

IN THE SUPREME COURT OF THE STATE OF NEW MEXICO

Opinion Number: 2016-NMSC-012

Filing Date: March 3, 2016

Docket No. S-1-SC-33884

CONCEPTION AND ROSARIO ACOSTA et al.,

Plaintiffs/Intervenors-Petitioners,

v.

**SHELL WESTERN EXPLORATION AND
PRODUCTION, INC., and SHELL OIL COMPANY,**

Defendants-Respondents.

**ORIGINAL PROCEEDING ON CERTIORARI
Freddie Joseph Romero, District Judge**

Tucker Law Firm, P.C.
Steven L. Tucker
Santa Fe, NM

Law Offices of Martin N. Buchanan
Martin N. Buchanan
San Diego, CA

for Petitioners

Rodey, Dickason, Sloan, Akin & Robb, P.A.
Edward R. Ricco
Jocelyn C. Drennan
Albuquerque, NM

for Respondents

OPINION

DANIELS, Justice.

I. INTRODUCTION

{1} From the 1920s through 1993, Defendants Shell Western Exploration and Production, Inc. and Shell Oil Company (collectively, Shell) engaged in oil and gas operations in Hobbs, New Mexico. Environmental contamination from these operations was discovered years later, and over two hundred residents of the contaminated area brought this toxic tort action against Shell for personal injury damages that included systemic lupus erythematosus (lupus) and other autoimmune disorders. Plaintiffs allege that toxic chemicals from crude oil caused their autoimmune disorders. They challenge the district court's exclusion of the scientific evidence and expert testimony they offered in support of this theory, and they challenge the resulting partial summary judgment in favor of Shell.

{2} We hold that the district court applied an incorrect standard of admissibility in its evidentiary rulings and that Plaintiffs' causation evidence should have been admitted. Because summary judgment as to Shell's culpability for the autoimmune disorders was granted to Shell as a result of this improper exclusion of evidence, we reverse the summary judgment and remand to the district court for further proceedings.

II. BACKGROUND

{3} Plaintiffs are residents of the Westgate subdivision in Hobbs. Westgate was built in the late 1970s on and near an unlined storage pit where, during its oil drilling operations, Shell had placed toxic hydrocarbons in direct contact with the earth from the 1940s until Shell covered the pit with "fill dirt" during the 1960s. Shell did not conduct any environmental risk assessment of the pit while it was in operation or after it was covered. Shell never reported releases or leaks of toxic chemicals to the New Mexico Oil Conservation Division and did not notify the Westgate subdivision builder that the hydrocarbon storage pit existed beneath Tasker Drive in the new subdivision.

{4} Shell had conducted oil and gas operations on the Grimes lease. The operations included storing crude oil in the Grimes tank battery, located just west of the Tasker pit. Shell decommissioned the Grimes tank battery in 1993 and turned the Grimes lease over to Altura Energy, Ltd. by 1997. Massive hydrocarbon contamination of the soil, extending sixty-five feet below ground level and into the aquifer, was discovered while dismantling the Grimes tank battery in 1997. Later that year, home builders in Westgate discovered a hard layer of hydrocarbon contaminants one to two feet below the ground surface that varied in thickness from several inches to several feet. Below that layer was oily soil saturated with toxic hydrocarbons. The contamination extended across properties on both sides of Tasker Drive. Parts of the area remained contaminated at the time of trial, despite years of attempted remediation involving removal of hundreds of truckloads of contaminated earth, massive tents, the closure of part of Tasker Drive, noise, foul smells, and dusty air.

{5} Plaintiffs asserted claims against Shell for negligence, strict liability, nuisance, and trespass and alleged that they had suffered injuries from their exposure to contamination from Shell's oil operations. The alleged injuries included lupus and other autoimmune disorders, neurological diseases, and respiratory diseases. Only the claims involving

autoimmune disorders are at issue in this appeal.

{6} Plaintiffs sought to offer the expert testimony of Dr. James Dahlgren that Plaintiffs' lupus and other autoimmune disorders were caused or aggravated by long-term exposure to a mixture of benzene and other organic solvents, hydrocarbons such as pristane and phytane, and mercury (the agents), all of which are toxic chemicals found in crude oil. As support for this causation opinion, Dahlgren provided numerous animal and human studies that linked the agents to immune system disruption, autoimmune diseases, and lupus.

{7} Lupus is a complex and potentially fatal inflammatory disorder that can affect various parts of the body, including the joints, skin, kidneys, heart, lungs, blood vessels, and brain. Physicians diagnose lupus when a patient presents with at least four of the American College of Rheumatology's "Eleven Criteria of Lupus." These criteria include rashes, ulcers, arthritis, and immunological disorders.

{8} Humans and mice with lupus also demonstrate a deficit of natural killer cells (NKC) that play a critical role in the normal functioning of a healthy immune system. Dahlgren proffered his own published and peer-reviewed epidemiological study that quantified Plaintiffs' exposure to the agents, gathered Plaintiffs' medical histories, studied their medical conditions, compared these results with those of a control group in a nonexposed community, and discussed a number of toxicological studies on mice that associated autoimmune disorders with exposure to pristane (the animal studies).

{9} Dahlgren's study obtained medical records and questionnaire responses from all Plaintiffs, samples of blood from a volunteer subgroup of Plaintiffs, samples of house dust from some of their Westgate homes, and analysis results of the air monitored by Shell at multiple locations in the exposed Westgate neighborhood. The sample analysis results were used to measure the presence of the agents in the subjects' environment and in the volunteer subjects themselves. As compared to the analysis results of equivalent samples obtained from volunteers in the control community and from their surroundings, Dahlgren measured elevated levels of pristane and phytane in Plaintiffs' blood samples and elevated levels of mercury, pristane and phytane, benzene, and other hydrocarbons in Plaintiffs' home environments.

{10} Dahlgren also compiled the medical histories and physical examination results from a "volunteer sample of 90 adult[]" Plaintiffs of the Westgate subgroup and from members of the nonexposed control community to compare the occurrence of disease and symptoms in the two communities. He observed an "[i]ncreased prevalence[]" of symptoms thought to be predictive of autoimmune disorder" in Plaintiffs' community. The NKC were significantly fewer in the Westgate residents than in residents of the control community while the presence of B-lymphocytes was significantly greater in the Westgate population. The deficit of NKC in combination with elevated B-lymphocytes indicated abnormalities in Plaintiffs' immune systems.

{11} Dahlgren’s study of Plaintiffs “by questionnaire and medical record review” identified thirteen diagnosed cases of lupus on two blocks in the area of the Westgate neighborhood on or near the Tasker pit. In the Westgate community, diagnoses of lupus and rheumatic disease, another autoimmune disorder, were ten times those in the control community. Plaintiffs “got the diseases . . . after moving into the neighborhood,” and many of them reported a lessening or even a total remission of the symptoms after leaving the Westgate neighborhood. To explain the prevalence of these diseases in Westgate compared to the unexposed control community, Dahlgren concluded that the agents caused the statistically significant elevation in occurrences of autoimmune disorders.

{12} Dahlgren’s study acknowledged that it could not point definitively to any human cases of pristane-induced lupus, but it referred to a number of other studies demonstrating that mercury is “conclusively known” to cause immune system disruption in animals and humans and that benzene adversely impacts the immune system in humans. Dahlgren also referred to at least thirteen animal studies establishing that pristane exposure in mice induces autoimmunity and lupus, one study stating that pristane could be involved in some cases of human lupus, and one study indicating that phytane would have a similar effect due to its comparable molecular structure and toxicity.

{13} In his affidavit, Dahlgren asserted that his study was conducted pursuant to the methodological standards set by the Federal Judicial Center’s *Reference Manual on Scientific Evidence* (2d ed. 2000). Dahlgren’s study included (1) an analysis of Plaintiffs’ medical conditions through patient history, medical records, physical examination, and diagnostic testing, (2) an analysis of exposure information and the temporal relationship between exposure and illness, (3) a review of the medical and scientific evidence to determine whether the exposure can cause the illness (“general causation”), and (4) an application of the general knowledge to the specific circumstances of the case to determine whether the exposure did cause the illness, including consideration of other possible causes (“specific causation”).

{14} Dahlgren presented evidence that extrapolation of the results of the animal studies referenced in his study provided the scientifically valid basis for his causation opinion in this case. He determined that the dose of pristane Plaintiffs received from prolonged household exposure, adjusted for the weight of a human, was within the range of the potentially “harmful dose” determined in the animal studies. He discussed several other studies of oilfield outbreaks of autoimmune disease, one of which cited a rate of lupus that was well above the national rate but only one eighth the rate of lupus found in Westgate.

{15} Dahlgren also ruled out several possible alternative explanations for the elevated occurrence of autoimmune disorders in Westgate, such as genetic susceptibility or drug-induced autoimmunity. He then hypothesized that the agents found in the Westgate community, including pristane, phytane, benzene, and mercury, have a synergistic effect and are more toxic in combination than their additive, comparable, individual toxicities. He cited the *Reference Guide on Toxicology* and the Environmental Protection Agency guidelines

regarding the concerted effect of mixtures of toxic agents on the body. See Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, in Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* 401, 429, 436 (2d ed. 2000); Guidelines for the Health Risk Assessment of Chemical Mixtures, 51 Fed. Reg. 34014, 34014-34022 (Sept. 24, 1986). Even Shell's expert agreed that it is important for an expert to consider how multiple chemicals interact and to use scientific judgment to determine whether they interact.

{16} Dahlgren considered all of this evidence and more before concluding that Plaintiffs' inhalation, ingestion, and absorption of the combination of various toxins from Shell's oil and gas operations caused or aggravated their lupus and other autoimmune disorders.

{17} The district court excluded Dahlgren's study of blood pristane data and his determination of Plaintiffs' cumulative dose from prolonged exposure as unreliable, and Plaintiffs did not challenge those rulings on appeal. But the district court also excluded the animal studies and Dahlgren's study itself as not relevant, reasoning they failed to show general causation between the mixture of identified chemicals and lupus. Contrary to Dahlgren's affidavit, the district court found that he had not considered the dose-response relationship necessary for inferring causation in humans from toxicological results in animals and stated that the animal studies were "so dissimilar to the facts presented [t]herein that the [district c]ourt d[id] not find them relevant." It concluded that Dahlgren's study had "limited value as a basis for a causal connection opinion" because it was a "hypothetical" study "admittedly done only for comparison purposes" and therefore that it had failed to "bridge the gap from association to causation" and could not stand alone to support Plaintiffs' claims of a causal relationship between the alleged mixture and lupus. As a result, the district court excluded Dahlgren's causation opinion due to "insufficient" evidentiary support and, based on the consequential lack of causation evidence, granted Shell's motion for summary judgment on Plaintiffs' claims for damages from lupus and other autoimmune disorders.

{18} Plaintiffs appealed the grant of summary judgment, challenging the district court's exclusions of the animal studies and Dahlgren's study based on relevance and its exclusion of Dahlgren's causation opinion for "insufficient" evidence. *Acosta v. Shell W. Expl. & Prod., Inc.*, 2013-NMCA-009, ¶¶ 1, 12, 293 P.3d 917. The Court of Appeals affirmed the district court's grant of summary judgment, holding that the district court did not abuse its discretion when it concluded that Dahlgren's study and the animal studies were "not sufficient" in either bridging the analytical gap from association to causation or in establishing causation between the agents and Plaintiffs' lupus and autoimmune disorders. *Id.* ¶¶ 27, 30, 33.

{19} The Court of Appeals interpreted the district court's evidentiary ruling that Dahlgren's study did not "fit" the case as an issue of relevance. See *id.* ¶¶ 20, 28 (stating that "the district court specifically chose not to address whether Dr. Dahlgren's methodology was reliable" and instead "excluded Dr. Dahlgren's epidemiologic study and his opinion testimony on the basis of relevance"). The Court of Appeals agreed with the district court, stating that "Plaintiffs could not articulate any study, other than Dr. Dahlgren's own study

of the Westgate population, that would support the opinion that the petrochemical mixture caused the lupus and other autoimmune disorders identified in this case” and that “Dr. Dahlgren’s study is only relevant to show a generally higher incidence of certain medical disorders in two community groups but is insufficient to establish a general causation link to lupus and other autoimmune disorders.” *Id.* ¶¶ 26, 32. We granted certiorari and now reverse the Court of Appeals and the district court. *See Acosta v. Shell W.*, 2012-NMCERT-012.

III. STANDARD OF REVIEW

{20} Generally, the admission or exclusion of evidence is reviewed on appeal for abuse of discretion. *State v. Downey*, 2008-NMSC-061, ¶ 24, 145 N.M. 232, 195 P.3d 1244. But in this case, the threshold question is whether the district court applied the correct evidentiary standard, a legal question we review de novo. *State v. Torres*, 1999-NMSC-010, ¶ 28, 127 N.M. 20, 976 P.2d 20. We also review de novo the related inquiry of whether the district court properly granted Shell’s motion for partial summary judgment. *See Smith v. Durden*, 2012-NMSC-010, ¶ 5, 276 P.3d 943 (“We view the facts in the light most favorable to the party opposing the summary judgment and indulge all reasonable inferences in favor of a trial on the merits.”). Summary judgment is appropriate only where “there is no genuine issue as to any material fact and . . . the moving party is entitled to a judgment as a matter of law.” Rule 1-056(C) NMRA.

IV. DISCUSSION

{21} “Admissibility is a minimal standard for individual items of evidence, including expert opinions.” *State v. Consaul*, 2014-NMSC-030, ¶ 67, 332 P.3d 850. “[T]o be admissible an item of evidence need only add something to the debate; it need only have a ‘tendency to make a fact more or less probable than it would be without the evidence.’” *Id.* (quoting Rule 11-401 NMRA).

A. The Admissibility of Expert Testimony Under New Mexico Law

{22} The admissibility of expert testimony in New Mexico is guided by Rule 11-702 NMRA, which sets out three requirements: “(1) that the expert be qualified; (2) that the testimony be of assistance to the trier of fact; and (3) that the expert’s testimony be about scientific, technical, or other specialized knowledge with a reliable basis.” *Downey*, 2008-NMSC-061, ¶ 25.

{23} The district court found that Dahlgren was qualified to provide opinions regarding specific and general causation in this case. Further, the district court “[d]isregard[ed] the questions of invalid data, unreliable methodology and the other issues raised by Shell,” focusing instead on the second admissibility requirement of whether Dahlgren’s testimony would assist the trier of fact. This requirement “goes primarily to relevance” as “[e]xpert testimony which does not relate to any issue in the case is not relevant and, ergo, non-

helpful.” *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 591 (1993) (internal quotation marks and citation omitted).

{24} Shortly after *Daubert*, this Court stated that the “pertinent inquiry” for determining whether expert testimony will assist the trier of fact under the second requirement of Rule 11-702 “must focus on the proof of reliability of the scientific technique or method upon which the expert testimony is premised.” *State v. Alberico*, 1993-NMSC-047, ¶ 53, 116 N.M. 156, 861 P.2d 192. A court must determine whether the proffered expert testimony is “sufficiently tied to the facts of the case that it will aid the jury in resolving a factual dispute.” *Downey*, 2008-NMSC-061, ¶ 30 (internal quotation marks and citation omitted). “[T]he scientific methodology [must] ‘fit[]’ the facts of the case and thereby prove[] what it purports to prove.” *Id.* The inquiry is “a flexible one,” and its focus “must be solely on principles and methodology, not on the conclusions that they generate.” *Daubert*, 509 U.S. at 594-95; see also *Alberico*, 1993-NMSC-047, ¶ 72 (“There is no requirement that a scientific technique or method prove conclusively what it purports to prove.”). As the United States Supreme Court emphasized, “it would be unreasonable to conclude that the subject of scientific testimony must be ‘known’ to a certainty; arguably, there are no certainties in science.” *Daubert*, 509 U.S. at 590. Rather, “the scientific procedure which supports the testimony [must be] capable of supporting opinions based upon a reasonable probability rather than conjecture.” *Alberico*, 1993-NMSC-047, ¶ 98 (internal quotation marks and citation omitted).

{25} Since *Daubert*, the federal courts have continued to refine the standards for admissibility of expert testimony, but New Mexico has not followed these changes in lockstep with the federal courts. In *Kumho Tire Co. v. Carmichael*, the United States Supreme Court extended a trial court’s gate-keeping function under *Daubert* to include expert testimony based on nonscientific experience and training. See *Kumho Tire Co.*, 526 U.S. 137, 151 (1999). Yet, in *Torres*, this Court limited application of the *Daubert/Alberico* requirements to expert testimony that requires scientific knowledge. See *Torres*, 1999-NMSC-010, ¶ 43. Accordingly, committee commentary on Rule 11-702 warns of the differences between federal and New Mexico law in applying the *Daubert* requirements. See Rule 11-702 comm. cmt. (“New Mexico has not adopted the changes made to the federal rule in 2000 to incorporate the requirements of *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), in light of the differences between federal law and New Mexico law regarding whether *Daubert* applies to nonscientific testimony.”); see also *Banks v. IMC Kalium Carlsbad Potash Co.*, 2003-NMSC-026, ¶ 19, 134 N.M. 421, 77 P.3d 1014 (observing that “after *Kumho Tire Co.*, we apply *Daubert* somewhat differently than do the federal courts”).

{26} In *General Electric Co. v. Joiner*, the United States Supreme Court affirmed a district court’s determination to exclude an expert’s causation testimony on grounds of relevance, noting that “[a] court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.” 522 U.S. 136, 146 (1997). In the case at bar, both the district court and the Court of Appeals cited this *Joiner* proposition as a key reason for

excluding Dahlgren’s general causation testimony pertaining to Plaintiffs’ development of lupus and other autoimmune conditions and determined that the analytical gap between the evidence presented and the inference drawn on the ultimate issue of causation was too wide. *See Acosta*, 2013-NMCA-009, ¶¶ 23-24, 27.

{27} But New Mexico has never adopted the *Joiner* rule that a judge may reject expert testimony where the “analytical gap” between the underlying evidence and the expert’s conclusions is “too great,” *see* 522 U.S. 146, and we refuse to do so in this case. Historically, this Court has placed great value on allowing a jury to hear evidence and decide a case on the merits. *See, e.g., Zamora v. St. Vincent Hosp.*, 2014-NMSC-035, ¶¶ 10-11, 335 P.3d 1243 (stating that this Court has maintained New Mexico’s notice pleading requirements based on our “policy of avoiding insistence on hypertechnical form and exacting language” and on a rationale of resolving disputes on their merits); *Romero v. Philip Morris Inc.*, 2010-NMSC-035, ¶ 8, 148 N.M. 713, 242 P.3d 280 (“New Mexico courts, unlike federal courts, view summary judgment with disfavor, preferring a trial on the merits.”).

{28} *Joiner* is inconsistent with longstanding New Mexico law that leaves credibility determinations and weighing of the evidence to the trier of fact. *See, e.g., State v. Hughey*, 2007-NMSC-036, ¶¶ 15, 17, 142 N.M. 83, 163 P.3d 470 (concluding that evidentiary rulings based on witness credibility usurp the role of the jury and that doubt as to scientific conclusions should be resolved not by exclusion but by cross-examination, rebuttal evidence, and argumentation). “Given the capabilities of jurors and the liberal thrust of the rules of evidence, we believe any doubt regarding the admissibility of scientific evidence should be resolved in favor of admission, rather than exclusion.” *Lee v. Martinez*, 2004-NMSC-027, ¶ 16, 136 N.M. 166, 96 P.3d 291. “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.” *Id.* ¶ 48 (quoting *Daubert*, 509 U.S. at 596).

B. Proof of Causation in Toxic Tort Litigation

{29} Although litigation of toxic torts is often complex, this does not alter the district court’s gatekeeping function. The crucial issue in this case is causation. To prove causation in a toxic tort case, a plaintiff must first show that a suspected cause actually “is capable of causing a particular injury or condition in the general population.” *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005). After establishing such general causation, the plaintiff must then demonstrate specific causation: that the suspected cause did actually cause the plaintiff’s injury. *Id.* Because the district court determined that Dahlgren’s study and the proffered testimony would not assist the trier of fact in determining whether the chemical mixture at issue in this case is capable of causing lupus or other autoimmune disorders, it never reached the question of specific causation.

{30} “When the connection between an agent and disease is strong and well documented, general-causation issues fade into the background.” Restatement (Third) of Torts: Physical

and Emotional Harm § 28 cmt. c(3) (Am. Law Inst. 2010). But demonstrating that a chemical is capable of causing a particular injury in the general population is often difficult in first-exposure cases where it has not been the subject of extensive scientific analysis.

{31} We agree with other jurisdictions that “[t]he first several victims of a new toxic tort should not be barred from having their day in court” simply because scientific analysis on a particular chemical cause has not yet been fully developed. *Turner v. Iowa Fire Equip. Co.*, 229 F.3d 1202, 1209 (8th Cir. 2000); *see also Best v. Lowe’s Home Ctrs., Inc.*, 563 F.3d 171, 180-81 (6th Cir. 2009) (“[T]here is no requirement 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.” (internal quotation marks and citation omitted)); *Norris*, 397 F.3d at 882 (“In cases where there is no epidemiology challenging causation available, epidemiological evidence would not necessarily be required.”).

{32} In some cases where epidemiology is absent, jurists and scientists have employed the nonformulaic guidelines of Sir Austin Bradford Hill to assess whether an epidemiological study’s finding of an association between a substance and an injury supported an inference of causation. Michael D. Green, D. Michal Freedman, & Leon Gordis, *Reference Guide on Epidemiology*, in Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* 599-600 & 599 n.141 (3d ed. 2011). The Bradford Hill factors that guide experts in making judgments about causation include: (1) temporal relationship, (2) strength of the association, (3) dose-response relationship, (4) replication of the findings, (5) biological plausibility (coherence with existing knowledge), (6) consideration of alternative explanations, (7) cessation of exposure, (8) specificity of the association, and (9) consistency with other knowledge. *Id.* at 600. These criteria are nonexclusive, and no one factor is dispositive in the general causation inquiry. *See id.*

{33} In essence, the Bradford Hill factors measure the ability of an epidemiological study to determine whether an association found by the study is sufficient to satisfy an ultimate question of fact regarding causation or whether the association is merely spurious. *See Milward v. Acuity Speciality Prods. Grp., Inc.*, 639 F.3d 11, 17 (1st Cir. 2011) (describing the Bradford Hill factors as a scientific method of weighing the evidence to make causal determinations, involving “a mode of logical reasoning often described as ‘inference to the best explanation.’” (citation omitted)); Green et al., *supra* at 598 (“[E]pidemiology cannot prove causation; rather, causation is a judgment for epidemiologists and others interpreting the epidemiologic data.”). As the First Circuit noted, “[i]n this mode of reasoning, the use of scientific judgment is necessary,” and “[t]he fact that the role of judgment in [this] approach is more readily apparent than it is in other methodologies does not mean the approach is any less scientific.” *Milward*, 639 F.3d at 18.

{34} While the relevance of a particular methodology depends on the relationship of the methodology to the facts and circumstances of each case, *see Downey*, 2008-NMSC-061, ¶ 30, application of the Bradford Hill factors in assessing general causation is widely accepted. *See, e.g.,* Green et al., *supra* at 598-606 (describing application of the Bradford Hill criteria

in determining whether an established association between exposure to an agent and development of a disease is causal); Restatement (Third) of Torts: Physical and Emotional Harm § 28 cmt. c(3) (Am. Law Inst. 2010) (describing the Bradford Hill criteria for assessing whether an association is causal in an analysis of general causation); Ian S. Spechler, *Physicians at the Gates of Daubert: A Look at the Admissibility of Differential Diagnosis Testimony to Show External Causation in Toxic Tort Litigation*, 26 Rev. Litig. 739, 742 (2007) (“General causation testimony should rely on hard science, and courts should evaluate the reliability of that science under the Bradford Hill criteria.”).

C. Dahlgren’s Testimony and the Studies He Relied on Were Probative of Causation and Should Have Been Admitted

{35} Dahlgren’s causation opinion, his study, and the animal studies it relied on are relevant and admissible if they demonstrate a valid scientific relationship that is probative of causation, regardless of their sufficiency to sustain Plaintiffs’ entire burden of proof. See *Ambrosini v. Labarraque*, 101 F.3d 129, 135 (D.C. Cir. 1996) (“The dispositive question [concerning relevance] is whether the testimony will assist the trier of fact to understand the evidence or to determine a fact in issue, not whether the testimony satisfies the plaintiff’s burden on the ultimate issue at trial.” (internal quotation marks and citation omitted)).

{36} Relying on *Joiner*, the district court excluded the animal studies because it found them “not . . . relevant” as “studies . . . so dissimilar to the facts” associated with Plaintiffs’ exposures, and it emphasized the need to consider the “dose-response relationship” when “inferring human causation from animal studies.” The district court concluded that in the absence of such “scientifically reliable evidence . . . to support general causation between the [agents] and lupus . . . , the study does not ‘fit’ any issue to be presented to the jury, and is inadmissible.”

{37} The science of toxicology provides evidence that a certain dose of a particular chemical causes particular effects in the bodies of humans or other animals. See Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, in Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* 633, 635-37, 641 (3d ed. 2011) (describing dose-response as how the response to the chemical varies with changing dose of the chemical). A dose-response relationship, while not essential evidence of a causal relationship between an agent and disease, is considered strong evidence of a causal relationship. See Green et al., *supra* at 603. In particular, consideration of a dose-response relationship is important when inferring human causation from animal studies. See *id.* at 563.

{38} To support Dahlgren’s inference from the animal studies that the agents caused Plaintiffs’ illnesses in this case, the results of the animal studies needed to be adjusted by extrapolation to humans. See Goldstein & Henifin, *Reference Guide on Toxicology*, in Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* at 646, 661-63 (3d ed. 2011) (describing scientific issues involved with extrapolating the results of an animal study to humans). Contrary to the assertions of the lower courts, Dahlgren presented evidence that the results

of the animal studies adjusted by extrapolation to the humans in this case could provide a scientifically valid basis for his inference of causation in Plaintiffs. His methodology reflected that the harmful dose of pristane the mice received, adjusted for the weight of a human, was comparable to Plaintiffs' dose determined by their daily inhaled exposure (dose per day or concentration), adjusted for the prolonged duration of that exposure to convert to dose. *See id.* at 638 & n.12 ("Dose is a function of both concentration and duration."), 681 (defining dose as "[a] product of . . . the concentration of a chemical . . . agent and the duration . . . of exposure"). This result of Dahlgren's extrapolation from the animal study is evidence that supports an inference of causation. The credibility of the extrapolation and the weight it should be accorded are questions for the jury.

{39} The district court's basis for excluding Dahlgren's study was the study's "fail[ure] to bridge the gap from association to causation." In his study, Dahlgren collected medical and environmental data from the Westgate Plaintiffs and their neighborhood and from the unexposed control community subjects and their neighborhood. He concluded that Plaintiffs' community showed both higher levels of the toxic agents at issue and a dramatic prevalence of lupus and symptoms of autoimmune disease compared to the control community. An epidemiological study compiles information on the determinants of a disease in a population to establish a correlation (or "association") between a toxic agent and the disease and to map the "incidence, distribution, and etiology of [the] disease in human populations." Green et al., *supra* at 551, 623.

{40} Having established an association between the chemical exposure and Plaintiffs' diseases, Dahlgren then demonstrated, consistent with the widely accepted Bradford Hill guidelines, that the association revealed by his epidemiological study was causal. Consistent with expectations of the dose-response in a causal relationship, his study found that the occurrences of lupus and other autoimmune diseases in the exposed Plaintiffs significantly exceeded the occurrences in the unexposed control group and that the symptoms manifested after Plaintiffs' exposures and lessened or disappeared if Plaintiffs moved away from the area. Dahlgren extrapolated the harmful dose of pristane found to induce lupus in mice to a human equivalent and determined that Plaintiffs had received a dose within that range. In addition to the animal studies involving pristane, Dahlgren cited numerous other studies where environmental contaminants consisting of the agents at issue here or similar chemicals were associated with clusters of autoimmune disorders believed to be environmentally induced. He provided a plausible biological basis for the theory that environmental toxins could induce autoimmune disease by referring to several other studies that had found such correlations. He summarized the current knowledge that benzene and mercury, two of the agents at issue here, adversely affect the human immune system, and he reviewed the studies showing that pristane triggers the formation of lupus-specific autoantibodies in mice. Dahlgren discussed alternative explanations for the association between the agents and Plaintiffs' autoimmune disorders, such as genetic susceptibility or drug-induced autoimmune disorders, and ruled them out. Having performed his study consistent with the Bradford Hill guidelines, Dahlgren was then able to use his expert judgment to assess whether the associations revealed by his own study, the animal studies, and other published studies

regarding chemical exposure provided reliable support for an inference of causation in humans. Any question regarding credibility of his judgment or interpretation is proper for the trier of fact to resolve.

{41} When the district court found that Dahlgren’s study “fail[ed] to bridge the gap from association to causation,” it improperly blurred the line between the district court’s province to evaluate the reliability of Dahlgren’s methodology and the jury’s province to weigh the strength of Dahlgren’s conclusions. *See Ambrosini*, 101 F.3d at 141 (“By attempting to evaluate the credibility of opposing experts and the persuasiveness of competing scientific studies, the district court conflated the questions of the admissibility of expert testimony and the weight appropriately to be accorded such testimony by a fact finder.”). In doing so, “the trial court failed to distinguish between the threshold question of admissibility of expert testimony and the persuasive weight to be accorded such testimony by a jury.” *Kennedy v. Collagen Corp.*, 161 F.3d 1226, 1228 (9th Cir. 1998). In *Kennedy*, the court focused on the methodology an expert applied to determine whether a medical product was capable of causing autoimmune diseases and stated, “The fact that a cause-effect relationship between [the agent] and lupus in particular [had] not been conclusively established [did] not render [the expert’s] testimony inadmissible.” *Id.* at 1230. Because the expert “set forth the steps he took in arriving at his conclusion in his deposition” and used “analogical reasoning . . . based on objective, verifiable evidence and scientific methodology of the kind traditionally used by” experts in his field, the court concluded that the proffered testimony was supported by scientific evidence and would assist the trier of fact in weighing the expert’s testimony against opposing evidence and in making a judgment about causation. *Id.* at 1230-31.

{42} In this case, Dahlgren similarly set forth the steps he took in arriving at his conclusions. His reasoning was based on scientific methodology of the kind traditionally used by experts addressing causation in toxicological epidemiology. We conclude that the methodology of Dahlgren’s study supports a valid scientific inference that is probative of causation, even if it does not conclusively establish that the specific chemicals at issue here, the agents, can cause lupus or other autoimmune disorders. Accordingly, Dahlgren’s study and his causation testimony should have been admitted.

D. Summary Judgment Was Improperly Granted

{43} Based on an improper conclusion that Plaintiffs’ general causation evidence was inadmissible, the district court granted partial summary judgment to Defendants on Plaintiffs’ claims concerning lupus and other autoimmune disorders. Because we reverse that determination of inadmissibility, we reverse the grant of summary judgment as the evidence creates a genuine issue of material fact that precludes summary judgment. *See* Rule 1-056(C).

V. CONCLUSION

{44} We reverse the district court’s exclusion of Dahlgren’s expert causation testimony,

his study, and the animal studies on which his study relied. We also reverse the summary judgment granted on the basis of those exclusions. We remand to the district court for proceedings consistent with this opinion.

{45} IT IS SO ORDERED.

CHARLES W. DANIELS, Justice

WE CONCUR:

BARBARA J. VIGIL, Chief Justice

PETRA JIMENEZ MAES, Justice

EDWARD L. CHÁVEZ, Justice

JUDITH K. NAKAMURA, Justice, not participating