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COURT OF APPEAL, FOURTH APPELLATE DISTRICT

DIVISION ONE

STATE OF CALIFORNIA

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| GARY GARNER, as Personal Representative, etc., Plaintiff and Appellant, v.BNSF RAILWAY COMPANY, Defendant and Respondent. |  D082229 (Super. Ct. No. CIVDS1720288) |

APPEAL from orders and judgment of the Superior Court of San Bernardino County, Lynn M. Poncin and John Nguyen, Judges. Reversed and remanded.

The Yarnall Firm and Delores A. Yarnall, for Plaintiff and Appellant.

Pacific Employment Law, Joseph P. Mascovich; Lewis Brisbois Bisgaard & Smith, Anthony E. Sonnett, V. Alan Arshansky; Sims Law Firm and Selim Mounedji, for Defendant and Respondent.

Plaintiff Gary Garner appeals from a judgment entered against him after the trial court granted BNSF Railway Company’s (BNSF) motions in limine to exclude his causation experts, which resulted in the dismissal of his wrongful death lawsuit before trial. Gary[[1]](#footnote-2) alleged that during the more than four decades his father Melvin Garner spent working for BNSF, Melvin was continuously exposed to toxic levels of diesel exhaust and its chemical constituents. According to Gary, this exposure was a cause of Melvin’s non-Hodgkin’s lymphoma, which Melvin developed after retiring from BNSF and which led to his death in 2014. Gary retained several experts to perform a cancer risk assessment and opine on whether diesel exhaust and its constituents are capable of causing cancer, including non-Hodgkin’s lymphoma, and whether Melvin’s workplace exposure to diesel exhaust in this case was in fact a cause of his cancer. At the outset of trial, however, the trial court granted BNSF’s motions in limine to exclude Gary’s three causation experts from trial, finding that the science the experts relied on was inadequate and there was too great an analytical gap between the data and their opinions. The trial court then entered judgment in favor of BNSF and dismissed the case.

Because the court’s in limine rulings resulted in the equivalent of a nonsuit, we conduct an independent review of the record to determine whether BNSF’s motions were properly granted. We conclude that the trial court erred in excluding Gary’s experts and therefore reverse the orders and judgment with instructions to the trial court to enter new orders denying BNSF’s motions in limine.

FACTUAL AND PROCEDURAL BACKGROUND

A. *Decedent’s Background*

Decedent Melvin Garner worked for BNSF as a trainman, a general term that covers several positions, from approximately 1957 to 1999. Melvin worked as a fireman from 1957 to 1964, an engineer for four months in 1964, again as a fireman from 1965 to 1972, and again as an engineer from 1972 until his retirement in 1999. As a trainman, Melvin operated locomotives in and outside of BNSF railyards in New Mexico, Arizona, and California.

In October 2014, Melvin was diagnosed with non-Hodgkin’s lymphoma, a type of blood cancer. He died on October 18, 2014.

B. *Complaint*

In October 2017, Melvin’s son, Gary Garner, filed this survival and wrongful death action against BNSF, alleging violation of the Federal Employers’ Liability Act (45 U.S.C. § 51, et seq.) (FELA). Gary alleged that Melvin’s non-Hodgkin’s lymphoma was caused by his occupational exposure to various toxic substances and carcinogens, including diesel exhaust, benzene, rock dust from railroad track ballast, asbestos fibers, and creosote. BNSF answered the complaint, generally denying Gary’s allegations and asserting several affirmative defenses.

C. *BNSF’s Motion for Summary Judgment*

In December 2019, BNSF moved for summary judgment on two grounds: (1) the FELA’s three-year statute of limitations barred Gary’s action; and (2) Melvin’s alleged exposure to various substances did not cause his non-Hodgkin’s lymphoma. On the latter ground, BNSF relied on a declaration by their expert Dr. Peter Shields, who summarized his review of all the available medical literature relevant to the causation allegations and opined that none of the substances identified in Gary’s complaint have been causally linked to the development of non-Hodgkin’s lymphoma.

Gary opposed. He submitted declarations from three expert witnesses, Dr. Andrew Salmon, Dr. Joseph Landolph and Dr. Robert Gale, whose depositions had not yet been taken in support of his causation argument. Collectively, these experts opined that the substances listed in the complaint can cause non-Hodgkin’s lymphoma and more probably than not were a cause of Melvin’s non-Hodgkin’s lymphoma.

At the summary judgment hearing, the trial court indicated that it would tentatively deny BNSF’s motion, finding that the question of whether “exposures to the kinds of chemicals and other substances that [Melvin] was subjected to” can and did cause his non-Hodgkin’s lymphoma was an issue for the jury. The parties presented additional argument regarding the issue of causation, and the trial court then stated that it would follow its tentative ruling, explaining: “I think these are triable issues of fact. And the arguments that Defendant has made essentially can be made to the jury going to the weight of the expert testimony.” In August 2020, the trial court denied BNSF’s motion.

D. *Motions in Limine*

After the court’s denial of BNSF’s summary judgment motion, the parties engaged in expert discovery in February and March 2021. The parties also began filing and opposing motions in limine in March 2021 in accord with the then-scheduled trial date.

In March 2021, both parties filed several motions in limine in anticipation of trial. In May 2021, the case was sent to another department for trial purposes, and trial was ultimately continued to September 2021. The trial court heard argument on the parties’ in limine motions on September 7 and 8, 2021.

1. *Motion in Limine No. 4 to Exclude Expert Opinion of Dr. Rosenfeld*

BNSF’s motion in limine number four moved to exclude Dr. Rosenfeld’s opinions that (1) while working for BNSF, Melvin was exposed to significant levels of diesel exhaust and its constituents that substantially increased his risk of developing non-Hodgkin’s lymphoma, and (2) BNSF failed to provide Melvin with a reasonably safe place to work. On appeal, Gary states that the trial court, by minute order and “without explanation,” granted this motion along with the others excluding his causation experts, and he asks us to reverse the trial court’s decision. However, BNSF claims the court did not decide the issue.

Although Gary correctly points out that the court’s September 7, 2021 minute order states that motion in limine number four was granted, this appears to be a mistake. The minute order also states that defense counsel requested an Evidence Code section 402[[2]](#footnote-3) hearing to determine preliminary facts regarding admissibility, which the court granted, and the reporter’s transcript shows that the court stated as follows: “The tentative is still to deny the motion in limine [number four] and allow Dr. Rosenfeld to testify based on the Los Altos case the Court cited, but I will grant the request for a 402 hearing for Dr. Rosenfeld.” The court never actually held the 402 hearing, however, and never issued a final ruling on motion in limine number four.

Where a conflict exists between the court’s statements in the reporter’s transcript and the minute order, “we presume the reporter’s transcript is the more accurate.” (*In re A.C.* (2011) 197 Cal.App.4th 796, 800–801; see also *Arlena M. v. Superior Court* (2004) 121 Cal.App.4th 566, 569–570 [where the minute order reflects something not contained in the reporter’s transcript, “the reporter’s transcript generally prevails as the official record of proceedings”].) We thus agree with BNSF that the trial court did not grant its motion in limine to exclude Dr. Rosenfeld, and we do not address Gary’s arguments as to his opinions.

2. *Motion in Limine No. 7 to Exclude Expert Opinion of Dr. Salmon*

BNSF’s motion in limine number seven moved to exclude Dr. Salmon’s opinions that: (1) Melvin’s exposure to diesel exhaust during his work at BNSF substantially increased his risk of developing non-Hodgkin’s lymphoma; and (2) Melvin’s exposure to diesel exhaust during his work at BNSF was more likely than not a cause of his non-Hodgkin’s lymphoma. BNSF argued that Dr. Salmon had no evidence linking exposure to diesel exhaust with non-Hodgkin’s lymphoma, and that he improperly relied on evidence suggesting a link between diesel exhaust and lung cancer to extrapolate and form his opinion that exposure to diesel exhaust must therefore also be linked to other kinds of cancer, including non-Hodgkin’s lymphoma. In support of its motion, BNSF attached as exhibits Dr. Salmon’s expert report and a handful of excerpts from his deposition. BNSF did not submit any declarations, deposition testimony, or documentation from Dr. Shields, on whom it had relied when seeking summary judgment, or any other experts, nor did it include any scientific studies or journal articles with the motion.

Dr. Salmon’s report explained that his prediction for Melvin’s “additional risk of cancer which he experienced as a result of these exposures” to diesel exhaust—which was “substantial” at between 2864 and 3875 excess cancers per million people—was for “overall cancer incidence, not confined to any particular site of tumor formation.” At deposition, Dr. Salmon described his methodology by stating that “the risk estimate is designed to provide an estimate as the overall risk of cancer and it doesn’t specify that that risk be confined exclusively to the lung cancer site or other specific sites that have been measured and considered in the risk assessment.” According to BNSF, because Dr. Salmon could not limit the excess cancer risk to non-Hodgkin’s lymphoma, his opinions are not specific to Melvin and are thus pure speculation.

BNSF asserted that because Dr. Salmon conceded he did not rely on any study finding that diesel exhaust causes non-Hodgkin’s lymphoma, his opinions were irrelevant and speculative, as they were unconnected to the data on which he purported to rely.

Gary opposed the motion, arguing that BNSF failed to cite any literature requiring a methodology focused solely on the cancer site at issue. Gary argued that Dr. Salmon had explained in his report and at deposition why an excess cancer risk calculation can properly be based on a method looking at any organ site rather than focusing on the organ site at issue. Dr. Salmon testified that “for chemicals that induce tumors at multiple sites, the single-site approach may underestimate the true carcinogenic potential,” so “a statistical procedure may be used to estimate an overall potency”—a methodology taken directly from the Office of Environmental Health Hazard Assessment (OEHHA) of the California Environmental Protection Agency (EPA). Gary also argued that Dr. Salmon’s opinions were supported by the opinions of expert Dr. Joseph Landolph, whose report explained that because of the way diesel exhaust and many of its chemical constituents directly act on DNA and cause genetic mutations, the effects of diesel exhaust are not just limited to one organ in the body.

Gary further argued that Dr. Salmon relied on credible literature providing a reasonable basis for his specific causation opinion, and that he was not required to point to a study conclusively finding that non-Hodgkin’s lymphoma is caused by diesel exhaust. According to Gary, epidemiological literature only establishes statistical *associations* between agents and various diseases—it does not provide conclusions regarding whether a particular agent *causes* a particular disease. Gary stated that epidemiology involves observational data and associations expressed as a statistic, rather than proving causation, which is expressed as a judgment based on the weight of the evidence.

3. *Motion in Limine No. 8 to Exclude Expert Opinion of Dr. Gale*

BNSF’s motion in limine number eight moved to exclude Dr. Gale’s opinions that: (1) diesel exhaust and its particulates benzene, dioxin, and formaldehyde are a cause of non-Hodgkin’s lymphoma in humans; and (2) it is more likely than not, to a reasonable degree of medical probability, that Melvin’s exposure to benzene, dioxin and formaldehyde via his exposure to diesel exhaust was a cause of his non-Hodgkin’s lymphoma. BNSF argued that although Dr. Gale acknowledged that a “dose” is necessary to calculate an excess cancer rate, he did not and could not give an opinion regarding the dose necessary to cause non-Hodgkin’s lymphoma. According to BNSF, this rendered Dr. Gale’s opinions speculative. In support of its motion, BNSF attached Dr. Gale’s expert report, 29 pages of testimony from his deposition, and the opinion from a District of Nebraska case. BNSF again did not submit any declarations, deposition testimony, or documentation from its own experts, nor did it attach any scientific studies or journal articles to the motion.

Gary argued in opposition that BNSF’s demand for a specific number reflecting the minimum dose necessary to causally connect diesel exhaust exposure with non-Hodgkin’s lymphoma was neither scientifically sound nor legally required. According to Gary, the linear no-threshold dose response model, which Dr. Gale used, is well-established and a widely accepted method for modeling cancer risk.

Gary claimed that BNSF was confusing the issue, as the relevant question is not what dose is necessary to *cause* cancer, but rather whether there is evidence of exposure to a dose that sufficiently *increases the risk* of cancer such that the exposure was, more likely than not, a cause of the cancer. Gary also argued that his experts presented a specific dose (“between 2864 and 3875 excess cancers per million persons exposed to that dose”) that is more than trivial through the excess cancer risk calculation performed by Dr. Salmon, which Dr. Gale relied on to support his causation opinion.

4. *Motion in Limine No. 9 to Exclude Expert Opinion of Dr. Landolph*

BNSF’s motion in limine number nine asked the court to exclude Dr. Landolph’s general causation[[3]](#footnote-4) opinion that exposure to diesel exhaust and its constituents can cause non-Hodgkin’s lymphoma. BNSF argued that because Dr. Landolph did not rely on any study that finds that diesel exhaust exposure causes non-Hodgkin’s lymphoma, has not published any literature related to non-Hodgkin’s lymphoma, and has no personal knowledge of how often Melvin would have been exposed to diesel exhaust and/or its constituents, his opinion was speculative and must be excluded. In support of its motion, BNSF attached Dr. Landolph’s expert report, 40 pages of testimony from his deposition, and the same case it attached to its motion seeking to exclude Dr. Gale. BNSF again did not submit any declarations, deposition testimony, or documentation from its own experts, nor did it attach any scientific studies or journal articles.

BNSF also argued that Dr. Landolph’s opinion was speculative because, like Dr. Gale, he could not pinpoint the dose necessary to cause non-Hodgkin’s lymphoma. Dr. Landolph conceded that the general population is exposed to diesel exhaust and its constituents in the background, but not every individual in the general population develops non-Hodgkin’s lymphoma as a result of that exposure. Therefore, BNSF argued, his failure to provide a specific dose of diesel exhaust needed to cause non-Hodgkin’s lymphoma rendered his opinion speculative.

Gary responded that BNSF had ignored a key aspect of Dr. Landolph’s analysis, which explained that diesel exhaust and its constituents are mutagenic, multi-organ carcinogens, meaning that analysis of cancer at one organ site is relevant to analysis of cancers at other sites.[[4]](#footnote-5) Gary also pointed to the fact that Dr. Landolph relied on numerous publications showing that diesel exhaust and its constituents are known human carcinogens and can induce non-Hodgkin’s lymphoma to support his causation opinion.

Gary further argued that, like Dr. Gale, Dr. Landolph was not required to determine the specific dose of Melvin’s toxic exposure to diesel exhaust to support his causation opinion. According to Gary, it is sufficient that Dr. Landolph relied on a specific, non-trivial, calculated dose of exposure to diesel exhaust described by Dr. Salmon as “substantial,” which was at least 3,000 times higher than de minimis. Dr. Landolph then used that dose and excess cancer risk to conclude that diesel exhaust and its constituents, particularly benzene, dioxin, and formaldehyde, are capable of causing many different types of tumors in humans exposed to it, including non-Hodgkin’s lymphoma. Gary argued that because there is no requirement that a plaintiff must show that a specific dose is required before exposure to a substance is harmful or causes the injury alleged, and Gary’s experts demonstrated in any event that Melvin’s exposure was non-trivial, Dr. Landolph’s opinions could not be excluded on that basis.

5. *Trial Court Ruling on* *BNSF’s Motions in Limine*

The trial court held two days of argument on the parties’ motions in limine. On the first day, the court stated that its tentative ruling was to deny each of BNSF’s motions that sought to exclude two of Gary’s experts, Dr. Rosenfeld and Dr. Salmon. The court did, however, grant BNSF’s request to hold a section 402 hearing for Dr. Rosenfeld, though it reiterated that its tentative ruling was to allow Dr. Rosenfeld to testify at trial. The parties argued the issue of causation extensively, focusing in particular on Dr. Salmon’s causation opinions, but also discussing Dr. Gale and Dr. Landolph.

The following day, the trial court announced that it intended to reverse its tentative ruling on the motion to exclude Dr. Salmon’s opinions, stating: “[T]his Court is finding that the science relied upon by Dr. Salmon is inadequate. There is no data, no study, and no testing linking non-Hodgkin’s lymphoma and exposure to diesel exhaust. The Court is further finding that, as stated in Sargon, there is simply too great an analytical gap between the data and the opinion proffered by Dr. Salmon. Therefore, the Court reverses the tentative and grants the defendant’s motion in limine Number 7 to exclude the opinions of Dr. Salmon on liability and causation.”

The trial court also stated that its tentative rulings as to the motions seeking to exclude Dr. Gale and Dr. Landolph would follow its ruling on Dr. Salmon’s opinions, finding that “[t]here is a gap in the analytical data to support the conclusions that diesel exhaust exposure is a causal link or a causal connection to non-Hodgkin’s lymphoma.” The parties then presented additional argument on those motions. After argument concluded, the court granted BNSF’s motions, stating: “[I]n looking at the documents presented, it does not seem that there is enough data for the experts to draw their conclusion much less the Court’s [*sic*] -- with Dr. Salmon, there is an analytical gap that causes the Court to grant the motions in limine to preclude the . . . opinion of Dr. Gale on liability and causation as set forth in the defense motion in limine Number 8 as well as the defense motion in limine Number 9 which the Court will grant . . . excluding certain opinions of Dr. Landol[ph] on causation.”

The court then stated that it would sign the proposed orders from BNSF and set a control date for dismissal to be submitted by the defense. The appellate record contains signed orders granting BNSF’s motions in limine numbers three, five, six, seven, eight, nine, ten, and eleven.

E. *Judgment and Dismissal*

After the trial court granted BNSF’s motions in limine numbers seven, eight, and nine, Gary had no expert witness who could establish a causal connection between BNSF’s conduct and Melvin’s injury. On November 5, 2021, Gary filed a notice of appeal from the orders, explaining that they operated as a nonsuit but did not expressly dismiss the case and acknowledging the appeal may be premature because no judgment had yet been entered.

On November 10, 2021, the trial court executed and filed judgment in favor of BNSF and dismissed the case. We exercise our discretion to construe Gary’s November 5 notice of appeal as being taken from the November 10 judgment. (See Cal. Rules of Court, rule 8.104(d).)

DISCUSSION

A. *Standard of Review*

The parties dispute which standard of review applies to the trial court’s orders granting BNSF’s motions in limine to exclude Gary’s experts from trial. BNSF argues that, under *Sargon Enterprises v. University of Southern California* (2012) 55 Cal.4th 747 (*Sargon*), the abuse of discretion standard of review applies to any evidentiary ruling regarding admissibility of an expert opinion. BNSF emphasizes the Court’s language stating that, “[e]xcept to the extent the trial court bases its ruling on a conclusion of law (which we review de novo), we review its ruling excluding or admitting expert testimony for abuse of discretion.” (*Sargon*, at p. 773.)

Gary, on the other hand, contends that because the trial court’s in limine rulings acted as a nonsuit or summary judgment motion, de novo review applies. He agrees that rulings on expert testimony and motions in limine are generally reviewed for abuse of discretion, but he points to Court of Appeal decisions concluding that this standard does not apply where granting the motion in limine results in dismissal of the cause of action (or entire case) before trial. (See, e.g., *McMillin Companies, LLC v. American Safety Indemnity Co.* (2015) 233 Cal.App.4th 518, 529 [abuse of discretion “standard does not apply where (as here) the grant of the motion becomes a substitute for a summary adjudication or nonsuit motion”].)

We agree with Gary. California courts regularly conclude that “if the trial court’s ruling on a motion in limine precludes an entire cause of action, the ruling is subject to independent review on appeal as though the court had granted a motion for nonsuit.” (*Kinda v. Carpenter* (2016) 247 Cal.App.4th 1268, 1279–1280; see also *Legendary Investors Group No. 1, LLC v. Niemann* (2014) 224 Cal.App.4th 1407, 1411 [“When a motion in limine ‘results in the entire elimination of a cause of action or a defense, we treat it as a demurrer to the evidence and review the motion de novo. . . .’ ”]; *City of Livermore v. Baca* (2012) 205 Cal.App.4th 1460, 1465 (*Baca*) [“When, as in the present case, the court’s order excludes all evidence on a particular claim and, as a result, operates as a motion for nonsuit, we review the court’s order de novo . . . .”]; *Dillingham–Ray Wilson v. City of Los Angeles* (2010) 182 Cal.App.4th 1396, 1402 [“When all evidence on a particular claim is excluded based on a motion in limine, the ruling is subject to independent review as though the trial court had granted a motion for judgment on the pleadings or, if evidence was offered, a motion for nonsuit.”]; *Fergus v. Songer* (2007) 150 Cal.App.4th 552, 569–570 (*Fergus*) [“Where, as here, the granting of a motion in limine disposes of one or more causes of action, it is the functional equivalent of the granting of a nonsuit as to those causes of action.”].)

Here, the trial court’s grant of BNSF’s motions in limine at the outset of trial acted as the “functional equivalent” of an order granting nonsuit, which is subject to de novo review. (See *Fergus, supra*, 150 Cal.App.4th at p. 569; *Edwards v. Centex Real Estate Corp.* (1997) 53 Cal.App.4th 15, 27.) The rulings deprived Gary of essential evidence on causation, resulting in a judgment of dismissal before trial. And unlike in *Sargon*, where the trial court had conducted an eight-day evidentiary hearing on the motion to exclude the expert witness (*Sargon,* *supra*, 55 Cal.4th at p. 755), the trial court here decided the issue solely on the papers. Though not dispositive, this also weighs in favor of de novo review because the trial court did not observe any witnesses testify in court, and we are in the “ ‘same position’ ” as the trial court “when reviewing a cold record.” (*People v. Vivar* (2021) 11 Cal.5th 510, 528 [adopting independent review standard].)[[5]](#footnote-6)

Where the trial court’s ruling operates as a nonsuit and we review the order de novo, “ ‘all inferences and conflicts in the evidence must be viewed most favorably to the nonmoving party.’ ” (*Baca,* *supra*, 205 Cal.App.4th at p. 1465.) In such circumstances, we resolve all presumptions, inferences, and doubts in the appellant’s favor, “and uphold the judgment for respondent . . . only if it was required as a matter of law.” (*Fergus, supra*, 150 Cal.App.4th at pp. 569–570.)

B. *Legal Principles*

1. *The Trial Court’s Gatekeeping Role*

Under California law, the trial court has the duty to act as a “gatekeeper” in determining whether to exclude expert testimony from trial. (*Sargon,* *supra*, 55 Cal.4th at pp. 753, 770.) In carrying out its gatekeeping function, the court is governed by sections 801 and 802.

Section 801 provides: “If a witness is testifying as an expert, his testimony in the form of an opinion is limited to such an opinion as is: [¶] (a) Related to a subject that is sufficiently beyond common experience that the opinion of an expert would assist the trier of fact; and [¶] (b) Based on matter . . . that is of a type that reasonably may be relied upon by an expert in forming an opinion upon the subject to which his testimony relates, unless an expert is precluded by law from using such matter as a basis for his opinion.” Under section 801, therefore, the trial court must exclude speculative or irrelevant expert opinion. (*Sargon,* *supra*, 55 Cal.4th at p. 770.)

Section 802, which also governs the trial court’s gatekeeping role, provides: “A witness testifying in the form of an opinion may state . . . the reasons for his opinion and the matter . . . upon which it is based, unless he is precluded by law from using such reasons or matter as a basis for his opinion. The court in its discretion may require that a witness before testifying in the form of an opinion be first examined concerning the matter upon which his opinion is based.” This section thus permits the court to inquire as to the reasons for an expert’s opinion and whether the material upon which the expert relies actually supports the expert’s reasoning. (*Sargon,* *supra*, 55 Cal.4th at p. 771.) “ ‘A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.’ ” (*Ibid*., citing *GE v. Joiner* (1997) 522 U.S. 136, 146 (*Joiner*).)

In short, “section 801 governs judicial review of the *type* of matter” relied on by the expert, while “section 802 governs judicial review of the *reasons* for the opinion.” (*Sargon,* *supra*, 55 Cal.4th at p. 771; see §§ 801, subd. (b), 802.) The Supreme Court has therefore explained that, under these sections, “the trial court acts as a gatekeeper to exclude expert opinion testimony that is (1) based on matter of a type on which an expert may not reasonably rely, (2) based on reasons unsupported by the material on which the expert relies, or (3) speculative.” (*Sargon*, at pp. 771–772; see §§ 801, subd. (b), 802.)

The Court has warned, however, that trial courts must “be cautious in excluding expert testimony. The trial court’s gatekeeping role does not involve choosing between competing expert opinions.” (*Sargon,* *supra*, 55 Cal.4th at p. 772.) “[T]he gatekeeper’s focus ‘must be solely on principles and methodology, not on the conclusions that they generate.’ ” (*Ibid*.) Nor should the court determine the persuasiveness of an expert’s opinion, weigh the opinion’s probative value, substitute its own opinion for the expert’s opinion, or resolve scientific controversies. (*Ibid*.) Rather, the goal “is simply to exclude ‘clearly invalid and unreliable’ expert opinion.” (*Ibid*.)

2. *Liability Under FELA*

In FELA actions, railroad employees (or their representatives) seeking to recover for on-the-job injuries have the right to sue their employer for any injury “ ‘resulting in whole or in part from the negligence’ of the railroad or its employees.” (*Fair v. BNSF Railway Co.* (2015) 238 Cal.App.4th 269, 275 (*Fair*), quoting *Woods v. Union Pacific Railroad Co.* (2008) 162 Cal.App.4th 571, 577.) “The standard under FELA is a relaxed one,” and it is well established that the “evidence required to establish liability in an FELA case is much less than in an ordinary negligence action.” (*Fair*, at p. 275.) The employer is liable where its negligence “played any part, however small, in the injury or death” at issue. (*Rogers v. Missouri Pac. R.R. Co.* (1957) 352 U.S. 500, 507–508 (*Rogers*).) In interpreting FELA, the United States Supreme Court “has insisted that plaintiffs have a broad primary right to go to the jury on factual issues” and made clear that a plaintiff should “reach the jury on the issue of liability when there is any evidence, ‘even the slightest,’ ” to support his case. (*Jehl v. Southern Pacific Co.* (1967) 66 Cal.2d 821, 834, quoting *Rogers*, at p. 506.)

We now consider whether the trial court properly discharged its gatekeeping responsibility here by excluding Gary’s causation experts from trial and dismissing his FELA action.

C. *Analysis*

The trial court here found that there was “simply too great an analytical gap between the data and the opinion[s] proffered by Dr. Salmon,” Dr. Gale, and Dr. Landolph on liability and causation. The court identified no other flaw in the methodology used by any of the experts. We address each expert separately, though much of the underlying data and substance of the experts’ opinions are overlapping.

1. *Dr. Salmon’s General Causation Opinion*

Dr. Salmon has 50 years of experience analyzing the carcinogenic effect of toxic exposure in humans, including 31 years working for what is now the OEHHA within the California EPA. He was retained in this case to provide an estimate of the excess cancer risk Melvin experienced due to his occupational exposure to diesel exhaust and its constituents, or diesel particulate matter (DPM), and whether this excess risk is more likely than not to have been a cause of his non-Hodgkin’s lymphoma. Dr. Salmon calculated that someone with Melvin’s DPM exposure from their employment with BNSF would have an estimated excess cancer risk between 2864 and 3875 excess cancers per million persons. He also opined that it was more likely than not that Melvin’s diesel exhaust exposure was a cause of his lymphoma.

In calculating Melvin’s excess cancer risk, Dr. Salmon relied on Melvin’s specific exposure information, air sampling data, air modeling studies involving railway workers, and the DPM inhalation cancer potency factor developed by OEHHA and the California Air Resources Board to estimate Melvin’s dose of diesel exhaust over the course of the relevant timeframe. In doing so, Dr. Salmon used standard methodology that does not appear to be the subject of dispute here.

What is in dispute is whether it was appropriate for Dr. Salmon to conclude that Melvin’s exposure was more likely than not a cause of his non-Hodgkin’s lymphoma, despite being unable to point to any specific study stating that exposure to diesel exhaust causes non-Hodgkin’s lymphoma. The trial court concluded that it was not, finding that the science Dr. Salmon relied on in reaching his opinion was “inadequate” because “[t]here is no data, no study, and no testing linking non-Hodgkin’s lymphoma and exposure to diesel exhaust.” The court further found that there was “too great an analytical gap between the data” on which Dr. Salmon did rely and the opinion he proffered. The trial court seems to have agreed with BNSF’s argument below that Gary’s experts, including Dr. Salmon, were not permitted to opine at trial that diesel exhaust and its constituents, more likely than not, are a cause of non-Hodgkin’s lymphoma, because there are no epidemiological or other scientific studies that have already stated that conclusion.

This ruling reflects a misunderstanding of the law. As Gary argues, there is no requirement that a causation expert rely on a specific study or other scientific publication expressing precisely the same conclusion at which the expert has arrived. (*Kennedy v. Collagen Corp*. (9th Cir. 1998) 161 F.3d 1226, 1229(*Kennedy*) [“it is scientifically permissible to reach a conclusion on causation without [epidemiological or animal] studies” showing a causal link]; *Wendell v. GlaxoSmithKline LLC* (9th Cir. 2017) 858 F.3d 1227, 1237 (*Wendell*) [“Perhaps in some cases there will be a plethora of peer reviewed evidence that specifically shows causation. However, such literature is not required in each and every case.”]; *Turner v. Iowa Fire Equipment Co.* (8th Cir. 2000) 229 F.3d 1202, 1208–1209 [“ ‘we do not believe that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness’ ”].)[[6]](#footnote-7)

This makes sense for several reasons. First, “[p]ublication . . . is not the *sine qua non* of admissibility; it does not necessarily correlate with reliability [citation], and in some instances well-grounded but innovative theories will not have been published. [Citation.] Some propositions, moreover, are too particular, too new, or of too limited interest to be published.” (*Daubert v. Merrell Dow Pharms., Inc.* (1993) 509 U.S. 579, 593 (*Daubert*); see also *Primiano v. Cook* (9th Cir. 2010) 598 F.3d 558, 565 [“Peer reviewed scientific literature may be unavailable because the issue may be too particular, new, or of insufficiently broad interest, to be in the literature.”].) As Dr. Salmon explained, this is such a case because few studies of the potential link between diesel exhaust and non-Hodgkin’s lymphoma have been conducted. “ ‘The first several victims of a new toxic tort should not be barred from having their day in court simply because the medical literature, which will eventually show the connection between the victims’ condition and the toxic substance, has not yet been completed.’ ” (*Wendell,* *supra*, 858 F.3d at p. 1237.)

Second, although “[e]pidemiology focuses on the question of general causation,” it “cannot prove causation; rather, causation is a judgment for epidemiologists and others interpreting the epidemiologic data.” (Green et al., *Reference Guide on Epidemiology*, in Reference Manual on Scientific Evidence (3d ed. 2011) 549, 552, 598.) Epidemiological studies merely identify *associations*, which do not equate to causation. (See *id*. at pp. 551–553.) It is up to the expert to “bridge the gap between association and causation” and make that informed judgment. (Kaye and Freedman, *Reference Guide on Statistics*, in Reference Manual on Scientific Evidence (3d ed. 2011) 211, 217–218 (Statistics); accord *Amador v. 3M Co.* (*In re Bair Hugger Forced Air Warming Devices Prods. Liab. Litig.*)(8th Cir. 2021) 9 F.4th 768, 778–780 [concluding it was not unreliable for an expert to rely on a study to draw an inference of causation even though the study found that the association did not establish causation, “[s]o long as the expert does the work ‘to bridge the gap between association and causation’ ”].)

“Whether an inference of causation based on an association is appropriate is a matter of informed judgment, not scientific methodology . . . .” (Rest.3d Torts, § 28 (2010) (Restatement), com. (c), subd. (3), p. 406; see also *id*. at subd. (1), p. 403 [“[A]n evaluation of data and scientific evidence to determine whether an inference of causation is appropriate requires judgment and interpretation.”]; *Milward v. Acuity Specialty Prods. Group, Inc.* (1st Cir. 2011) 639 F.3d 11, 18–19 (*Milward*) [same]; Statistics*,* *supra*, at p. 222 [“In the end, deciding whether associations are causal typically is not a matter of statistics alone, but also rests on scientific judgment.”].) And “scientific inference typically requires consideration of numerous findings, which, when considered alone, may not individually prove the contention. . . . In applying the scientific method, scientists do not review each scientific study individually for whether by itself it reliably supports the causal claim being advocated or opposed. Rather, as the Institute of Medicine and National Research Council noted, ‘summing, or synthesizing, data addressing different linkages [between kinds of data] forms a more complete causal evidence model and can provide the biological plausibility needed to establish the association’ being advocated or opposed.” (Berger, *The Admissibility of Expert Testimony*, in Reference Manual on Scientific Evidence (3d ed. 2011) 11, 19–20; see also *Milward*, at pp. 17–19 [discussing use of scientific judgment applying “weight of the evidence” approach for determining general causation].) It was therefore appropriate for Gary’s experts to use their experience and judgment to interpret the available epidemiological and other data they reviewed in reaching their causation opinions.

Finally, in many cases where the available scientific evidence is limited or inconclusive, there will inevitably be *some* analytical gap between the underlying data and the expert’s ultimate causation opinion. But *Sargon* should not be construed so broadly that the gatekeeper effectively supplants both the expert’s reasonable scientific judgment and the jury’s role. That would be at odds with *Sargon*’s emphasis on the limited role of the evidentiary gatekeeper. (*Sargon,* *supra*, 55 Cal.4th at p. 772.) In keeping the gate, it is not the trial court’s proper function to second-guess the judgment of a qualified expert who has provided a reasonable scientific explanation for his conclusions and used a scientifically accepted methodology for reaching them based on the available data, even if the data itself is inconclusive. “So long as an expert’s testimony rests upon ‘good grounds, based on what is known’ [citation], it should be tested by the adversarial process, rather than excluded for fear that jurors will not be able to handle the scientific complexities.” (*Milward,* *supra*, 639 F.3d at p. 15, internal quotation marks omitted.)

In reaching the opposite conclusion, the trial court took issue with Dr. Salmon’s reliance on the overall excess cancer risk and his opinion that such risk is relevant to determining the risk of non-Hodgkin’s lymphoma: “The Court notes in this paragraph in Dr. Salmon’s conclusion that he uses the word ‘cancer,’ not non-Hodgkin’s lymphoma. . . . [¶] . . . [¶] In this case, the decedent’s injury was non-Hodgkin’s lymphoma. Therefore, this Court is finding that the science relied upon by Dr. Salmon is inadequate.”

But Dr. Salmon explained that his overall cancer risk assessment was appropriate because it necessarily included an analysis of the risk of developing non-Hodgkin’s lymphoma—particularly given that “bone marrow (the cellular origin of Mr. Garner’s Diffuse Large B Cell Lymphoma) is a known human target site for both cancer and non-cancer effects of the of [*sic*] several components of diesel exhaust, including (but not limited to) benzene, dioxins, formaldehyde, butadiene and the polycyclic hydrocarbons, which are suspected significant contributors to DPM’s carcinogenic effect.” (Cf. *Milward,* *supra*, 639 F.3d at pp. 19–20 [expert relied on studies showing that benzene can cause “significant chromosomal damage at the stem cell level in the bone marrow”].) Dr. Salmon also testified at deposition that “for chemicals that induce tumors at multiple sites, the single-site approach may underestimate the true carcinogenic potential,” so “a statistical procedure may be used to estimate an overall potency”—a methodology taken directly from OEHHA. Dr. Salmon noted that diesel engine exhaust is classified as a known human carcinogen by the State of California and the International Agency for Research on Cancer (IARC). He further explained that the IARC has described “several studies in which elevated risks of leukemias, lymphomas and myelomas were found in workers exposed to diesel exhaust.” Dr. Salmon cited several scientific publications in support of his opinions.

Dr. Landolph agreed with Dr. Salmon, explaining in his report: “It is well-established in science that diesel exhaust and many of its chemical constituents act directly on DNA, causing mutations. Thus, diesel exhaust can be referred to as a mutagenic, multi-organ carcinogen, which consists of many mutagenic multi-organ carcinogens.” According to Dr. Landolph, “much epidemiological literature exists to show associations between diesel exhaust and its chemical constituents (such as PAHs like benzo(a)pyrene, and benzene, dioxin, and formaldehyde) and the development of cancer in multiple organ sites. Further, because diesel exhaust has been shown to be a mixture of mutagenic carcinogens, as just explained, occurrence of tumors in one site are relevant to development of tumors in other sites.” In support, Dr. Landolph cited multiple epidemiological and animal studies and explained that “strict concordance of target organs . . . in human epidemiological studies is not necessary in order to extrapolate the results of animal carcinogenicity studies to predict the carcinogenicity of chemicals to humans,” and that it “has become [a] common and accepted practice in [his] field of expertise to utilize the results of animal carcinogenicity studies to estimate the increase in cancer risk above background in humans that each dose of chemical carcinogen causes,” citing five sources for his opinion.

BNSF presented no evidence calling into question the scientific validity or reliability of any of this reasoning or methodology. It submitted no expert declarations or reports or scientific publications contradicting plaintiffs’ experts’ opinions or suggesting that Dr. Salmon’s use of the overall cancer risk assessment was improper or unreliable.[[7]](#footnote-8) Indeed, it failed to submit any legal or scientific authority in support of its argument—including any of the scientific materials on which Dr. Salmon relied and which the trial court, despite not having reviewed, found to be inadequate. Although BNSF complains in its briefing that excess cancer risk is a concept used by regulatory agencies, there is no categorical bar to a causation expert’s reliance on data of the type used by regulatory agencies. (*Davis v. Honeywell Internat. Inc.* (2016) 245 Cal.App.4th 477, 488–489.) In these circumstances, the trial court overstepped its limited role as an evidentiary gatekeeper by excluding Dr. Salmon’s expert opinions.

The error here is similar to the one committed by the district court in *Kennedy*. There, the plaintiff’s causation expert gave an opinion that her atypical systemic lupus erythematosus (SLE) was caused by Zyderm injections for facial wrinkles. (*Kennedy,* *supra*, 161 F.3d at pp. 1228–1229.) The expert “relied upon a wide variety of objective, verifiable evidence in forming his opinion that Zyderm *causes autoimmune disorders such as atypical SLE* . . . .” (*Id*. at p. 1228, italics added.) But the district court excluded his opinions because “no epidemiological or animal studies link[ed] Zyderm *to SLE or atypical SLE*.” (*Id.* at p. 1229, italics added.) In ruling that the district court “abused its discretion” by finding “too great an analytical gap . . . between the existing data and the expert’s conclusion,” the Ninth Circuit stated: “The fact that a cause-effect relationship *between Zyderm and lupus in particular* has not been conclusively established does not render [the expert]’s testimony inadmissible.” (*Id*. at p. 1230, italics added.) The expert “set forth the steps he took in arriving at his conclusion” and his “analogical reasoning was based on objective, verifiable evidence and scientific methodology of the kind traditionally used by rheumatologists.” (*Ibid*.) And the defendant had “not introduced any evidence that [the expert]’s reasoning [was] not scientifically valid.” (*Ibid.*) Accordingly, the Ninth Circuit concluded that the analytical “gap was of the district court’s making.” (*Ibid*.; see also *Milward,* *supra*, 639 F.3d at pp. 22–23 [similarly holding that “ ‘the gap was of the district court’s making’ ”].)

The same is true here. Dr. Salmon gave a reasonable scientific explanation for his causation opinions, including his reliance on the overall cancer risk, and he cited objective, verifiable evidence supporting his opinions. BNSF submitted no evidence that his reasoning or methodology was scientifically invalid. The trial court also found no fault with his methodology. The mere fact that a cause-effect relationship between exposure to diesel exhaust and non-Hodgkin’s lymphoma “in particular” has not been conclusively established in the scientific literature does not render Dr. Salmon’s opinions inadmissible. (*Kennedy,* *supra*, 161 F.3d at p. 1230; see also *Milward,* *supra*, 639 F.3d at pp. 16, 19–20 [causation expert properly relied on scientific evidence that benzene can cause acute myeloid leukemia (AML) “as a class” as support for his opinion that workplace exposure to benzene caused plaintiff’s specific rare type of AML].)

On this record, leaving adequate space for the exercise of reasonable scientific judgment based on the available data, we conclude that the analytical gap was not “too great” for Dr. Salmon to bridge using his own scientific training and expertise. (*Sargon,* *supra*, 55 Cal.4th at p. 771.) The trial court strayed beyond its gatekeeping role by weighing the probative value of Dr. Salmon’s opinion, and the studies on which he relied, rather than merely excluding a clearly invalid and unreliable expert opinion. (See *id.* at p. 772; *Cooper v. Takeda Pharmaceuticals America, Inc.* (2015) 239 Cal.App.4th 555, 592 (*Cooper*).)

BNSF contends on appeal that IARC must have considered all the same data Dr. Salmon relies on to opine that diesel exhaust can be a cause of non-Hodgkin’s lymphoma, but IARC Monograph 105 does not include non-Hodgkin’s lymphoma among the types of cancer that diesel exhaust has been demonstrated to cause. According to BNSF, Gary’s experts ignore this “critical” conclusion, and their opinions must therefore be unreliable.

We disagree. First, although trial counsel for BNSF read to the trial court a quote purporting to be from IARC Monograph 105, stating that for “lymphoma, the overall evidence did not support an effect of exposure to diesel exhaust and/or gasoline engine exhaust,” BNSF decided for strategic reasons not to submit the publication in support of its motion in limine.[[8]](#footnote-9) It also chose not to submit a respondent’s appendix on appeal or otherwise seek to include the publication in the appellate record. We find it difficult to see how we could conclude that IARC Monograph 105 requires exclusion of Gary’s experts given that the record contains only a one-page excerpt of the publication, and that excerpt does not include the statement on which BNSF relies. We cannot decide the appeal based on evidence not included in either the trial court or appellate record.

In any event, we are not persuaded by BNSF’s argument. Even if we assume that IARC Monograph 105 disclaims a link between diesel exhaust and lymphoma, we do not agree that it is inherently unreliable for an expert to infer causation from epidemiological studies simply because IARC or another agency has not yet done so.[[9]](#footnote-10) As we have explained, “[w]hether an inference of causation based on an association is appropriate is a matter of informed judgment, not scientific methodology, *as is a judgment whether a study that finds no association is exonerative or inconclusive*.” (Restatement, § 28, com. (c), subd. (3), p. 406, italics added.) “[I]n some cases, reasonable scientists can come to differing conclusions on whether a body of epidemiologic data justifies an inference of causation. Similarly, reasonable scientists may, in some instances, disagree on whether the absence of an association is exonerative of the agent or is merely inconclusive.” (*Ibid*.) Dr. Salmon was entitled to reach a different conclusion than that of IARC so long as it is not “ ‘clearly invalid and unreliable.’ ” (*Sargon,* *supra*, 55 Cal.4th at p. 772.)

Further, Dr. Salmon explained in his report that the “comment that epidemiology studies have not identified diesel exhaust as a risk factor for non-Hodgkin’s lymphomas is, like all such claims based on negative epidemiological evidence, unconvincing since few such studies have been undertaken, and those that were have relatively low power to detect such an effect. The great majority of substantial epidemiological work on diesel exhaust effects has concentrated on lung cancer, the most widely understood risk, and these studies excluded consideration of any other endpoint.” Dr. Salmon also relied on epidemiological literature, including other IARC volumes, showing associations between diesel exhaust’s chemical constituents and cancer development in multiple organ sites, which as we have explained, he is permitted to do.

BNSF relies primarily on *Sargon* and *Lockheed Litigation* *Cases* (2004) 115 Cal.App.4th 558 (*Lockheed*) to argue that the trial court properly excluded Dr. Salmon’s testimony as speculative. We find these cases factually distinguishable.

In *Sargon*, the manufacturer of a newly patented dental implant sued a university for breach of the parties’ contract for the university to perform clinical testing of the implant. (*Sargon,* *supra*, 55 Cal.4th at p. 753.) In support of its claim, the small company—whose annual net profits peaked at $101,000—presented expert testimony opining that it would have become a worldwide leader in the dental implant industry and earned profits ranging from $200 million to over $1 billion had the university not breached its contract. (*Id*. at pp. 753, 757.) The trial court excluded the expert’s opinion as speculative, and the Supreme Court affirmed that ruling. (*Id*. at p. 753.)

The excluded expert in *Sargon* was an accountant who based his lost profits calculation on a market share approach, by which he compared Sargon to six large, multinational dental implant companies (the “Big Six”) that were the dominant market leaders in the industry. (*Sargon,* *supra*, 55 Cal.4th at p. 756.) Unlike the other companies, however, Sargon “had no meaningful marketing or research and development organization and no parent company to assist it.” (*Id*. at p. 757.) In fact, the expert “admitted that by no objective business metric, such as sales or number of employees, was Sargon in fact comparable to the ‘Big Six’. Instead, he based his comparison solely on his belief that Sargon, like the ‘Big Six’, and unlike the rest, was innovative, and that innovation was the prime market driver.” (*Id*. at p. 777.) The expert also acknowledged that he had no expertise regarding how innovative Sargon’s dental implant was, and the trial court found he had no expertise in the dental implant industry at all. (*Id*. at pp. 759, 766.) The trial court therefore found, and the Supreme Court agreed, that the expert’s opinion was devoid of factual or logical basis and rested solely on “speculation and unreasonable assumptions.” (*Id*. at pp. 766–767, 781.)

Paraphrasing from the opinion of another appellate court, “[t]he nature and reliability of Dr. [Salmon]’s testimony in this case bears no resemblance to the expert testimony in *Sargon*. In *Sargon*, the expert had no reasonable basis for his opinion on lost profits, and reached his conclusions only by speculating and making readily discernible leaps of logic. The same cannot be said about Dr. [Salmon]’s testimony.” (*Cooper,* *supra*, 239 Cal.App.4th at p. 593.) Unlike the expert in *Sargon*, Dr. Salmon has over 50 years of experience in the precise subject matter about which he opined—the carcinogenic effect of toxic exposure in humans—and he reached his conclusions after using a well-established methodology and relying on various epidemiological studies. He and his colleagues at OEHHA formulated the same excess cancer risk methodology he used in this case, and he explained in detail why reliance on an all-cancer risk assessment is appropriate where, as here, the carcinogens are mutagenic, causing cancer throughout the body. Scientists utilizing this methodology take the available data from studies that have been conducted, which tend to focus on “the larger risk sites,” such as the lungs, and use it “to proximate the overall risk of cancer” to somebody exposed to the particular carcinogen. Dr. Salmon explained that the methodology is designed to “provide an estimate as the overall risk of cancer and it doesn’t specify that that risk be confined exclusively to the lung cancer site or other specific sites that have been measured and considered in the risk assessment.” It is therefore appropriate to rely, as Dr. Salmon did, on the epidemiological literature demonstrating associations between diesel exhaust and its chemical constituents and cancer development in multiple organ sites.

In *Lockheed*, another case on which BNSF relies, the plaintiffs’ expert opined that their exposure to five particular chemicals supplied by defendants was a substantial factor resulting in an increased risk of cancer. (*Lockheed,* *supra*, 115 Cal.App.4th at p. 564.) He based his opinion solely on a study finding that painters who were potentially exposed to more than 130 chemicals and other substances—including known carcinogens—contracted cancer at a higher rate than the general population. (*Id*. at pp. 564–565.) The trial court found that the expert’s opinion was “based on conjecture and speculation as to which of the many substances to which the study subjects were exposed contributed to the greater incidence of cancer,” and the Court of Appeal agreed. (*Id*. at p. 565.) Here, by contrast, Dr. Salmon relied on various studies showing associations between exposure to diesel exhaust and its chemical constituents and the development of cancer in multiple organ sites.

BNSF also submitted a notice of supplemental authority pointing to the recent opinion in *Onglyza Product Cases* (2023) 90 Cal.App.5th 776 (*Onglyza*), where the appellate court affirmed the trial court’s exclusion of the plaintiffs’ general causation expert, but we find that case distinguishable as well. There, as in *Lockheed*, the expert relied on a single study, and the trial court found that, as in *Sargon*, the expert’s opinions “went beyond [his] expertise and were not supported by a reliable methodology.” (*Onglyza*, at p. 782.) The court explained in detail why the expert’s application of the methodology he used was unreliable and concluded that his opinion contained “shifting results-based methodology that fails to logically and consistently weigh all relevant evidence.” (*Ibid*.) The trial court here made no such findings, and BNSF does not challenge Dr. Salmon’s methodology or expertise. We therefore conclude that the trial court erred in finding Dr. Salmon’s testimony inadmissible.[[10]](#footnote-11)

2. *Dr. Landolph’s General Causation Opinion*

Dr. Landolph is a molecular chemical carcinogenesis biologist professor with 50 years of experience in research, teaching, and scientific advisory and consultative activities involving the disciplines of chemistry, biochemistry, cell biology, cell and molecular toxicology, molecular biology, and molecular carcinogenesis, particularly as these disciplines relate to the study of chemical carcinogenesis and chemically induced morphological and neoplastic cell transformation. He was retained in this case to render a general causation opinion as to whether diesel exhaust and diesel exhaust constituents are carcinogenic in animals and/or humans, and whether these substances and chemicals can cause various types of tumors, including non-Hodgkin’s lymphoma. Dr. Landolph concluded that diesel exhaust and its constituents, particularly benzene, dioxin, and formaldehyde, “are capable of causing and/or contributing to the development [of] many different types of tumors in humans exposed to diesel exhaust and its components, including lung cancer and many other cancers, in particular Non-Hodgkin’s Lymphoma (NHL) and at least one of the NHLs, DBCL [diffuse large B cell lymphoma].”

The trial court granted BNSF’s motion to exclude Dr. Landolph’s opinion for “the same” reason it excluded Dr. Salmon’s opinions, finding that “[t]here is a gap in the analytical data to support the conclusions that diesel exhaust exposure is a causal link or a causal connection to non-Hodgkin’s lymphoma.” The court stated: “[I]n looking at the documents presented, it does not seem that there is enough data for the experts to draw their conclusion . . . . [T]here is an analytical gap that causes the Court to grant . . . the defense motion in limine Number 9 . . . excluding certain opinions of Dr. Landol[ph] on causation.”

BNSF contends that the trial court properly excluded Dr. Landolph’s testimony for two main reasons: (1) he, like Dr. Salmon, failed to cite a study concluding that diesel exhaust exposure causes non-Hodgkin’s lymphoma; and (2) he could not state with specificity what dose of diesel exhaust is required to cause non-Hodgkin’s lymphoma.

We disagree with BNSF’s first argument for the same reasons we have already explained in connection with Dr. Salmon’s opinions. Dr. Landolph was not required to identify a study conclusively stating that non-Hodgkin’s lymphoma is caused by diesel exhaust. Instead, it was appropriate for him to use his scientific judgment and expertise to evaluate the available data and determine whether to draw an inference of causation. (Restatement, § 28, com. (c), subd. (1).) In reaching his conclusions in this case, Dr. Landolph relied on decades of education and research, scientific data, numerous human epidemiological and animal studies showing positive associations, including specifically to non-Hodgkin’s lymphoma, other peer-reviewed relevant publications, and the conclusions of multiple agencies that diesel exhaust and its constituents are multi-system mutagenic carcinogens. He examined the data indicating a link between diesel exhaust and lung cancer and also analyzed studies showing associations with at least a dozen other sites, including two diesel exhaust studies finding elevated risks for leukemias and non-Hodgkin’s lymphoma. Dr. Landolph also relied on scientific literature containing similar data for the chemical constituents of diesel exhaust, including benzene, which he stated “is well known to induce many different types of leukemias and lymphomas,” including non-Hodgkin’s lymphoma. He explained that, “[t]aken together, the sum of the weight of all of the evidence” supports his conclusion to a reasonable degree of scientific probability that “diesel exhaust and its constituents . . . are capable of causing and/or contributing to the development many different types of tumors in humans exposed to diesel exhaust and its components, including . . . in particular Non-Hodgkin’s Lymphoma[.]” Absent any challenge to Dr. Landolph’s methodology, we conclude that he provided sufficient support for his scientific judgment on causation.

We further conclude that Gary’s experts, including Dr. Landolph, were not required to identify the exact dose of diesel exhaust at which point the exposure becomes toxic. The notion that there even exists such “a threshold dose before an effect can occur is a controversial concept for which current scientific thinking resists any universal answers and instead examines what is known about the pathological mechanisms of the disease.” (Restatement, § 28, reporter’s notes, com. (c), subd. (2), p. 437, citing Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960, 17993 (Apr. 23, 1996).) Dr. Landolph echoed this thought at his deposition, testifying that although some people insist there must be a threshold dose to be able to causally connect exposure to a particular chemical to increased risk of cancer, the existence of such a threshold has never been substantiated, and it “is still a current area of controversy today.” It is outside the scope of the trial court’s gatekeeping duty to resolve such scientific controversies. (*Sargon,* *supra*, 55 Cal.4th at pp. 772–773.) “The courts’ evidentiary gatekeeping function is . . . not a warrant for judicial intervention in genuine scientific debates over substantive principles.” (*People v. Superior Court* (*Vidal*) (2007) 40 Cal.4th 999, 1014.)

Dr. Landolph further testified that he agreed with the EPA that such dose-response curves are linear, meaning that there is no threshold dose of diesel exhaust necessary to cause non-Hodgkin’s lymphoma. Instead, Dr. Landolph explained, on a linear no-threshold dose response model, “basically any amount confers a risk,” and the “larger the amount, the larger the risk. So we don’t worry about trivial amounts.” (See also *Johnson & Johnson Talcum Powder Cases* (2019) 37 Cal.App.5th 292, 305 (*Talcum Powder Cases*) [a “dose-response pattern” means there is “increased risk with increased exposure”].)

BNSF presented no contrary evidence establishing that the existence of a threshold dose is required. “[W]hile precise information concerning the exposure necessary to cause specific harm to humans and exact details pertaining to the plaintiff’s exposure are beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial exposure and need not invariably provide the basis for an expert’s opinion on causation.” (*Westberry v. Gislaved Gummi AB* (4th Cir. 1999) 178 F.3d 257, 264; accord *Sarkees v. E.I. Dupont De Nemours & Co.* (2d Cir. 2021) 15 F.4th 584, 593; *Clausen v. M/V New Carissa* (9th Cir. 2003) 339 F.3d 1049, 1059–1060; *Hardyman v. Norfolk & W. Ry. Co.* (6th Cir. 2001) 243 F.3d 255, 265–266.)

We find it sufficient here that Dr. Salmon calculated Melvin’s dose according to a well-established methodology, explaining that the additional risk of cancer Melvin experienced as a result of his exposures was “substantial (between 2864 and 3875 per million).” This number was “well in excess of what would be considered a level requiring remedial action. 1 in a million is regarded as the *de minimis* level of cancer risk, whereas even more permissive programs considering cost-effectiveness as well as health effects generally regard 100 in a million as a level at which immediate remedial action would be required.” Dr. Landolph relied on these calculations and his opinion, based on the linear no-threshold dose response model, that the risk of developing cancer increases with any dose increase, to conclude that Melvin’s substantial exposure to diesel exhaust while working for BNSF—which was “at least 3,000 times higher than deminimus [*sic*]”—was more than trivial and led to a nontrivial elevation in his cancer risk.

Although other experts (and the jury) may disagree regarding whether there exists a threshold dose necessary to demonstrate a link between diesel exhaust and non-Hodgkin’s lymphoma, and whether Dr. Landolph has sufficiently shown that such a causal link even exists, BNSF’s arguments go to the weight and not the admissibility of those opinions. “Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence.” (*Daubert, supra*, 509 U.S. at p. 596.) We conclude that Dr. Landolph provided a reasonable basis for his opinions such that he should have been permitted to present them to the jury.

3. *Dr. Gale’s General and Specific Causation Opinions*

Dr. Gale is a physician specializing in hematology and oncology with substantial experience in statistics and epidemiology, and he has published over 1,200 scientific articles and more than 20 books, mostly on leukemia, transplantation, cancer biology, and immunology and radiation biology. He was retained in this case to opine whether occupational exposures to diesel engine exhaust and its constituents, including but not limited to benzene, dioxin, and formaldehyde were, to a reasonable degree of medical probability, a cause of Melvin’s non-Hodgkin’s lymphoma. Using the weight-of-the-evidence methodology, Dr. Gale concluded that diesel exhaust and its particulates cause cancer in humans, that they are a cause of non-Hodgkin’s lymphoma in humans, and that it is more likely than not, to a reasonable degree of medical probability, that Melvin’s occupational exposure to benzene, dioxin, and formaldehyde via his exposure to diesel exhaust was a cause of his non-Hodgkin’s lymphoma.

As with Dr. Landolph, the trial court declined to provide detailed reasoning for its decision to exclude Dr. Gale, stating that “in looking at the documents presented, it does not seem that there is enough data for the experts to draw their conclusion much less the Court’s [*sic*] . . . there is an analytical gap that causes the Court to grant the motions in limine to preclude . . . the opinion of Dr. Gale on liability and causation[.]” BNSF contends on appeal that Dr. Gale’s specific causation opinion was properly excluded both because there was no admissible general causation opinion on which to base his opinion and because, like Dr. Landolph, he could not identify a specific dose of diesel exhaust that causes non-Hodgkin’s lymphoma.

We are not persuaded by BNSF’s arguments for the reasons we have already explained. Because we have concluded that the experts’ general causation opinions are admissible, Dr. Gale may properly base his specific causation on those opinions. Dr. Gale also explained that “[d]etermination of whether a substance or chemical causes cancer in humans is not tissue or organ specific.” In other words, he agreed with Dr. Salmon and Dr. Landolph that analysis of cancer at one organ site is relevant to analysis of cancers at other sites. There is nothing in the record that would allow us to reject this as a scientifically invalid or unreasonable opinion.

Dr. Gale further explained that “[s]everal studies report an association between exposure to diesel engine exhaust and particulates and lymphomas.” Further, “[c]onsiderable data in experimental animals indicate exposure to diesel engine exhaust and particulates cause lymphomas,” and two IARC Monographs “based on considerable additional epidemiological data” found that “[m]ost studies show a positive association between benzene exposure and NHL [non-Hodgkin’s lymphoma].” Dr. Gale similarly analyzed the data on dioxin and formaldehyde, which both showed positive associations with non-Hodgkin’s lymphoma. Based on his extensive review of the data, Dr. Gale opined that “diesel engine exhaust and particulates, benzene, dioxin and formaldehyde are a cause of non-Hodgkin lymphoma in humans including diffuse large B-cell lymphoma.”

Dr. Gale also testified at deposition that there is no “magical dose” at which point it becomes possible for diesel exhaust to cause non-Hodgkin’s lymphoma. Rather, he explained that for him “to opine that it is more likely than not, to a reasonable degree of medical probability, that an exposure to [diesel] exhaust and particulates was a substantial contributing factor or a cause of [Melvin] developing Non-Hodgkin’s lymphoma, it’s a dose at which the calculated likelihood of developing cancer is more than trivial.” Here, Dr. Gale explained, Melvin’s exposure was sufficiently high to result in a nontrivial and indeed substantial excess cancer risk. He performed a differential diagnosis—a widely accepted method explained in the Reference Manual on Scientific Evidence from the Federal Judicial Center that considers other exposures and variables in determining specific causation—and properly used his expertise and scientific judgment to opine that it is more likely than not, to a reasonable degree of medical probability, that Melvin’s diesel exhaust exposure was a cause of his non-Hodgkin’s lymphoma. (See *Talcum Powder Cases,* *supra*, 37 Cal.App.5th at pp. 327–332 [trial court erred by rejecting expert’s differential diagnosis for specific causation opinion].) Yet again, BNSF does not challenge Dr. Gale’s methodology in formulating his general and specific causation opinions.

While BNSF may argue there are weaknesses in the data relied upon by Gary’s experts or their reasoning, their opinions were not clearly invalid or unreliable. We therefore reverse the trial court’s orders excluding the causation opinions of Dr. Salmon, Dr. Landolph, and Dr. Gale. We emphasize, however, that our decision is a narrow one—we draw no conclusions regarding the probative value or persuasiveness of their testimony. It will be up to the jury to decide whether to accept their opinions that it is more likely than not that exposure to diesel exhaust can and did cause Melvin’s non-Hodgkin’s lymphoma.

DISPOSITION

The judgment is reversed. The matter is remanded with directions to the trial court to vacate its orders granting BNSF’s motions in limine to exclude the expert opinions of Dr. Salmon, Dr. Landolph, and Dr. Gale, issue new orders denying those motions, and conduct further proceedings consistent with this opinion. Gary is entitled to his costs on appeal.

BUCHANAN, J.

WE CONCUR:

McCONNELL, P. J.

DO, J.

1. We refer to Gary and his father Melvin Garner by their first names to avoid confusion. [↑](#footnote-ref-2)
2. All undesignated statutory references are to the Evidence Code. [↑](#footnote-ref-3)
3. “General causation” refers to whether a substance is capable of causing a particular injury or condition in the general population. “Specific causation” refers to whether the substance caused a particular individual’s injury or condition. (*Knight v. Kirby Inland Marine Inc.* (5th Cir. 2007) 482 F.3d 347, 351.) [↑](#footnote-ref-4)
4. According to Merriam-Webster Online Dictionary, “mutagenic” is defined as “inducing or capable of inducing genetic mutation.” (Merriam-Webster Online Dictionary (2023) <https://www.merriam-webster.com/medical/mutagenic> [as of Dec. 26, 2023], archived at <https://perma.cc/D657-VQ5V>.) In his expert report, Dr. Landolph describes a “mutagenic” constituent as one that “act[s] directly on DNA, causing mutations.” Dr. Salmon defines the term in his expert report as meaning “affecting DNA.” [↑](#footnote-ref-5)
5. As we will explain, the trial court’s ruling was also based on a misunderstanding of the law under *Sargon*. We would thus find reversible error even applying the abuse of discretion standard. (*Hernandez v. Amcord, Inc.* (2013) 215 Cal.App.4th 659, 678 [“evidentiary rulings which are based on a misunderstanding of the law are an abuse of discretion”].) [↑](#footnote-ref-6)
6. Because *Sargon* took its “analytical gap” language from the United States Supreme Court’s decision in *Joiner* (*Sargon,* *supra*, 55 Cal.4th at p. 771), we may consider post-*Joiner* federal authorities on the issue for their persuasive value. [↑](#footnote-ref-7)
7. We note, however, that even if BNSF had submitted studies or expert testimony conflicting with Dr. Salmon’s opinion, that alone would not necessarily justify its exclusion. Rather, it would more likely demonstrate that the use of the overall cancer risk and reliance on certain studies to show an increased risk of non-Hodgkin’s lymphoma is subject to debate. It is not the trial court’s role to resolve such scientific controversies. (*Sargon,* *supra*, 55 Cal.4th at pp. 772–773.) [↑](#footnote-ref-8)
8. Counsel for BNSF stated that she had “wanted to save it for cross-examination” of Gary’s experts at trial. [↑](#footnote-ref-9)
9. We also note that Dr. Gale cited IARC Monograph 105 on diesel exhaust in his report as specifically acknowledging that “[i]dentification of a specific organ or tissue does not preclude the possibility that this agent may cause cancer at other sites.” [↑](#footnote-ref-10)
10. BNSF also moved to exclude Dr. Salmon’s specific causation opinion, but on appeal, it does not challenge any specific aspect of this opinion. Instead, BNSF argues only that once the trial court excluded the general causation opinions offered by Dr. Salmon and Dr. Landolph, its exclusion of all specific causation opinions followed as a matter of course. The trial court did not specifically address or reject Dr. Salmon’s specific causation opinion. For those reasons, and because we have concluded that Dr. Salmon’s general causation opinion is admissible, we also reverse the trial court’s ruling as to Dr. Salmon’s specific causation opinion. [↑](#footnote-ref-11)