1999) 2

Waskowski v State Farm Mut Auto Ins Co, 970 F Supp 2d 714 (ED Mich, 2013)..... 7

Whaley v CSX Transp, Inc, 362 SC 456; 609 SE2d 286 (2005)..... 9

Woodard v Custer, 473 Mich 1; 702 NW2d 525 (2005) 3

Wright v Willamette Industries, Inc, 91 F3d 1105 (CA 8, 1996)..... 6

Rules

MCR 2.116(C)(10)..... 2

I. INTRODUCTION

Lowery struggles mightily to shoehorn this case into the Court of Appeals' decision in *Genna v Jackson*, 286 Mich App 413; 781 NW2d 124 (2009), but even assuming *Genna* was correctly decided, it simply does not fit. Lowery did not live in a house with acknowledged levels of toxic mold. Nor did he walk into a room filled with a cloud of pesticides, as in *Gass v Marriott Hotel Services, Inc*, 558 F3d 419 (CA 6, 2009). Lowery lived more than ten miles downstream from a release of crude oil into the Kalamazoo River, and claims to have experienced headaches, nausea, and vomiting some *three weeks later*, and more than a week after he says the smell of oil went away. Not only that, Lowery claims that his sudden bout of vomiting was so severe that it led to the rupture of an artery in his abdomen, an exceedingly rare injury by any standard. And Lowery seeks to advance all of these claims without reliable expert testimony.

But as the Court of Appeals dissent properly recognized, “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). Indeed, even aside from the lack of proximity in time or distance between the oil release and Lowery’s claimed exposure, the need for expert testimony is apparent given the potential alternative causes of Lowery’s symptoms, including his antidepressant medication, Lamictal, and his consumption of Vicodin around the time of the vomiting that he claims led to the rupture of his gastric artery. While Lowery insists that these are simply questions of fact to be resolved by a jury, they instead demonstrate precisely why qualified expert testimony is so critical in cases like this.

Because Lowery failed to present reliable expert testimony in support of his claims, or any other evidence permitting a reasonable inference of causation – as opposed to pure juror

speculation – the trial court’s decision granting summary disposition to Enbridge should be affirmed.

II. ARGUMENT

A. **Lowery misstates the appropriate standard for reviewing Enbridge’s motion for summary disposition.**

As an initial matter, Lowery misstates the standard for reviewing motions brought under MCR 2.116(C)(10), asserting that the test is “whether the kind of record which *might be developed* . . . would leave open an issue upon which reasonable minds might differ,” and that “[b]efore summary disposition may be granted, the court must be satisfied that it is *impossible* for the claim asserted to be supported by evidence at trial.” (Pl’s Br at 15) (citation and internal quotation marks omitted; first emphasis added)). This Court long ago overruled that standard in *Smith v Globe Life Ins Co*, 460 Mich 446; 597 NW2d 28 (1999):

We take this occasion to note that a number of recent decisions from this Court and the Court of Appeals have, in reviewing motions for summary disposition brought under MCR 2.116(C)(10), erroneously applied standards derived from *Rizzo v Kretschmer*, 389 Mich 363; 207 NW2d 316 (1973). These decisions have variously stated that a court must determine whether a record “might be developed” that will leave open an issue upon which reasonable minds may differ . . . and that summary disposition under MCR 2.116(C)(10) is appropriate only when the court is satisfied that “it is impossible for the nonmoving party to support his claim at trial because of a deficiency that cannot be overcome.” *Paul v Lee*, 455 Mich 204, 210; 568 NW2d 510 (1997); *Horton v Verhelle*, 231 Mich App 667, 672; 588 NW2d 144 (1998).

These *Rizzo*-based standards are reflective of the summary judgment standard under the former General Court Rules of 1963, not MCR 2.116(C)(10). . . . [I]t is no longer sufficient for plaintiffs to *promise to offer* factual support for their claims at trial. As stated, a party faced with a motion for summary disposition brought under MCR 2.116(C)(10) is, in responding to the motion, required to present evidentiary proofs creating a genuine issue of material fact for trial. Otherwise, summary disposition is properly granted. MCR 2.116(G)(4).

Consequently, those prior decisions of this Court and the Court of Appeals that approve of *Rizzo*-based standards for reviewing motions for summary disposition brought under MCR 2.116(C)(10) are overruled [*Id.* at 455 n 2 (some internal citations omitted).]

B. The need for expert testimony in order to establish causation in toxic tort cases is well recognized.

Despite Plaintiff's assertion, Enbridge is not attempting to "raise the evidentiary threshold" in toxic tort cases. (See Pl's Br at 3, 23). Instead, Enbridge's position is based on basic causation principles, along with the well-established requirement of expert testimony when issues requiring scientific knowledge are presented. Under *Skinner v Square D Co*, 445 Mich 153; 516 NW2d 475 (1994), a plaintiff "must present substantial evidence from which a jury may conclude that more likely than not, but for the defendant's conduct, the plaintiff's injuries would not have occurred." *Id.* at 164-165. This includes "exclud[ing] other reasonable hypotheses with a fair amount of certainty." *Craig v Oakwood Hosp*, 471 Mich 67, 87-88; 684 NW2d 296 (2004). As courts have widely recognized, in order to do that in toxic tort cases, expert testimony is ordinarily required. Why? Because causation inquiries in those cases – which involve analyzing the impact of various chemical compounds upon human beings – are *scientific* in nature. See *Pluck v BP Oil Pipeline Co*, 640 F3d 671, 677 (CA 6, 2011).

Contrary to Plaintiff's assertion, there is nothing unusual or unfair about requiring the assistance of an expert when causation involves scientific assessments that are beyond the common knowledge and experience of jurors. It is no different than showing causation in medical malpractice cases, in which this Court has *repeatedly* held that expert testimony is generally required. See, e.g., *Woodard v Custer*, 473 Mich 1; 702 NW2d 525 (2005); *Bryant v Oakpointe Villa Nursing Centre*, 471 Mich 411, 429; 684 NW2d 864 (2004). In fact, this Court has long recognized that determining the cause of *any* "physical ailment" ordinarily calls for expert testimony. 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.").

Of course, expert testimony may not *always* be required. For example, in *Higdon v Kelly*, 371 Mich 238, 247; 123 NW2d 780 (1963), the Court did not believe expert testimony was necessary “to show that 10 to 12 glasses of beer in an afternoon are sufficient to cause intoxication.” And while it presents a closer call, *Genna v Jackson*, 286 Mich App 413; 781 NW2d 124 (2009), involved a situation in which there was no dispute that the plaintiffs’ formerly healthy children were exposed to “extremely high levels” of toxic mold in their home and experienced an immediate onset of “severe health problems” – consistent with mold exposure – that went away as soon as the children were removed from the home.

This case is vastly different. As discussed more fully in Enbridge’s principal brief, the circumstances surrounding Lowery’s alleged exposure and the symptoms he claims to have suffered are such that expert testimony was required to establish causation. Lowery claims to have been exposed to harmful levels of VOCs despite the fact that he lived more than ten miles away from the release site. That distance is significant because the VOCs in the oil would have begun to dissipate into the air as the oil traveled downriver.¹ Moreover, Lowery asserts that the vomiting he 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 himself said the smell of oil went away. (See Enbridge’s Br at 6-7).² This lack of temporality, combined with the considerable distance that

¹ In the Court of Appeals, Lowery submitted congressional testimony by a scientist from the National Institutes of Health, Scott Masten, Ph.D., about the chemical nature of crude oil and the potential for human health effects. (See **Exhibit 1** (attached as Exhibit E to Plaintiff’s Court of Appeals Reply Br)). One of the things Dr. Masten noted was that “[t]he oil nearest the source of a spill contains higher levels of some of the more volatile hazardous components.”

² Lowery cites an unsigned “affidavit” from his girlfriend, Ashlee Green, in which she claimed that the smell of oil was “so strong in the first two weeks after the spill, it was almost unbearable.” (See Pl’s Br at 6). Not only is that statement inconsistent with Lowery’s own deposition testimony, in which he testified that the smell went away after “about five to seven days” (Lowery Dep at 43, App 32a), but even if it were to be credited, the date of Lowery’s

Footnote continued on next page ...

the oil had to travel before it reached the section of the river where Lowery lived (as well as the indisputable fact that the VOCs that Lowery claims caused his symptoms are not unique to crude oil),³ is precisely why expert testimony was required to demonstrate causation here.

Perhaps more important, Lowery's alleged symptoms can be explained just as plausibly by his use of Lamictal and the Vicodin he took at some point before the vomiting he claims caused his gastric artery to rupture – an injury that raises yet another causation issue. (*Id.* at 8-10). As other courts have widely held, “when there is no obvious origin to an injury and it has multiple potential etiologies, expert testimony is necessary to establish causation.” *Brown v Burlington Northern Santa Fe Railway Co*, 765 F3d 765, 771 (CA 7, 2014).

Lowery is critical of Enbridge's reliance on *Pluck* and other cases recognizing the need for expert testimony in cases like this one because “they have to do with plaintiffs who claim to be suffering from diseases, such as lymphoma, liver disease, lung cancer, squamous cell carcinoma, or leukemia.” (Pl's Br at 18-19). While the causal connections in those cases might have been even *more* attenuated, the point is the same – establishing injury as a result of exposure to a toxic substance, except perhaps in unusual cases where the causal link is manifestly obvious, involves scientific inquiries requiring the assistance of an expert.

C. A jury can only speculate as to whether Lowery's alleged injuries were caused by exposure to oil vapors.

With or without expert testimony, Lowery's claims fail because there is no evidence permitting a jury to reasonably infer causation. In finding there to be a jury-submissible

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alleged vomiting episode and artery rupture on August 18, 2010 was *still* more than a week after Ms. Green claims the smell of oil was “so strong.”

³ See U.S. Environmental Protection Agency, “Volatile Organic Compounds' Impact on Indoor Air Quality,” <<https://www.epa.gov/indoor-air-quality-iaq/volatile-organic-compounds-impact-indoor-air-quality>> (accessed July 26, 2016) (“VOCs are emitted by a wide array of products numbering in the thousands.”).

causation issue, the Court of Appeals majority relied on nothing more than 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. (See COA Op at 3, App 251a). Yet as federal courts from around the country have held, a plaintiff in a toxic tort case has to 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., *Pluck*, 640 F3d at 679 (“[T]he mere existence of a toxin in the environment is insufficient to establish causation without proof that the level of exposure could cause plaintiff’s symptoms.”). Instead, a plaintiff must have at least *some* evidence that he or she was actually exposed to a harmful chemical at a level sufficient to cause the symptoms being alleged. See, e.g., *Wright v Willamette Industries, Inc*, 91 F3d 1105, 1107 (CA 8, 1996) (“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. Lowery’s medical expert, Dr. Nosanchuk, admitted that he did not review any of the available air monitoring results or sampling data gathered in the weeks and months following the Line 6B incident.⁴ This is fatal to Lowery’s claims. See *Pluck*, 640 F3d at 679 (affirming summary judgment because the plaintiffs’ expert “did not ascertain

⁴ While Lowery asserts that Enbridge “never argued” in the trial court that there was “air sampling and air monitoring by the EPA” (Pl’s Br at 7 n 6), the fact is that Enbridge repeatedly stressed Dr. Nosanchuk’s lack of information concerning Lowery’s alleged exposure. (See Enbridge’s Mot for Summ Disp at 9-12, App 21a-24a; Enbridge’s Summ Disp Reply Br at 2-6, App 182a-186a). Moreover, contrary to Lowery’s assertion at p 20 n 16 of his brief, it can hardly be said that there is “no dispute” that VOCs were released “near [Lowery’s] home.” That is the whole point here – there was no such evidence presented.

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").⁵

In an effort to get around this failure, Plaintiff points to what he claims to be "extensive evidence of his exposure to toxic fumes." (See Pl's Br at 25-27). Lowery cites the fact that he lived near the Kalamazoo River and that he and others could "smell" oil. But there is no record support for the proposition that "smelling" crude oil means that a person has been exposed to VOCs at a level sufficient to cause injury. And even if there were some connection between "smelling" crude oil and the symptoms Lowery allegedly suffered, Lowery testified that while he smelled oil during the first several days after the incident, the odor went away a week before his alleged vomiting episode. (See discussion at pp 6-7 of Enbridge's Br). Lowery certainly presented no evidence suggesting that his potential exposure to VOCs could be ascertained by his ability to "smell" oil that had traveled for more than 10 miles in an open waterway before it reached the area where Lowery lived.

Lowery also cites to what he claims to have been a "voluntary evacuation" of the "area where [his] home was located," but that claim is both lacking in evidentiary support and beside the point. Lowery cites no evidence that the voluntary evacuation notices issued by the Calhoun

⁵ Lowery argues that the reliability of Dr. Nosanchuk's testimony cannot be assessed without a full-blown *Daubert* hearing, but that is plainly wrong. As discussed at pages 30-31 of Enbridge's principal brief, both this Court and the Court of Appeals have rejected unreliable expert testimony at the summary disposition stage. Most recently, in *Elher v Misra*, 499 Mich 1; 878 NW2d 790 (2016), the Court reinstated a trial court's grant of summary disposition to the defendants that was based on the trial court's agreement with the defendants that the plaintiff's expert's testimony was unreliable. The trial court made that determination in the course of ruling on the defendants' motion for summary disposition, and without conducting a separate *Daubert* hearing. See also *Waskowski v State Farm Mut Auto Ins Co*, 970 F Supp 2d 714, 722 (ED Mich, 2013) (rejecting unreliable expert report, granting summary judgment to the defendant, and observing as follows: "[A] district court is not obligated to hold a *Daubert* hearing . . . and this Court declines to do so here. A *Daubert* hearing is unnecessary here in light of the full briefing of the issues by the parties and the evidence submitted to date.").

County Public Health Department extended to the area where he lived (they in fact did not). But even if there was a voluntary evacuation, it does not establish Lowery's actual exposure, if any, to VOCs weeks after the release and any evacuation. The same can be said about cleanup workers wearing "Hazmat suits" "in the area." There is no evidence concerning when or where this was, and once again, it sheds no light on whether Lowery, who was not a cleanup worker, was exposed to harmful levels of VOCs in his home *three weeks* after the oil incident.

Nor is there any merit to Lowery's assertion that he can prove his own exposure to VOCs by citing to claims by other unidentified residents living at unknown distances from the oil release that they suffered "similar symptoms." In *Savage v Peterson Distributing Co, Inc*, 379 Mich 197; 150 NW2d 804 (1967), this Court stated the applicable evidentiary rule:

An issue as to the existence or occurrence of particular fact, condition, or event, may be proved by evidence as to the existence or occurrence of similar facts, conditions, or events, *under the same, or substantially similar, circumstances*. [*Id.* at 202 (emphasis added).]

That rule, however, has no application here because Lowery submitted no evidence concerning who those residents were, where they lived in relation to Lowery, or how their alleged exposures to VOCs compare with Lowery's own alleged exposure.⁶ In other words, Lowery failed to present any evidence that those other residents were exposed to VOCs under "the same, or substantially similar circumstances." See *Burton v CSX Transp, Inc*, 269 SW3d 1, 12 (Ky, 2008) ("Without any indication that other 'CSX workers' were subject to similar workplace conditions as Burton—such as working in the same area or being exposed to the same types and amounts of solvents—the fact that other CSX workers may have suffered similar symptoms is of diminished probative value in proving that Burton's ailments were caused by exposure to solvents at

⁶ Despite Lowery's assertion, there was no "deposition testimony" from any of those individuals. (See Pl's Br at 25).

CSX.”); *Whaley v CSX Transp, Inc*, 362 SC 456, 483-484; 609 SE2d 286 (2005) (“[T]he trial judge permitted Whaley to submit evidence that, between 1984 and 2000, CSX had received ninety-seven employee complaints about heat. . . . We hold that this evidence should not have been admitted. Whaley did not establish that the reported complaints and injuries stemmed from the same or similar circumstances as his injuries.”).⁷

Finally, Lowery cites a “CDC-NIOSH Pocket Guide to Chemical Hazards” (App 170a), which provides information concerning potential harmful effects of exposure to chemicals such as xylene, toluene, and benzene *at certain levels*. This “pocket guide,” however, provides no support for Plaintiff’s assertion that “any level of exposure” can cause such symptoms, let alone that exposure to VOCs caused, or could have caused, Plaintiff’s alleged symptoms. (See Pl’s Br at 4). As cases such as *Pluck* demonstrate, there must be proof – through a qualified expert – of exposure at a level sufficient to cause the claimed symptoms.

The lack of evidence concerning Lowery’s potential exposure, if any, to VOCs, is even more striking given the other explanations for Lowery’s alleged symptoms. Lowery’s medical records are replete with references to a pre-incident history of headaches and nausea that Lowery had long attributed to his use of the antidepressant drug Lamictal, especially when he smoked.⁸

⁷ Thus, the trial court was wrong to the extent it believed that because it had previously recognized a causal link between the oil release and other plaintiffs’ alleged symptoms, that was enough for Lowery to show causation with respect to his “vomiting and headaches.” (App 243a). Lowery was required to provide evidence of *his* exposure to VOCs. *Pluck*, 640 F3d at 679.

⁸ Lowery makes much of Dr. Nosanchuk’s dismissal of Lamictal as a potential cause of his headaches (despite lacking any information about the dosages Lowery was taking (see Enbridge’s Br at 41)), as well as the testimony of his treating psychiatrist, Anoop Thakur, M.D., who doubted that Lamictal was the cause (although she conceded that physicians “have to go by patient’s reporting how they [feel], because they know their body best”). (See Pl’s Br at 7-8, 28). Neither physician, however, offered any scientific basis for excluding Lamictal as a potential cause of Lowery’s headaches, *even though headaches are one of that medication’s*

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And he was so convinced that taking Vicodin caused him to vomit the day his gastric artery ruptured that he was afraid to take it in the hospital after his surgery. (Enbridge's Br at 9-11). Despite the Court of Appeals majority's assertion that "this only serves to highlight that there are genuine issues of material fact to be resolved by a jury" (App 251a), the dissent correctly recognized that it instead exposes Lowery's claims as based on nothing more than speculation.

The same goes for the claimed causal link between Lowery's alleged vomiting and the rupture of his gastric artery. Lowery maintains that it is "easily understood by the average juror that an extended bout of coughing and vomiting . . . could result in the tearing of the short gastric artery" (Pl's Br at 19). But it is hard to see how an average juror could reach that conclusion given that Lowery's *own surgeon* could not say what caused the rupture. (Koziarski Dep at 18 (App 60a)). Dr. Nosanchuk once again offered nothing but conclusory and unsupported assertions having no foundation whatsoever in science or medicine. (See Enbridge's Br at 48-50).⁹ A jury cannot be permitted to engage in the very speculation that Lowery's own surgeon, a medical doctor, rejected.

III. RELIEF REQUESTED

For all of these reasons, and as further discussed in its principal brief, Enbridge requests that the Court reverse the Court of Appeals' decision and remand for reinstatement of the trial court's decision granting summary disposition to Enbridge.

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recognized side effects. See the U.S. Food and Drug Administration's "Medication Guide" for Lamictal, <<http://www.fda.gov/downloads/Drugs/DrugSafety/UCM152835.pdf>> (accessed July 23, 2016).

⁹ Lowery asserts that Dr. Nosanchuk relied on "scientific literature supporting a causal link between Plaintiff's vomiting and his ruptured gastric artery" (Pl's Br at 21), but neither of the article abstracts he cited purport to establish a causal connection between vomiting and avulsion of the gastric artery. (See Exhibits A and B to Enbridge's Br).

Respectfully submitted,

DICKINSON WRIGHT PLLC

/s/ Phillip J. DeRosier

By: _____

Kathleen A. Lang (P34695)
Michael G. Vartanian (P23024)
Phillip J. DeRosier (P55595)
Kelley M. Haladyna (P63337)
500 Woodward Avenue, Suite 4000
Detroit, MI 48226
(313) 223-3500

Dated: July 29, 2016

Attorneys for Defendants-Appellants

DETROIT 40856-38 1394405

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EXHIBIT TO DEFENDANTS-APPELLANTS' REPLY BRIEF

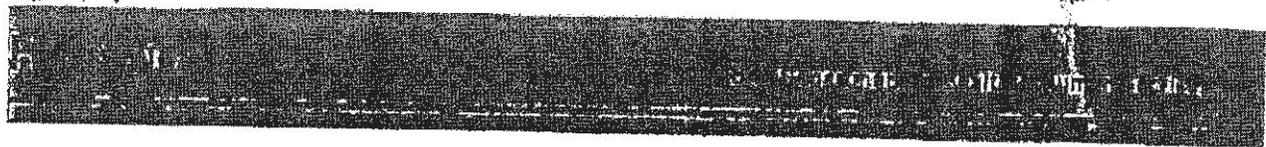
DICKINSON WRIGHT PLLC
Kathleen A. Lang (P34695)
Michael G. Vartanian (P23024)
Phillip J. DeRosier (P55595)
Kelley M. Haladyna (P63337)
500 Woodward Avenue, Suite 4000
Detroit, MI 48226
(313) 223-3500

Attorneys for Defendants-Appellants

TABLE OF CONTENTS

Exhibit 1 September 15, 2010 Statement by NIH Staff Scientist Scott Masten, Ph.D., on
“Enbridge Pipeline Oil Spill in Michigan” before the Committee on
Transportation and Infrastructure, United States House of Representatives

Exhibit 1



Testimony

Statement by
Scott Masten, Ph.D.
Staff Scientist, National Toxicology Program
National Institutes of Environmental Health Sciences
National Institutes of Health
U.S. Department of Health and Human Services (HHS)

EXHIBIT 3
WIT: Jusan Chuk
DATE: 9-25-13
Helen J. Ogden, CBR, RPR

on
Enbridge Pipeline Oil Spill in Michigan
before
Committee on Transportation and Infrastructure
United States House of Representatives

Wednesday September 15, 2010

Chairman Oberstar, Ranking Member Mica, and members of the Committee, thank you for the opportunity to provide information about the potential human health issues associated with oil spills. My name is Scott Masten, and I am a staff scientist at the National Institute of Environmental Health Sciences (NIEHS), one of the Institutes of the National Institutes of Health (NIH), an agency of the Department of Health and Human Services (HHS). My work supports the National Toxicology Program, an interagency program that is administratively housed at the NIEHS, whose mission is to evaluate agents of public health concern by developing and applying tools of modern toxicology and molecular biology. The program maintains an objective, science-based approach in dealing with critical issues in toxicology and is committed to using the best science available to prioritize, design, conduct, and interpret its studies.

I am testifying today on behalf of NIH, and I shall present a brief overview of our current understanding of possible human health effects of exposures related to oil spills, along with a preview of some of our research efforts aimed at increasing our understanding of and hopefully preventing adverse health impacts among oil spill response workers and exposed communities.

Chemical nature of crude oil

Crude oil is a complex combination of chemicals consisting predominantly of carbon and hydrogen, known collectively as hydrocarbons. These elements are predominantly present in straight chains or in a variety of cyclic configurations. Oil may also contain small amounts of nitrogen, oxygen, and sulfur compounds, and trace amounts of metals. Crude oils are broadly categorized as light, medium, or heavy depending on the relative proportion of different sized hydrocarbons. A light crude oil has a higher proportion of smaller, more volatile hydrocarbons. The chemical composition of crude oils can vary substantially from different geographic regions and even within a particular geological formation. [1] It is worth noting that the crude oil released from the Deepwater Horizon rig in the Gulf of Mexico is a light crude, while the oil spilled from the Enbridge pipeline in Michigan is a heavy crude oil.

There are hundreds if not thousands of chemicals in crude oil, and we have incomplete knowledge of the toxicity of many of them. We are most concerned about a particular class of hydrocarbons, known as aromatic hydrocarbons as well as other volatile organic compounds (VOCs), such as benzene, naphthalene and polycyclic aromatic hydrocarbons (PAHs). Sulfur compounds, such as hydrogen sulfide, and heavy metals such as aluminum, lead, nickel and vanadium can also be present to varying degrees in crude oil. These substances may also be of concern depending on their level in the crude oil. From studies of these chemicals individually, we know quite a bit about their hazardous properties and we believe these are some of the chemicals most likely to be encountered in air, sediment or water subsequent to an oil spill. The composition of spilled oil changes over time, and the oil nearest the source of a spill contains higher levels of some of the more volatile hazardous components. Oil that has been exposed to air and water for a period of time, so-called "weathered oil", has lost most of these volatile components. Nonetheless, weathered oil still contains less volatile hazardous chemicals, and therefore skin contact should be limited. If aerosolized by wind or physical disturbance, weathered oil also could be taken into the body through respiration. It is critically important to note that the specific risks of developing adverse health effects are dependent on many factors, but most importantly, risks increase with prolonged exposures to higher concentrations of the chemicals. Protective equipment can be effective at reducing exposures and thereby reducing risks.

Effects on human health from oil spills

Determination of actual exposure and risk for any hazardous chemical release is not a trivial task. Given the chemicals present in crude oil, the potential for human health effects exists; however, understanding and quantifying these effects requires further study. There has been relatively little long-term research into the human health effects from oil spills, although between 1970 and 2009 there were 356 accidental spills of more than 700 metric tons from oil tankers worldwide, with approximately 38 of those spills affecting coastal populations. In a recent article in the *Journal of Applied Toxicology*, the authors reviewed the results of studies of human health

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effects related to oil tanker spills as reported in 34 publications.[2] The clearest conclusion from the examination of these studies is that we have very little data; follow-up of exposed people has occurred only for a handful of the tanker spill incidents from the past several decades. The few studies that have evaluated the human health consequences of oil spills have primarily focused on acute physical effects and psychological sequelae. Historically, the workers involved in cleanup have reported the highest levels of exposure and the most acute symptoms, when compared to subjects exposed in different ways, as seen in the reporting of higher levels of lower respiratory tract symptoms in fishermen who participated in cleanup following the Prestige tanker accident off the coast of Spain. Studies have examined the Exxon Valdez (Alaska, 1989), Braer (Shetland Islands, UK, 1993), Sea Empress (Wales, UK, 1996), Nakhodka (Oki Islands, Japan, 1997), Erika (Brittany, France, 1999), Prestige (Galicia, Spain, 2002) and Tasman Spirit (Karachi, Pakistan, 2003) oil tanker spills. A number of the studies reported respiratory symptoms, including cough and shortness of breath [3] and decreased lung function [4], among workers involved in cleanup operations. Other commonly reported symptoms in these studies include itchy eyes, nausea/vomiting, dizziness, headaches, and skin irritation/dermatitis. [5] Additionally, several studies of Prestige oil spill clean-up workers have found evidence of genetic and endocrine effects in exposed individuals. Findings include increased DNA damage and chromosomal alterations in white blood cells and decreases in blood prolactin and cortisol levels. [6] Other studies have looked at psychological effects of spills, both among workers and in affected communities; follow-up studies of affected populations from the Exxon Valdez spill, for example, reported higher levels of generalized anxiety disorder, post-traumatic stress disorder, and depressive symptoms approximately one year after the spill occurred. [7] Similar patterns of higher anxiety and depression were observed among communities near the Sea Empress spill. [8] The Braer spill was associated with increased anxiety and insomnia [9], and lower levels of mental health were related to proximity to the Prestige spill. [10] Such research findings remind us of the importance of keeping longer-term, less obvious sequelae in mind, not just the immediate toxicity effects, when considering the overall human health impact of this type of disaster.

NIH-Funded Research

The NIH is using a variety of funding mechanisms and programs to carry out important research related to the human health impacts of oil spills. We expect this research to provide useful information for policy makers, health care providers, and the public.

The NIEHS, through the NTP, has completed important steps in identifying knowledge gaps for oil spill-related exposures of concern. The NTP has reached out to key agency partners to assess ongoing research activities within the federal government and to begin compiling common toxicology research needs. Initial NTP research efforts are focused on chemical characterization of oil and dispersant samples collected in the Gulf region to gain a better understanding of the physical and chemical changes associated with weathering and biodegradation. The NTP has also partnered with the National Institute for Occupational Safety and Health (NIOSH) within HHS's Centers for Disease Control and Prevention to provide analytical chemistry support for NIOSH's planned toxicology studies. The output from these various chemistry analyses will guide the development and conduct of additional toxicological studies to identify important biological and tissue targets for the mixed exposure encountered during oil spills. The NTP toxicology studies will be aimed at characterizing long-term health hazards of exposures that are relevant to oil spill response workers and seafood consumers.

In June, NIH Director Francis Collins announced that the NIH will devote at least \$10 million to support NTP studies and initial stages of an NIEHS-led large prospective health study of oil spill clean-up workers and volunteers, termed the "Gulf Worker Study". In addition, BP has contributed \$10 million through its Gulf of Mexico Research Initiative (GRI) to help fund the Gulf Worker Study. [11]

The Gulf Worker Study will focus on exposure to oil and potential health consequences such as respiratory, neurobehavioral, carcinogenic, and immunological conditions. The study plan also includes evaluation of mental health concerns and other oil spill-related stressors such as job loss, family disruption, and financial uncertainties. A draft protocol for this study was published on the NIEHS website last week [12] and will be reviewed at an Institute of Medicine workshop in Tampa, Florida on September 22, 2010. [13] The study plan will be updated as comments and suggestions are received from the Gulf communities and scientific experts via a series of NIEHS-sponsored meetings, community fora, and webinars.

In addition, the NIEHS has a grants program for time-sensitive research and community education. We are using this program to fund research on the public health impact of the oil spill in the Gulf region. Topics considered for funding include environmental monitoring and characterization related to the Gulf oil spill; toxicity testing of complex mixtures using high-throughput techniques and innovative statistical approaches; exposure assessment for individuals and populations; and research on various health effects, including understanding the unique risks of vulnerable populations, such as children, pregnant women, the elderly, and people with chronic health problems.

Although the above mentioned research activities are focused on the Gulf region, our expectation is that the research results will have widespread applicability to future public health activities relating to oil spills.

Conclusion

It is clear from our current and ongoing review of the available research studies regarding human health effects of spilled crude oil that there is a need for additional health monitoring and research. Follow-up of exposed people has only occurred for a handful of the tanker spill incidents from the past several decades. These incidents involved exposure to different types of

crude oil) and in some cases refined petroleum products. Historically, cleanup workers have reported the highest levels of exposure, although for most of these studies, there is a lack of quantitative exposure information. Human health impacts are dependent on the scale of the release and on our ability to minimize exposure through proper safety precautions, training and spill containment. Ongoing and planned research in the Gulf by NIEHS and others will increase our collective understanding and provide a better foundation for making public health decisions for future oil related incidents.

Thank you. I am happy to answer any questions.

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