IN THE UTAH SUPREME COURT

LOIS SMITH, Plaintiff/Appellee, v. VOLKSWAGEN SOUTHTOWNE, INC., Defendants/Appellants.	Appellate Case No. 20190382-SC District Court Case No. 130908362

BRIEF OF *AMICUS CURIAE* PRODUCT LIABILITY ADVISORY COUNCIL, INC.

Appeal from the Third Judicial District Court, Salt Lake County, Utah
Honorable Barry G. Lawrence, Presiding

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I.

STATEMENT OF INTEREST OF *AMICUS CURIAE*

The Product Liability Advisory Council, Inc. ("PLAC") is a non-profit professional association of corporate members representing a broad cross-section of American and international product manufacturers.[1] PLAC seeks to contribute to the improvement and reform of law in the United States and elsewhere, emphasizing the law affecting the liability of product manufacturers and others in the supply chain. PLAC's national perspective arises from experiences of its corporate members in diverse manufacturing industries in courts across the United States. In addition, several hundred leading product liability defense attorneys are sustaining (non-voting) members of PLAC and contribute their breadth and depth of experience to PLAC's activities. Since 1983, PLAC has filed over 1,100 briefs as *amicus curiae* in both state and federal courts, including this Court, presenting the broad perspective of product manufacturers seeking fairness and balance in the law affecting product liability and risk management.

Member companies of PLAC, and other product manufacturers throughout the nation, are sued for personal injuries allegedly caused by toxic exposures arising from use of various types of products. These suits often involve expert testimony on toxic exposures and causation that rest on questionable scientific foundations. This case presents several recurring issues posed in such cases, and to some extent in expert-intensive product liability

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[1] A current list of PLAC's corporate members is available at https://plac.com/PLAC/Membership/corporate_members_pdf.aspx.

cases generally, relating to (1) the admissibility of expert medical causation opinions, and (2) the dose-response evidentiary foundation needed to prove medical causation in toxic tort cases.

These issues pose important ramifications for the reliability of fact-finding and the resulting integrity of judgments in product liability and toxic tort cases depending on expert causation testimony. For these reasons, the case presents questions of vital interest to PLAC and its members.

II.

SUMMARY OF PERTINENT FACTUAL AND PROCEDURAL BACKGROUND

The following background is based largely on the opinion of the District Court granting JNOV and conditionally granting a new trial.

The jury in this case returned a significant damages award in favor of Plaintiff-Appellant Lois Smith ("Smith"). Smith alleged that she had been exposed to an excessive level of carbon monoxide (CO) sufficient to cause not only CO poisoning but permanent brain damage. She alleged her injuries were caused by a diesel fuel leak that developed during a road trip in December 2011 in her 2011 Volkswagen Jetta. She claimed that Volkswagen Southtowne, Inc. ("Southtowne"), the retail seller of the used car, was liable in strict liability and negligence, leading to her alleged CO exposure and injuries.

A. Testimony of Peter Leiss

The verdict rested in part on expert testimony admitted at trial, over defense objections, from automotive engineer Peter Leiss ("Leiss"). He testified that Smith *could have been* exposed to CO from the fuel leak, based on his opinion testimony that (1) CO

could have been produced under the hood of the Jetta and (2) the design of the vehicle included a potential "passageway" for the CO to intrude into the passenger compartment of the Jetta. This caused Smith to be exposed to an undetermined amount of CO for an undetermined period of time. For ease of reference we refer to (1) as his "production opinion." The second opinion is not at issue.

The production opinion was based on (1) a "Fish Tank Test" ("FTT") performed by a lab technician using a methodology and equipment of undetermined functionality and reliability, designed to demonstrate that CO might be produced when diesel fuel drips onto a hot surface at certain temperatures, and (2) Leiss's opinion that those temperatures could be reached in the engine compartment during operation of the Jetta. His ultimate opinion was that "some" CO could have been produced by the fuel leak,[2] and given the passageway, Smith may have been exposed to *some undetermined level of CO* during the December 2011 road trip. Leiss expressly disclaimed having the data (or expertise) necessary to estimate the amount of CO that could have been, much less was, produced during the road trip. He also acknowledged that the FTT data – based on experimentally dripping diesel fuel onto a hot plate within the confines of a fish tank – could not be extrapolated to the production of CO in an engine compartment, nor explain the level of

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[2] The parties disagree over whether Leiss opined to a *possibility* or a *probability* that CO was produced. Certainly testimony of no more than a *possibility* of exposure would present an additional level of disqualifying speculation to the production opinion. *Thurston v. Workers Compensation Fund of Utah*, 83 P.3d 391, 395-97 (Utah Ct. App. 2003). There is no need to resolve this dispute; any probability opinion by Leiss would be (1) unsupported and inadmissible and (2) insubstantial and insufficient to support causation.

exposure, if any, in the passenger compartment. He also admitted that he lacks expertise in chemistry and chemical reactions. And he lacked detailed familiarity with the FTT, including the nature and reliability of the CO monitoring equipment used in the test.

After admitting Leiss's testimony at trial, the trial court granted Southtowne's post-trial motions, holding that the production opinion (1) could not support causation and (2) should have been excluded.

B. Testimony of Dr. Lindell Weaver

Smith called retained physician Dr. Lindell Weaver ("Weaver") to testify that Smith suffered from CO poisoning and permanent brain damage that was caused by CO exposure during the road trip. His diagnostic opinion, that when he saw Smith in April 2014 (almost 2.5 years after the road trip) she had permanent brain damage attributable to CO poisoning, is not addressed by PLAC.

Rather, PLAC's concern is Dr. Weaver's attribution of Smith's CO poisoning and brain damage to the December 2011 road trip, based on his clinical experience, Smith's subjective account of her symptoms and observations during the trip, her description of a temporal relationship between seeing smoke coming from under the hood and experiencing symptoms, and a "differential etiology" analysis. Weaver acknowledged that a carboxyhemoglobin test within a week of the road trip revealed a normal level of CO in Smith's blood.

The trial court admitted Weaver's causation opinion, but post-trial held it (1) insufficient to prove causation and (2) partially inadmissible.[3]

Dr. Weaver's opinion tying Smith's condition and deficits in April 2014 to the 2011 road trip was plagued by several methodological and foundational flaws.

First, given the lengthy gap between the 2011 road trip and his 2014 evaluation and diagnosis of Smith's condition and its cause, Weaver had to reliably account for the possibility that her observed condition was caused by some other exposure or factor that occurred in the intervening period. On this record he did not. Failure affirmatively to consider whether there are significant alternative explanations for the etiology of the injury is the epitome of an unreliable methodology, and renders a causation opinion speculative and scientifically invalid.

Second, there was, in fact, a compelling, alternative explanation for Smith's condition that occurred in the intervening period – she had spent time during those years living, and sleeping, in her car, and would sometimes run the heater in the stationary car for extended periods. This was an alternative, intervening source of *CO exposure*, meaning that even if Weaver had an adequate foundation to conclude Smith's injuries were caused

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[3] The trial court drew an apparent distinction between (1) Weaver's opinion that exposure to CO during the road trip was the source of Smith's CO poisoning and brain damage Weaver diagnosed in 2014 and (2) "actual medical causation." Post-trial, the court held that (1) should have been excluded but (2) was admissible. On cross-appeal, Southtowne challenges the second opinion. Lacking a reliable basis to conclude the Jetta's fuel leak was the source of any CO causing Smith's injuries, Weaver necessarily lacked an adequate basis to opine that the fuel leak was the actual cause of her harm. The specific causation opinion also should have been excluded.

by CO exposure, a reliable specific causation opinion (tying Smith's injuries to the 2011 diesel fuel leak) would need to reliably assess and rule out this known competing explanation for the source of Smith's CO poisoning.

Third, it does not appear that Dr. Weaver offered a reliable explanation reconciling the normal carboxyhemoglobin test and his causation opinion.

Fourth, Dr, Weaver also failed to consider, address, and reliably rule out other alternative causes for the symptoms Smith described and her injuries. Facts apparently unknown to Weaver when he formed his opinion, and not disclosed to him until cross-examination at trial, suggested that Smith may have been suffering from an advanced upper respiratory infection and strep throat – conditions that could explain the symptoms she claimed resulted from the fuel leak.

Importantly, this constellation of nonspecific symptoms were experienced *before and during,* as well as after her alleged exposure to CO on the road trip. Failure to consider and reliably rule out these alternative explanations fundamentally compromised the reliability of Weaver's differential etiology analysis and the conclusions drawn from it. This lack of reliability did not disappear when the alternatives were *later* brought to Weaver's attention, *after* he had reached his causation opinion and expressed it to a jury, even if he maintained, without any scientific explanation, that these alternative possibilities did not *change* his opinion.[4]

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[4] Weaver also apparently learned for the first time at trial that Smith had experienced several medical conditions that cast significant doubt on any attribution of her symptoms and injuries to the fuel leak. Smith had a concussion in September 2010, failed a Romberg

Fifth, Dr. Weaver lacked sufficient facts and data on both ends of the critical dose-response relationship equation. On one side he lacked reliable scientific knowledge of the dose-threshold for CO exposure – the amount and duration of exposure to CO that scientific knowledge in the field of toxicology has shown to be needed to cause CO poisoning and permanent brain damage. On the other side, he lacked data reliably estimating the level of CO to which Smith actually had been exposed in December 2011. Instead, he relied on Leiss's production opinion and the circular reasoning that Smith must have been exposed to enough CO to cause her injuries because the exposure had (in Weaver's view) caused her symptoms and injuries. These analytical gaps left Dr. Weaver with no reliable scientific basis, and the jury without any factual basis, to conclude that Smith was exposed to a toxic level of CO capable of producing the injuries.

C. <u>The Verdict and Post-Trial Motions</u>

The jury returned a verdict for $2.7 million against Southtowne, but the trial court granted post-trial motions for JNOV and new trial. As to JNOV, the court concluded that the expert testimony of Leiss and Weaver did not amount to substantial evidence of causation because there were critical links missing from the causal chain – Leiss had failed to establish that CO was actually produced under the hood of the Jetta, and therefore within the passenger compartment, and there was no substantial evidence that Smith was exposed

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test in October 2010 (indicating a loss of motor coordination), and in November 2010 reported experiencing daily headaches. Leading up to the December 2011 road trip she complained of headache, fever, fatigue, loss of sleep, dizziness, depression, anxiety, coughing and sore throat. She was treated for upper respiratory infection and strep throat *just before and during* her trip, and was diagnosed with an upper respiratory infection soon after the trip.

to a sufficient level of CO to cause the claimed injuries. In the alternative, the court granted a conditional new trial, finding that it had prejudicially erred in admitting Leiss's production opinion and Weaver's medical causation opinion insofar as it tied the December 2011 road trip fuel leak to Smith's injuries.

III.

ISSUES ADDRESSED BY PLAC

PLAC primarily discusses three issues raised by these appeals.

First, Smith argues that *Alder v. Bayer*, 2002 UT 115, 61 P.3d 1068 (2002) obviates the need for evidence that she was exposed to a level of CO that was scientifically capable of causing her injuries. Her broad reading of *Alder* as applicable generally in toxic tort cases, and applicable here, would undermine judicial gatekeeping and allow experts to opine and juries to impose liability based largely on speculation in many cases.

Second, Smith argues that under *Alder* the temporal relationship between Smith's exposure in December 2011 and her symptoms, by itself, and her injury, by itself, supports both a jury finding of causation and an admissible expert causation opinion. That position misreads *Alder*, is fundamentally out of step with the way scientists and courts have treated evidence of temporal connection between exposure and injury, and would authorize speculative verdicts based primarily on subjective evidence and circular reasoning. It should be rejected.

Third, Smith argues that Weaver's medical causation opinion is "presumptively admissible" and sufficiently reliable here because he engaged in the recognized technique

of differential etiology[5] to reach his conclusion. The courts have universally rejected this talismanic approach, and instead require the proponent of the evidence to affirmatively demonstrate that the proffered cause was reliably "ruled in" and leading alternative causal explanations reliably "ruled out" based on valid scientific reasoning. *Tamraz*, 620 F.3d at 674. This Court should reject any rule or standard that admits unreliable and unscientific causation opinions simply because the expert invokes this technique. On this record, it appears that Weaver's differential etiology analysis was compromised because he lacked a reliable scientific and factual basis for ruling in the fuel leak as a potential cause of Smith's injuries and failed to consider and reliably rule out several alternative explanations for the injuries.

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[5] The term "differential diagnosis" is often used to describe the method of isolating a cause by systematically ruling out all the possibilities but one. The term is a misnomer. "Differential diagnosis" is the diagnostic technique of ruling out the various possible conditions suggested by the patient's signs and symptoms to settle on a surviving *diagnosis*. The art and science of determining the patient's *disease* is very different from the much more complex and science-based analysis of determining what may have *caused* the patient's disease. *See generally, e.g.,* Federal Judicial Center, Reference Manual on Scientific Evidence, *Epidemiology*, at 617-18, 690-91 (3d ed. 2011); *Tamraz v. Lincoln Elec. Co.*, 620 F.3d 665, 674 (6th Cir. 2010); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1252 (11th Cir. 2005). We therefore use the more appropriate term "differential etiology" in this brief.

IV.

DISCUSSION

A. Leiss's Production Opinion Was Inadmissible and Could Not Support a Causation Opinion or Finding

As the trial court recognized, Leiss's opinion that CO was created under the hood of Smith's car during the road trip was an essential predicate in the chain of causation. Without the creation of CO from the fuel leak, there would be no toxic exposure in the passenger compartment and therefore no causation. The court was right to find a failure of proof, and right to rule inadmissible Leiss's production opinion, because it lacked an adequate foundation.

First, as the court noted, Leiss lacked the basic expertise to offer an opinion about the production of CO. An engineer, he lacked expertise in chemistry. The process of producing CO is a chemical reaction.

Second, Leiss based his opinion on the FTT, a crude test consisting of dripping diesel fuel on a hot plate in a fish tank and measuring for the presence of CO. Having not conducted the test himself, and lacking the necessary expertise to evaluate the mechanisms, methods and data generated, Leiss could not reasonably rely on the test to draw scientifically valid conclusions and assist the jury. The most basic requirement for expert testimony, helpfulness to the jury, depends on the expert applying his expertise to exercise independent professional judgment and explain issues to the jury that are beyond their ken, but within the expert's. *See, e.g.*, *Cholakyan v. v. Mercedes-Benz USA, LLC*, 281 F.R.D. 534, 544 (C.D. Cal. 2012).

Third, in any event, Leiss candidly acknowledged that he could not extrapolate from the FTT data to what was happening under the hood. The FTT was conducted under static conditions, while the conditions under the hood of a fast moving car are quintessentially dynamic. The FTT data could not reflect the dynamic variables, and Leiss, beyond the scope of his expertise, could not bridge the gap. His lack of familiarity with chemistry principles, as well as the test equipment and methodology, left him incompetent to validate, interpret, and apply the FTT data and tell the jury anything useful about how it would lead to a reliable conclusion that there was any actual production of CO in the engine compartment. *See* *Jennings v. Palomar*, 114 Cal.App.4th 1108, 1117-18 (2004) (explaining that the expert's value depends on his ability to explain how the jury can apply the facts to reach its own conclusion, and why that conclusion is the right one). Leiss's testimony about the FTT data therefore could not create an adequate foundation for an exposure opinion, and ultimately, a causation opinion and finding.[6]

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[6] This illustrates how qualifications and reliable foundation often interact to support, or undermine, opinion testimony. The weak qualifications of an expert witness may exacerbate weaknesses in the foundational predicates for the opinion, thereby justifying exclusion under Rule 702. *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 749 (3d Cir. 1994); *Wehling v. Sandoz Pharms, Corp.*, 1998 U.S. App. LEXIS 38866, *8 (4th Cir. Aug. 20, 1998).

B. The Medical Causation Testimony Offered by Smith Was Inadmissible and Insufficient

1. General Principles Governing Evaluation of Causation Testimony in Toxic Tort Cases

Decades of experience assessing the proffered scientific bases for claims that exposure to a substance caused certain injuries in a specific individual has produced a consensus that medical causation consists of two primary elements:

(1) general or generic causation, which asks whether the substance has been shown to be capable of causing the injury in humans generally under reasonably expectable circumstances of exposure to the substance; and

(2) specific or individual causation, which asks whether the plaintiff's exposure to the substance actually caused their plaintiff's injury.

General causation is a threshold question; if the substance is not capable of causing the injury in the general population, then it cannot logically be the cause of an individual's injury. *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005); *Ruggiero v. Warner-Lambert Co.*, 424 F.3d 249, 254 (2d Cir. 2005).

Specific causation is further subdivided, for ease of analysis: (1) the initial process of "ruling in" the exposure as a potential explanation for plaintiff's injury, and (2) the process of "ruling out" the various competing potential explanations to determine, if possible, whether the exposure is more likely than not the cause of plaintiff's injury to a reasonable medical probability. *See Tamraz*, 620 F.3d at 674; *Pluck v. BP Oil Pipeline Co.*,640 F.3d 671, 678 (6th Cir. 2011).

The threshold ruling-in phase of specific causation[7] requires examining the scientific literature supporting general causation and analyzing whether or not, based on the evidence, the conditions under which the injury can be produced by the exposure appear to exist in the plaintiff's exposure scenario. The toxicologist (or physician applying the principles and methods of toxicology) looks at the details of plaintiff's exposure, including most critically the *manner* (ingestion, inhalation, etc.) and *dose* of his exposure, and compares it to what is known to be needed to cause the injury. The purpose is to determine whether a scientifically supportable inference can be drawn that the exposure caused the injury. If so, then and only then the exposure has been "ruled in" as a potential cause and is eligible for comparison with other available explanations for the injury.

If the exposure has been adequately and reliably ruled in, then a physician may complete the differential etiology analysis by examining each of the competing alternative explanations and systematically ruling them out (or at least assessing their relative causal probabilities) to select the likely cause.[8]

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[7] *See* *Claussen v. M/V New Carissa,* 339 F.3d 1049, 1057-58 (9th Cir. 2003) (absent reliable evidence ruling in the exposure as one of the possible causes of the injury, there is no reliable basis to include the exposure within the differential etiology and to consider whether it did cause the injury).

[8] A caveat is in order here. The expert is not seeking the "most likely" of the various possible causal explanations, but determining whether the exposure more likely than not caused the injury, compared to all other possibilities. For example, if the exposure were to be assigned a 33% likelihood that it produced the injury, and that was the highest percentage of all the competing explanations, that would not support a finding that the exposure was more likely than not the actual cause. This is particularly important where, as is often the case, science has not yet been able to identify all the potential causes of an illness, and a significant percentage of cases in the general population are deemed

These general principles and backdrop frame the following discussion of the specific issues presented by this case.

2. To Properly "Rule In" the Exposure as a Potential Cause, the Medical Expert Must Account for the Fundamental Principle of Toxicology, Dose-Response

Smith argues that the JNOV and new trial orders must be reversed in part because they improperly required Smith and Weaver to demonstrate that Smith was exposed to a level of CO capable of causing CO poisoning and permanent brain damage in humans. While in exceptional cases it may be unnecessary to produce empirical data of the level of exposure, as this Court recognized in *Alder*, those cases are the exception, not the rule. Smith's argument would essentially eliminate the requirement that an expert rigorously consider the dose-response relationship, flying in the face of a central tenet of toxicology.

There is no more fundamental precept in toxicology than that coined by Paracelsus, the father of toxicology, five centuries ago:

> All substances are poisons; there is none which is not a poison.
>
> The right dose differentiates a poison from a remedy.

Curtis D. Klaasen ed., CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS (6th ed. 2001) pp. 4-5. This principle has become known as the dose-response relationship, the idea that whether or not exposure to a substance causes specific beneficial or specific adverse effects is a function of the dose to which one is exposed. *Id.* at 13.

"idiopathic" (having no established cause). *See, e.g.*, *Tamraz*, 620 F.3d at 670-71; *Chapman v. Procter & Gamble Distrib., LLC*, 766 F.3d 1296, 1311 (11th Cir. 2014); *Henricksen v. Conoco Phillips Co.*, 605 F.Supp.2d 1142, 1162 (E.D. Wash. 2009); *Perry v. Novartis Pharms. Corp.*, 564 F.Supp.2d 452, 469-70 (E.D. Pa. 2008).

That basic principle remains gospel. Centuries of scientific study have confirmed that causal relationships between adverse effects and chemical compounds or their by-products ordinarily are dose-related; below the "no observable effect level ("NOEL"), the exposure ordinarily does not cause harm, while above the threshold, causation of certain types of harm is thought possible. Determining that threshold and calculating the dose is, consequently, a crucial step in evaluating causality from purportedly toxic exposures. *See McClain*, 401 F.3d at 1241-42 ("When analyzing an expert's methodology in toxic tort cases, the court should pay careful attention to the expert's testimony about the dose-response relationship …. The expert who avoids or neglects this principle of toxic torts without justification casts suspicion on the reliability of his methodology."); Reference Manual at 635-37, 641-42, 669-70. *See also* Reference Manual at 669 ("If the level of exposure was below [the] no observable effect, or threshold, level, a relationship between the exposure and disease cannot be established.").

Though the law lags science,[9] it eventually aligns with it, and the courts have repeatedly recognized the important role dose-response plays in causality determinations. Accordingly, courts throughout the country have honored and implemented the principle, developing a sophisticated and consistent jurisprudential consensus that the element of dose is a critical consideration when evaluating medical causation.

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[9] *See Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996) ("the courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it.").

To reliably rule in the exposure "[i]n cases claiming personal injuries from exposure to toxic substances, it is essential that the plaintiff demonstrate that [she] was, in fact, exposed to harmful levels of such substances. However, 'precise data on the exact degree of exposure to each chemical' is not required." *Abuan v. General Elec. Co.*, 3 F.3d 329, 333-34 (9th Cir. 1993)(citations omitted); *see also, e.g., McClain*, 401 F.3d at 1241 ("Scientific knowledge of the harmful level of exposure to a chemical plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to to sustain plaintiff's burden of proof in a toxic tort case."); *Mitchell v. Gencorp.*, 165 F.3d 778. 781 (10th Cir. 1999) (same); *Allen v. Pennsylvania Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996) (same); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996) (same). A medical causation opinion is ordinarily inadmissible if the expert relies on inadequate or unreliable data to conclude that the exposure was sufficient to cause the injury. *Glastetter v. Novartis Pharms. Corp.*, 252 F.3d 986, 989 (8th Cir. 2001).

This Court considered a variation of this issue in *Alder*. There, two radiology technicians allegedly were exposed on a daily, systematic basis to a stream of chemicals, including a steady dose of at least two known toxins, glutaraldehyde and hydroquinone. The record clearly demonstrated that the plaintiffs had received a very substantial and harmful dose on a daily basis over a long period of time:

- The radiological machine involved the use and release of the known toxins "on a regular basis", and plaintiffs used the machine all day every day in the radiological suite where they were employed.

- The ventilation system in the suite was operating 80% below the performance specifications designed to protect workers from toxic exposures; or as the Court put it, it was "clear" that the "ventilation was woefully inadequate."
- The two plaintiffs, as well as three others who worked in the suite near the machine, "experienced regular symptomatic irritative responses in the work environment." *Id.* at ¶¶67-70.

Under these circumstances of daily, constant, long-term high exposures to known toxic substances that consistently provoked contemporaneous symptoms in a group of similarly exposed individuals, the Court found there was ample and sufficient evidence of toxic exposure. It was not necessary to define the toxic threshold and demonstrate an excess level of exposure. *Id.* at ¶71.

In reaching this conclusion, *Alder* found support from the approach taken by courts in other jurisdictions that found it unnecessary, in circumstances strongly demonstrating substantial overexposure, to require plaintiff to establish the "precise" level of exposure they had experienced. For example, in *Curtis v. M&S Petroleum, Inc.*, 174 F.3d 661 (5th Cir. 1999) six plaintiffs were exposed to high levels of benzene in daily work at a refinery. It was undisputed that benzene was a distillate of the refinery process and highly toxic, with a very low (1 ppm) occupational exposure limit, and air monitoring at the refinery showed exposures were at least ten times that limit. All six worker plaintiffs suffered from contemporaneous symptoms associated with exposure to high levels of benzene. Under these circumstances, plaintiffs were found to have met their burden of demonstrating their

harmful level of exposure, and they were not required "to show the precise level of benzene to which they were exposed." The court observed that an expert's causation opinion without "sufficient information of the level of benzene to which plaintiffs were exposed would not be based on a reliable methodology. *Id.* at 665-67, 671-72. [10]

In *McCullock v. H.B. Fuller Co.*, 61 F.3d 1038, 1040 (2nd Cir. 1995), plaintiff's four years of daily occupational exposure to pungent fumes from a hot glue pot near his work station, producing throat polyps, together with expert testimony that fumes were in plaintiff's breathing zone, was deemed sufficient to prove his hazardous level of exposure.[11] *See also* *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 264 (4th Cir. 1999)

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[10] Notably, the court affirmed the dismissal of another set of plaintiffs with far more subtle levels of exposure, due to their failure reliably to demonstrate they had experienced a harmful level of exposure. *Id.* at 671 n.9. And in other cases, both before and after *Curtis*, where the circumstances of exposure were less extreme, courts in the 5th Circuit have required more reliable proof that the adverse effects threshold was exceeded. *See, e.g.*, *Moore v. Ashland Chem., Inc.*, 151 F.3d 269, 278 (5th Cir. 1998) (en banc) (holding that a causation opinion regarding exposure to toluene was unreliable where the expert lacked data on the level of plaintiff's exposure); *Allen*, 102 F.3d at 199 (exposure to a known carcinogen and subsequent development of brain cancer inadequate to prove causation; where experts lacked information on level needed to cause cancer and level of plaintiff's exposure, they had no reasonable basis for causation conclusion); *Johnson v. Arkema, Inc.*, 685 F.3d 452, 472 (5th Cir. 2012) (distinguishing *Curtis* and holding that summary judgment was proper where plaintiff "did not offer any evidence that the actual amounts of tin oxide to which he was exposed were of a sufficient concentration to cause his [disease]."

[11] Subsequent 2nd Circuit cases have required a reliable estimate of plaintiff's excessive exposure. *See, e.g.*, *Amorgianos v. National R.R. Passenger Corp.*, 303 F.3d 256, 268-69 (2nd Cir. 2002); *Wills v. Amerada Hess Corp.*, 379 F.3d 32, 48-50 (2nd Cir. 2004) (lack of reliable evidence establishing harmful dose of exposure supported exclusion of expert's causation testimony and summary judgment); *Mallozzi v. EcoSMART Technologies, Inc.*, 2013 WL 2415677, *8-9 (E.D.N.Y. May 31, 2013) ("Here, on the other hand, there is no evidence that plaintiff experienced 'substantial exposure' to peppermint oil, and therefore the level and duration of exposure is crucial."); *Sarkees v. E.I. DuPont de Nemours & Co.*, 2020 WL 906331, *13, 16 (W.D.N.Y. Feb. 25, 2020) (noting requirement that plaintiff

(though air levels were never measured, a long history of exposure to visible levels of ambient talc, a known toxin, obviated the need to quantify the level of over-exposure);[12] *Kannankeril v. Terminiz Int'l, Inc.*, 128 F.3d 802, 808 (3d Cir. 1997) (in case involving numerous exposures to pesticide applied frequently over seventeen month period producing cognitive impairment symptoms, expert "had sufficient knowledge of exposure from his review of Terminix's application records, showing when, how much, and where pesticide had been applied").[13]

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prove exposure to dose capable of causing the harm, but finding it met by evidence and reliable extrapolations of plaintiff's daily occupational exposure); *Smolowitz v. Sherwin-Williams Co.*, 2008 WL 4862981, *6 (E.D.N.Y. Nov. 10, 2008) (in case involving uncertain low level exposures to benzene residue in paint, expert could not prove causation because he could not prove the level of exposure and its ability to cause plaintiff's disease).

[12] Subsequent 4th Circuit cases draw a distinction between the dose evidence required simple and very substantial exposure scenarios compared to more complex and more subtle exposure contexts. *See, e.g., Zellers v. Nextech Northeast, LLC*, 533 Fed. App'x 192, 198-99 & n.8 (4th Cir. 2013) ("[I]n *Westberry*, we held that the plaintiff's expert did not need to cite specific quantitative evidence regarding the plaintiff's level of exposure because the record in that case clearly established that the plaintiff had been substantially exposed to the allegedly harmful substance in such a way that specific evidence was unnecessary, In particular, the allegedly harmful substance in that case was talc powder, and the record was replete with evidence of the plaintiff's substantial exposure to talc … Here, there is no evidence of such substantial exposure. Thus, *Westberry* does not support Mrs. Zellar's claim that she need not put forth specific evidence regarding her level of exposure." (citations omitted)); *In re Lipitor*, 2015 WL 6941132, *5 (D.S.C. Oct. 22, 2015) ("*Westberry* stands for the proposition that generally, a plaintiff must show the minimum level of exposure that is capable of causing injury (general causation) and that any opinion on specific causation must state that plaintiff's level of exposure met this minimum. However, in obvious cases of substantial exposure, the expert need not state a particular quantitative amount for her causation testimony to be admissible.").

[13] Subsequent 3rd Circuit cases continue to require compelling evidence of toxic level of exposure in more complex exposure scenarios. *See, e.g., Henry v. St. Croix Alumina, LLC*, 2009 WL 982631, *10 (D.V.I. Apr. 13, 2009) (though *Kannankeril* and *Heller v. Shaw Indus., Inc.*, 167 F.3d 146 (3rd Cir. 1999) do not require "hard evidence", they did not

Thus, the cases cited in *Alder* and subsequent cases applying them generally draw a sensible distinction: where there is strong evidence that the plaintiff was exposed to a dose capable of causing the harm, evidence quantifying the dose may not be needed. But in cases presenting less extreme and less clear exposures, the courts usually require some form of empirical and scientific analysis of the exposure level compared to the adverse effects threshold, or a reasonable substitute. And likewise, how much precision and detail is required regarding the level of exposure also depends on the particular exposure circumstances in the case.

The common through-line in the cases, including and consistent with *Alder*, is the need for substantial and reliable evidence that the plaintiff(s) were exposed to a high enough level of a known toxin to prove the exposure caused the harm alleged. *See, e.g.*, *Alder*, at ¶¶ 68-70 (ample evidence demonstrated continuous, daily occupational exposure to glutaraldehyde and hydroquinone producing a toxic exhaust stream in a room with woefully inadequate ventilation). Where the exposure is less frequent, less substantial, more subjective, or more remote, then a more rigorous demonstration that plaintiff has experienced an injurious level of exposure will be required.

This case falls into that category.

"abandon[] the element of exposure in toxic tort cases" and in a complex exposure scenario, lack of reliable evidence of the dose requires exclusion of causation opinions); *McMunn v. Babcock & Wilcox Power Generation Group, Inc.*, 2014 WL 814878, *14 (W.D. Pa. Feb. 27, 2014) (finding dose-response requirement requires evidence plaintiffs "were regularly and frequently exposed to a substantial, though unquantifiable, dose of ionized radiation"), *aff'd* 869 F.3d 246, 268-72 (3d Cir. 2017) (requiring either evidence calculating the dose of exposure or evidence of frequent, regular and proximate exposure).

Here, in stark contrast to *Alder*, a single plaintiff at best experienced an unknown level of exposure to CO, a substance ever-present in our environment, and claims that her limited exposure to CO during the road trip produced CO poisoning and permanent brain damage.[14] The evidence of excess exposure to CO is not only not compelling, it is nonexistent. There was a fuel leak in the engine compartment, Smith observed what looked like smoke escaping from the compartment to the outside air, and Smith recounted experiencing non-specific symptoms, many or all of which were consistent with the upper respiratory infection and strep throat she was contemporaneously battling. There was conflicting testimony about the temperatures that were generated under the hood and the temperatures necessary to produce CO even under *static conditions*, but no reliable evidence that CO could be produced under the dynamic conditions at the time of the alleged exposure, much less that CO actually was produced. Even if it were assumed that "some" CO was produced and found its way into the passenger compartment, there was no reliable evidence demonstrating that the level of Smith's actual exposure was high or substantial or reached or exceeded a threshold capable of producing CO poisoning, much less permanent brain damage.

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[14] As Southtowne persuasively argues, the evidence suggests that Smith's exposure to "vapor" (rather than CO) probably occurred during a specific short segment of the road trip, the 44 mile leg between Baker City, OR and La Grande, OR. Brief of Appellee at 9-10. That interpretation is somewhat generous – Smith apparently did not observe the vapor escaping from under the hood until she had reached La Grande and the car was idling in the Wendy's drive-thru line. *Ibid.* On this record, Smith's exposure, even assuming some production of CO, may have been quite fleeting. In any event, the exposure, on this record, is nothing approaching the heavy, ubiquitous exposure to a known toxin demonstrated in *Alder* and the cases the Court discussed in *Alder*.

Under these circumstances, a relaxed standard like that applied in *Alder* is unjustified.

Nevertheless, Smith maintains that *Alder* states a rule of general applicability, rather than illustrates an exceptional case, and argues this Court should reject any requirement that a plaintiff produce dose-response evidence to prove causation. In her view, the approach to causation applied in *Alder* should apply to all toxic tort cases, including hers, where the very fact of exposure is uncertain and the level of exposure, if any, is arguably minimal or moderate. Adoption of such a rule would drastically change the liability landscape and authorize expert opinions and verdicts in toxic tort cases untethered to scientific principles and lacking any reliable basis. The Court should decline that invitation.

Smith also advances several specific arguments to support the admissibility of Weaver's opinion and the jury's causation finding.

First, Smith argues that the temporal connection between the fuel leak and plaintiff's experience of symptoms during her trip adequately demonstrates that she was exposed to CO at a level sufficient to produce her injuries. ***Second***, similarly, she argues that the fact that she sustained her injury is, in and of itself, sufficient to infer that she experienced a level of exposure to CO that was harmful *to her*, relying on the eggshell plaintiff principle. ***Third***, she argues that the causation opinion and finding is adequately supported because Dr. Weaver's used a differential etiology methodology to reach his conclusion. None of these arguments can circumvent the fatal lack of the dose-response

foundation necessary to rule in the fuel leak as a likely cause of CO poisoning and permanent brain damage, or otherwise support the opinion and verdict.

3. A Temporal Connection Ordinarily Cannot Prove Causation, and This Case Is No Exception

Courts in toxic tort cases have long struggled against the tendency of jurors to give unwarranted weight to temporal proximity in evaluating the connection between exposure and disease.

> The issue of the chronological relationship leads to another important point – proving a temporal relationship between taking Metabolife and the onset of symptoms does not establish a causal relationship. In other words, simply because a person takes drugs and then suffers an injury does not show causation. Drawing such a conclusion from temporal relationships leads to the blunder of the *post hoc ergo propter hoc* fallacy.

McClain, 401 F.3d at 1243 (emphasis in original).[15] Ascribing causation based on the speculative possibility that the relationship is causally connected rather than coincidental elevates the anecdotal over the empirical and superstition over science. As one court put it,

> Dr. Hamburger's opinion is bottomed in part on the temporal relationship between between Cerna's ingestion of the pharmaceutical drugs at issue and the onset of Leber's disease. Expert causation theories based solely on the temporal proximity between an ingested pharmaceutical and the resulting injury are not methodologically sound. An opinion based on such methodology is akin to a rooster's belief that because dawn breaks shortly after he stands on the weathercock and sounds his morning crow, he, the rooster,

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[15] "The *post hoc ergo propter hoc* fallacy assumes causality from temporal sequence. It literally means 'after this, because of this." *McClain*, 401 F.3d at 1243.

> causes the sun to rise each day. What the rooster doesn't know is that temporal proximity alone does not prove causation.

Cerna v. South. Florida Bioavailability Clinic, Inc., 815 So. 2d 652, 655-56 (Fla. 3rd Dist. Ct. App. 2002). *See also Rolen v. Hansen Beverage Co.*, 193 Fed.Apx. 468, 473 (6th Cir. 2006) ("Expert opinions based upon nothing more than the logical fallacy of *post hoc ergo propter hoc* typically do not pass muster under *Daubert*."); *Roche v. Lincoln Property Co.*, 278 F. Supp. 2d 744, 764 (E.D. Va. 2003) ("An opinion based primarily, if not solely, on temporal proximity does not meet *Daubert* standards."). *See also* Reference Manual, at 668-69.

For these reasons, only in the most compelling circumstances can an expert support a causation opinion with a close temporal relationship between exposure and injury. *See, e.g., Johnson,* 685 F.3d at 467 ("temporal connection is entitled to little weight in determining causation" but "is entitled to greater weight when there is an established scientific connection between exposure and illness or other circumstantial evidence supporting the causal link.") (quoting *Curtis*, 174 F.3d at 670). *See also Cavallo v. Star Enter.*, 892 F. Supp. 756, 774 (E.D. Va. 1995) (observing that temporal connection is ordinarily entitled to little weight, but there may be instances where the connection is so compelling that it can support a causal inference), *aff'd in part* 100 F.3d 1150(4th Cir. 1996).

Here, the temporal connection is anything but compelling and cannot support a causal inference. Smith was already suffering from a viral illness and nonspecific symptoms when she began her journey. Such symptoms were present before, during, and

after the road trip and her alleged exposure. Furthermore, even if there had been a strong temporal connection between the alleged exposure and the symptoms Smith experienced during and immediately after the trip in December 2011, there is no strong temporal sequence with Smith's alleged subsequent development of permanent brain damage.[16]

In sum, no case has held that a simple temporal relationship can substitute for evidence of dose-response, and in any event, there is no basis on this record for an inference of causation based primarily on temporality.

4. Assuming Causation From the Fact of Injury or Symptoms is Circular Reasoning, Which is Neither Admissible nor Sufficient to Prove Causation

Dr. Weaver and Smith also attempted to support causation by suggesting that Smith's symptoms and injuries after she was allegedly exposed to CO during the road trip were themselves proof that she was exposed to a sufficient level of CO to cause her injury. This is patently unscientific, and courts wisely exclude experts who rely on this type of circular inference. *See, e.g.,* *Sorensen v. Shaklee Corp.*, 31 F.3d 638, 649 (8th Cir. 1994) ("Instead of reasoning from known facts to reach a conclusion, the expert [reasons] from

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[16] *Johnson* illustrates the potential importance of this distinction. There, plaintiff experienced a very high exposure to toxic chemicals while servicing a machine, immediately developed respiratory irritation symptoms, and was diagnosed a few days later with pneumonia. He serviced the same machine a month later and again experienced the symptoms and sought immediate medical treatment. He was diagnosed a month later with chemical pneumonitis. He later brought suit alleging the exposure caused chronic lung conditions, restrictive lung disease and pulmonary fibrosis. The court found that the expert lacked a foundation for his causation opinion that tied the exposure to the chronic conditions, but allowed plaintiff to seek recovery for the acute injuries (i.e., the temporary pneumonia) that manifested immediately after the exposure. 685 F.3d at 467, 471.

an end result in order to hypothesize what needed to be known but was not."); *Nelson v. Tennessee Gas Pipeline Co.*, 243 F.3d 244 (6th Cir. 2001) (excluding circular causation testimony); *O'Conner v. Commonwealth Ed. Co.*, 807 F.Supp. 1376, 1396-98 C.D. Ill. 1992) ("Dr. Scheribel … presumed that the cataracts were radiation induced, and then presumed that the plaintiff must have somehow been exposed to a high enough dose to exceed the threshold in order to have caused the cataracts, thereby justifying his initial diagnosis. This is circular reasoning."); *Mancuso v. Consolidated Edison of N.Y., Inc.*, 967 F.Supp.1437, 1450 ("it is improper for an expert to presume that the plaintiff must have somehow been exposed to a high enough dose to exceed the threshold necessary to cause the illness, thereby justifying his initial diagnosis"). Rather, the expert must reach a conclusion through scientific methods and procedures, such as dose-response analysis, and must not resort to "subjective beliefs or unsupported speculation." *Claussen*, 339 F.3d at 1058.

Moreover, again, the symptoms Weaver relied on based on Smith's testimony that she experienced nonspecific symptoms during the road trip were consistent with Smith's viral illness. The notion that the "fact" of such symptoms under these circumstances would support an inference of causation strains credulity as well as the scientific method.

Nevertheless, based on language in *Alder*, Smith argues that the fact of injury is a proper basis for inferring causation, and dose-response evidence is unnecessary, because she was an "eggshell plaintiff", and what matters is that the dose was harmful *to her*. But this simply begs the question and once again assumes rather than proves causation. Smith's argument would essentially wipe out the causation requirement, as the individual biological

variation tautology would always be an available refuge to dispense with the requirement that the plaintiff demonstrate that the dose was sufficiently high to cause the injury.

Moreover, on a conceptual level, Smith's argument impermissibly bootstraps and converts the eggshell plaintiff doctrine from one authorizing the recovery of the full extent of *damages* from the tortious act (i.e., where causation already has been demonstrated), to a substitution for proof of causation. This facile sleight of hand would cripple the causation element as an important limitation on the reach of tort law. This Court should disclaim this reasoning.

Smith's effort to avoid her burden of proving causation, by resort to circular reasoning and misuse of the eggshell plaintiff doctrine, should be rejected.

5. Use of the "Differential Etiology" Method Does Not By Itself Show Reliability or Support a Causation Finding

Smith is not the first to argue that a well-credentialed medical expert's performance of a differential etiology analysis to reach a causation conclusion is admissible per se. But the courts have rejected this simplistic conclusion, recognizing that though differential etiology is a valid methodology for evaluating causation, it can be misused or distorted like any other approach. Accordingly, the courts have uniformly rejected the notion that a causation opinion is per se admissible if the expert applies this method. *See Claussen*, 339 F.3d at 1057 (only "a *properly conducted* differential diagnosis is admissible under *Daubert*") (emphasis added); *McClain*, 401 F.3d at 1253 ("[A]n expert does not establish the reliability of his techniques or the validity of his conclusions simply by claiming that he performed a differential diagnosis on a patient.").

Rather, the rule is, as it must be, that a differential etiology analysis is admissible and supportive of a finding of causation only if reliably performed. A valid differential etiology analysis requires that the appropriate potential causes be reliably "ruled in", and the leading alternative causes be reliably "ruled out", based on reliable scientific evidence, not subjective beliefs or unsupported speculation. *Claussen*, 339 F.3d at 1057. As discussed above, Dr. Weaver's failure to properly rule in CO exposure as a potential cause of Smith's injuries undermines the validity of his causation opinion.

Dr. Weaver also failed on the other end, by not rigorously and scientifically ruling out leading alternative explanations. He attributed the various nonspecific symptoms Smith claimed to have experienced during and after the road trip to CO exposure despite the undisputed evidence that she was suffering from an upper respiratory infection and sore throat before, during and after the road trip. And even as to CO, Davis failed to reasonably and reliably consider the alternative explanation that Smith was exposed to CO by sleeping in her car with the heater running over the course of the two-year period between the road trip and his diagnosis.[17]

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[17] Smith urges that Weaver did "know" that she had lived in her car, arguing that this was brought to Weaver's attention at deposition, though never elicited before the jury. Even assuming that this knowledge, standing alone, would be relevant – that matters outside the trial record can supply foundation for a challenged trial opinion – it only deepens the reliability concerns. Knowing of but then disregarding, i.e., failing reliably to rule out, potential alternative causal explanations to reach the opinion desired by the plaintiff is a particularly strong hallmark of junk science. *See, e.g. Cooper v. Smith & Nephew*, 259 F.3d 194, 202 (4th Cir. 2001); *Schultz v. Akzo Nobel Paints*, LLC, 721 F,3d 426, 434 (7th Cir. 2013); *Viterbo v. Dow Chem. Co.*, 826 F,2d 420, 424 (5th Cir. 1987).

"Not every opinion that is reached via a differential-diagnosis method will meet the standard of reliability required by *Daubert*." *Best v. Lowe's Home Ctrs., Inc.*, 563 F.3d 171, 179 (6th Cir. 2009). "Although a reliable differential diagnosis need not rule out all possible alternative causes, it must at least consider other factors that could have been the sole cause of the plaintiff's injury." *Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245, 1253 (11th Cir. 2010); *see also Westberry*, 178 F.3d at 265 ("A differential diagnosis that fails to take serious account of other potential causes may be so lacking that it cannot provide a reliable basis for an opinion on causation."); *In re Lipitor*, 892 F.3d 624, 644-45 (4th Cir. 2018). Accordingly, Weaver's lack of proper consideration of reasonable alternative causal explanations, and failure to reliably rule them out, rendered his methodology unreliable and his conclusion speculative.

In sum, Weaver's use of differential etiology analysis does not make his causation opinion admissible, especially where he failed to reasonably consider and reliably rule out leading alternative explanations for Smith's symptoms and injuries.

V.

CONCLUSION

This case presents an opportunity for this Court to clarify the showing needed to prove causation in toxic tort cases, and several associated evidentiary principles governing such cases, where the alleged toxic exposure is uncertain, arguably fleeting or de minimis, or otherwise potentially benign. In such cases, the burden of proof should include satisfaction of toxicology's fundamental dose-response requirement, a showing that the

exposure more likely than not met or exceeded the dose threshold necessary to cause the claimed harm.

An admissible medical causation opinion must conform to these requirements and be founded upon a reliable scientific basis to support a conclusion that the toxic exposure was capable of producing the harm. Circular reasoning is not a scientific basis, nor is reliance on a temporal relationship, absent extraordinary circumstances. And the use of a differential etiology technique to evaluate causation results in an admissible causation opinion only where it is reliably performed and based on scientific methods and principles; it is unreliable and inadmissible where it does not include rigorous consideration and elimination of the leading alternative explanations.

DATED this 10th day of March, 2020.

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CERTIFICATE OF COMPLIANCE

1. This brief complies with the type-volume limitation of Utah R. App. P. 24(g)(1) because this brief contains 8,918 words, excluding parts of the brief exempted by Utah R. App. P. 24(g)(2).

2. This brief complies with the requirements of Utah R. App. P. 21(g).

DATED this 10th day of March, 2020.

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CERTIFICATE OF SERVICE

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