

**IN THE UNITED STATES COURT OF APPEALS
FOR THE FIFTH CIRCUIT**

United States Court of Appeals
Fifth Circuit

FILED

June 20, 2012

No. 11-50193

Lyle W. Cayce
Clerk

GREGORY SCOTT JOHNSON,

Plaintiff – Appellant,

v.

ARKEMA, INCORPORATED,

Defendant – Appellee.

Appeal from the United States District Court
for the Western District of Texas

Before REAVLEY, ELROD, and HAYNES, Circuit Judges.

PER CURIAM:

In this toxic tort case, we consider whether the district court erred in: (1) excluding the opinions of Gregory Johnson’s expert witnesses on the element of causation; and (2) granting summary judgment in favor of Arkema, Inc. because Johnson was unable to prove causation without the opinions of his excluded causation experts. We **AFFIRM** the district court’s judgment in all respects except as to Johnson’s claims regarding his acute injuries, on which we **REVERSE** and **REMAND** for further proceedings.

No. 11-50193

I.

Johnson worked as a machine repairman at Owens Illinois Inc.'s glass bottling plant in Waco, Texas from May 1998 to the end of 2008. On two separate occasions, first in early June 2007 and again on July 15, 2007, Johnson was directed to perform work in close proximity to a device known as a C-4 Hood, which was designed, manufactured, and installed by Arkema. C-4 Hoods are utilized by Owens Illinois to apply a chemical known as Certincoat to the glass bottles it produces as the bottles are transported along a conveyor belt.¹ Certincoat is composed mostly of monobutyltin trichloride (MBTC), an organometallic compound based on tin. Under the elevated temperatures of the C-4 hoods, MBTC vaporizes and then decomposes when it contacts the glass bottles on the conveyer belt. Hydrochloric acid (HCl) and tin oxide are byproducts of MBTC. Arkema's C-4 Hoods are designed to vacuum up and capture any vapors that are not deposited on the glass bottles, thus preventing the escape of MBTC, HCl and tin oxide into the workplace environment. According to Johnson, the C-4 Hood he worked near on those two occasions in the summer of 2007 failed to perform its proper preventative function, resulting in his exposure to Certincoat and its chemical byproducts.

Specifically, Johnson alleges that within fifteen minutes of first approaching the C-4 hood in early June 2007 he: (1) smelled a sweet, unique chemical odor; (2) noticed chemical buildup on the conveyer belt; (3) developed a sore throat; (4) felt burning and watery eyes; and (5) experienced chest pain and breathing difficulty. Johnson nevertheless continued to work in these conditions for approximately four to five hours and, thereafter, neither reported the incident to his supervisor nor sought immediate medical attention. A few days later, on June 9, 2007, Johnson's family doctor diagnosed him with

¹ Arkema is also the designer and manufacturer of Certincoat.

No. 11-50193

pneumonia. At his June 18, 2007 follow-up visit, Johnson reported that he “fe[lt] a lot better” and his doctor concluded that he could return to work the following day.

The next month, on July 15, 2007, Johnson was again instructed to work near the C-4 Hood. While doing so for approximately two to three hours, Johnson experienced the same symptoms that he felt during his first alleged instance of Certincoat exposure. This time, however, Johnson reported the incident to his supervisor and sought immediate medical attention at a local emergency room.

On August 8, 2007, upon Johnson’s disclosure of the two exposure incidents to his treating physician, Dr. Camille Hinojosa, Johnson was diagnosed with chemical pneumonitis and advised to see a pulmonologist. According to Johnson, his lung condition progressively worsened over the course of the years following the exposure incidents, culminating in a diagnosis of severe restrictive lung disease and pulmonary fibrosis.²

II.

On November 3, 2008, Johnson filed a personal injury lawsuit against Arkema in the 60th Judicial District Court of Jefferson County, Texas, claiming that Arkema’s C-4 Hood proximately caused his restrictive lung disease and pulmonary fibrosis.³ Arkema removed the matter to the Eastern District of Texas and, on April 30, 2009, this matter was transferred to the Western District of Texas. In his complaint, Johnson raises theories of negligence and strict liability based on Arkema’s design, manufacture, marketing, and

² Although Arkema disputes this diagnosis, the dispute is not material to the disposition of this appeal.

³ Johnson’s brief defines pulmonary fibrosis as the “inflammation and progressive fibrosis of the pulmonary alveolar walls”; it is “one of a family of related diseases called interstitial lung diseases. All of these diseases can result in lung scarring.”

No. 11-50193

installation of its C-4 Hoods. Johnson seeks compensatory and punitive damages, including, but not limited to, compensation for past physical pain and medical expenses.

Arkema filed motions to exclude the opinions of Dr. Richard Schlesinger, Johnson's expert toxicologist, and Dr. Charles Grodzin, Johnson's expert pulmonologist, under Federal Rule of Evidence 702 and the Supreme Court's decision in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993). Arkema also filed a motion for summary judgment, contending that Johnson was unable to present scientifically reliable evidence establishing that exposure to the chemicals in Certincoat can cause restrictive lung disease and pulmonary fibrosis.

On December 16, 2010, the magistrate judge issued a report and recommendation to the district court regarding Arkema's *Daubert* motions. The magistrate judge recommended: (1) excluding Dr. Schlesinger's opinion, which only addressed causation, as unreliable and irrelevant; and (2) limiting Dr. Grodzin's opinion so that he could only opine on the nature and extent—but not the cause—of Johnson's illness. The district court adopted the report and recommendation and subsequently granted summary judgment in favor of Arkema. The district court reasoned that summary judgment was appropriate because—given the exclusion of Dr. Schlesinger's opinion and the limitation of Dr. Grodzin's opinion—Johnson “ha[d] no evidence that any lung injury he suffered [was] a result of his exposure to MBTC and/or HCl.” In so doing, the district court rejected Johnson's claim that the similar symptoms experienced by his co-workers provided sufficient summary judgment evidence of causation:

The only thing presented with the summary judgment material which was not presented to Magistrate Judge Manske is Plaintiff's evidence that other Owens employees suffered lung injuries similar to his. However, an inspection of this evidence fails to reveal a single employee who has suffered a permanent, or chronic, lung

No. 11-50193

injury after exposure to Arkema's chemicals. While other employees may have been exposed to the same chemicals, they suffered only transitory symptoms which quickly resolved.

This appeal followed.

III.

The first issues we consider concern the district court's evidentiary rulings under Rule 702 and *Daubert*, which we review for abuse of discretion. *Curtis v. M&S Petroleum, Inc.* 174 F.3d 661, 668 (5th Cir. 1999). "A trial court abuses its discretion when its ruling is based on an erroneous view of the law or a clearly erroneous assessment of the evidence." *Bocanegra v. Vicmar Servs., Inc.*, 320 F.3d 581, 584 (5th Cir. 2003). In conducting our review, "[w]e are mindful that under *Daubert* and Fed. R. Evid. 702, a district court has broad discretion to determine whether a body of evidence relied upon by an expert is sufficient to support that expert's opinion." *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 354 (5th Cir. 2007); *see also Rider v. Sandoz Pharm. Corp.*, 295 F.3d 1194, 1197 (11th Cir. 2002) ("[J]udges have considerable leeway in both how to test the reliability of evidence and determining whether such evidence is reliable." (citing *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 151–53 (1999))).

The admissibility of expert testimony is governed by Federal Rule of Evidence 702, which provides:

A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:

- (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
 - (b) the testimony is based on sufficient facts or data;
 - (c) the testimony is the product of reliable principles and methods;
- and

No. 11-50193

(d) the expert has reliably applied the principles and methods to the facts of the case.

Fed. R. Evid. 702.

In *Daubert*, the Supreme Court “explained that Rule 702 assigns to the district judge a gatekeeping role to ensure that scientific testimony is both reliable and relevant.” *Curtis*, 174 F.3d at 668 (citing *Daubert*, 509 U.S. at 597). The reliability prong mandates that expert opinion “be grounded in the methods and procedures of science and . . . be more than unsupported speculation or subjective belief.” *Id.* (citing *Daubert*, 509 U.S. at 590); *see also Moore v. Ashland Chem., Inc.*, 151 F.3d 269, 276 (5th Cir. 1998) (en banc) (“[T]he party seeking to have the district court admit expert testimony must demonstrate that the expert’s findings and conclusions are based on the scientific method, and, therefore, are reliable.”). The relevance prong requires the proponent to demonstrate that the expert’s “reasoning or methodology can be properly applied to the facts in issue.” *Curtis*, 174 F.3d at 668 (citing *Daubert*, 509 U.S. at 592–93).

Furthermore, courts consider the following non-exclusive list of factors when conducting the reliability inquiry:

(1) whether the theory or technique has been tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) the known or potential rate of error of the method used and the existence and maintenance of standards controlling the technique’s operation; and (4) whether the theory or method has been generally accepted by the scientific community.

Id. at 668–69 (citing *Daubert*, 509 U.S. at 593–94). “The proponent need not prove to the judge that the expert’s testimony is correct, but she must prove by a preponderance of the evidence that the testimony is reliable.” *Moore*, 151 F.3d at 276; *see also Wells v. Smithkline Beecham Corp.*, 601 F.3d 375, 378 (5th Cir. 2010) (“Although there are ‘no certainties in science,’ the expert must present

No. 11-50193

conclusions ‘ground[ed] in the methods and procedures of science.’” (alteration in original) (quoting *Daubert*, 509 U.S. at 590)).

A.

Johnson contends that the district court abused its discretion in excluding Dr. Schlesinger’s expert opinion that MBTC and HCl⁴ can cause restrictive lung disease and pulmonary fibrosis.⁵ The district court excluded Dr. Schlesinger’s testimony after determining that: (1) Dr. Schlesinger could not cite to one epidemiological or controlled study of humans indicating that exposure to MBTC or HCl could cause restrictive lung disease and pulmonary fibrosis; (2) Dr. Schlesinger relied, in part, on two animal studies that were highly distinguishable from and not correlated to Johnson’s two instances of MBTC and HCl exposure; and (3) the scientific literature is devoid of any data or peer-reviewed articles indicating that exposure to MBTC or HCl will result in chronic lung disease, and such a proposition is not generally accepted in the scientific community. Johnson argues that the district court erred in so ruling because: (1) MBTC and HCl are part of a toxicological class of chemicals labeled as irritants that are known to potentially cause pulmonary fibrosis; (2) Dr. Schlesinger based his opinion on reliable scientific data concerning MBTC and HCl exposure—including animal studies, material safety data sheets, and guidelines from regulatory and advisory bodies—that support his conclusions; and (3) Dr. Schlesinger’s opinion is buttressed by the temporal connection between Johnson’s exposure and illness. As set forth below, because we are unable to conclude that the district court abused its broad discretion in

⁴ Although Certincoat also contains tin oxide, Dr. Schlesinger did not offer the opinion that tin oxide can cause restrictive lung disease and pulmonary fibrosis.

⁵ Dr. Schlesinger has an extensive background in the field of inhalation toxicology and is currently a professor in the Department of Biology and Health Sciences at Pace University. In pursuing its *Daubert* motion, Arkema did not dispute Dr. Schlesinger’s qualifications as a toxicologist.

No. 11-50193

performing its gatekeeping function under *Daubert*, we affirm the exclusion of Dr. Schlesinger's expert opinions.

1.

Johnson first claims that the district court erred in discounting Dr. Schlesinger's "class of chemicals" theory. Johnson asserts that Dr. Schlesinger's opinion is reliable because "MBTC and HCl are part of a group of chemicals labeled by toxicologists as 'strong irritants.'" According to Johnson, this classification is significant because "[a]ll 'strong irritants' have the same physiological effect when they contact biological tissue—production of inflammation." Moreover, numerous peer-reviewed studies of exposure to other chemicals labeled as irritants—including chlorine, ammonia, and nitric acid vapor—have reported lung scarring following acute exposure to those respective irritants. Thus, although Dr. Schlesinger only relied on one MBTC and one HCl study in forming his opinions, Johnson contends that Dr. Schlesinger's conclusions are reinforced by the more prevalent studies involving other irritants.

Our review of Supreme Court and this circuit's case law confirms that, in forming a reliable opinion regarding the effects of exposure to a particular chemical, an expert may extrapolate data from studies of similar chemicals. *See Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997) ("Trained experts commonly extrapolate from existing data."); *Moore*, 151 F.3d at 278–79. However, "[t]o support a conclusion based on such reasoning, the extrapolation or leap from one chemical to another must be reasonable and scientifically valid." *Moore*, 151 F.3d at 279. Thus, courts are free to reject a theory based on extrapolation when "there is simply too great an analytical gap between the data and the opinion proffered." *Joiner*, 522 U.S. at 146.

We applied the foregoing principles in our decision in *Wells*, 601 F.3d at 380. In that case, three experts relied on a study of a class of drugs known as

No. 11-50193

“dopamine agonists” in support of their conclusion that a specific drug within the class, Requip, could have potentially caused the appellant’s compulsive gambling problem.⁶ *Id.* We held that the district court did not abuse its discretion in excluding the experts, in part, because they failed to bridge the analytical gap between the generalized nature of the class-wide dopamine agonist study and the specific characteristics of Requip, “a drug that functions differently than other dopamine agonists.” *Id.*; *cf. Knight*, 482 F.3d at 350, 352–53 (exclusion of expert who relied on studies of organic solvents was not an abuse of discretion, in part, because of the expert’s failure to address how exposure to benzene, the specific organic solvent at issue, could be correlated to those studies involving exposure to other organic solvents in addition to benzene).

We also view the Tenth Circuit’s *Rider* decision as particularly instructive in this case. 295 F.3d at 1200–02. In *Rider*, the experts relied on evidence that drugs in a class known as “ergot alkaloids” caused vasoconstriction to support the proposition that a specific drug within the class, bromocriptine, did so as well. *Id.* at 1200–01. The *Rider* court first reiterated that, in *Joiner*, the Supreme Court:

established the important test of analytical “fit” between the methodology used and the conclusions drawn. . . . The [C]ourt reasoned that just because a methodology is acceptable for some purposes, it may not be acceptable for others, and a court may not admit evidence when there is “simply too great an analytical gap between the data and the opinion proffered.”

Id. at 1197 (quoting *Joiner*, 522 U.S. at 146). The court then affirmed the exclusion of the experts because ergot alkaloids have diverse chemical compositions and the experts failed to demonstrate that bromocriptine “should have the same effects as other drugs in that class.” *Id.* at 1201–02.

⁶ A dopamine agonist is a drug “that stimulates the dopamine receptors in the brain to alleviate symptoms of Parkinson’s [Disease].” *Id.* at 377.

No. 11-50193

In this case, we conclude that the district court did not abuse its discretion in excluding Dr. Schlesinger’s “class of chemicals” theory. Dr. Schlesinger opined that MBTC and HCl can cause pulmonary fibrosis because they are part of a class of chemicals labeled as irritants:

It is generally accepted in the field of toxicology that both HCl and MBTC belong to a class of chemicals known as irritants. Toxicologically, all irritants have the same effect when they contact biological tissue, namely production of inflammation.

...

It is an accepted fact that acute inhalation of irritants can result in chronic diseases, including restrictive lung disease and pulmonary fibrosis.

...

While all irritants produce inflammation, as described above, *respiratory irritants are different in their specific chemical structure*. These differences relate to toxic potency (the exposure concentration needed to produce damage) and solubility (which affects the area of the lung an inhaled irritant would be expected to reach). Exhibit A #6. However, while chemicals within a class may differ in toxic potency and solubility, the mechanism of toxicity is the same, as described above. Therefore, *if exposure to an irritant is of sufficient concentration to cause inflammation*, there are no other differences among irritants in the same class in terms of capability to cause a particular lung injury.

(Emphasis added). Dr. Schlesinger did not go further, however, and explain how, based on any of the specific properties and toxicities of similar irritants when compared with those of MBTC and HCl, Johnson’s exposure to MBTC and HCl was at a sufficient concentration level to cause restrictive lung disease and pulmonary fibrosis.⁷ See *Moore*, 151 F.3d at 278–79 (“Dr. Jenkins made no attempt to explain his conclusion by asserting that the Toluene solution [to

⁷ Instead, Dr. Schlesinger’s opinion that the concentration levels of MBTC and HCl were sufficient to cause lung damage was based on other scientific evidence deemed unreliable by the district court, discussed *infra* at III.2–3.

No. 11-50193

which plaintiff was exposed] had properties similar to another chemical exposure to which [reactive airway dysfunction syndrome, or (RADS)] had been scientifically linked.”); *see also Mitchell v. Gencorp. Inc.*, 165 F.3d 778, 782 (10th Cir. 1999) (although the “record contain[ed] some testimony about the similarities between benzene and [d]efendant’s products,” there was no “additional testimony explaining exactly what these similarities [were] and how the similarities cause[d] the human body to respond to [d]efendant’s chemicals in a manner similar to benzene”). Put differently, save for highlighting their shared classifications as irritants, Dr. Schlesinger did not attempt to explain any direct correlation or “fit” between the chemicals in Certincoat and the known scientific data concerning exposure to, for example, chlorine, ammonia, or nitric acid vapor. Accordingly, given the diverse chemical structures and toxicities of irritants, which Dr. Schlesinger acknowledged,⁸ we hold that the district court did not abuse its discretion in concluding that Dr. Schlesinger’s “class of chemicals” theory presented “too great an analytical gap between the data and the opinion proffered.”⁹ *Joiner*, 522 U.S. at 146.

2.

Johnson next asserts that reliable and relevant scientific data concerning exposure to HCl supports Dr. Schlesinger’s conclusion that HCl causes scarring to lung tissue. Johnson first points to the material safety data sheet (MSDS) issued by Airgas, Inc., a company wholly unrelated to Arkema, which warns that

⁸ One of the articles Dr. Schlesinger submitted with his report also implicitly addressed the diverse characteristics of irritants, providing that “[t]he health effects of an acute exposure to an irritant gas or vapor are dependent on the physiochemical properties of *that particular gas or vapor*, as well as specific host factors.” (Emphasis added).

⁹ This outcome may have been different had Dr. Schlesinger presented other reliable scientific evidence to support his causation opinion. For instance, if Dr. Schlesinger had other reliable evidence demonstrating that the concentration levels of MBTC and HCl were sufficiently high to impair respiratory function, then the analytical leap found in his “class of chemicals” theory could potentially have been reduced to a mere step, rendering Dr. Schlesinger’s opinion reliable.

No. 11-50193

HCl can be “severely corrosive to the respiratory system.” The district court found the warning irrelevant and unreliable because: (1) “the Airgas MSDS does not state that exposure to HCl can cause severe restrictive lung disease and pulmonary fibrosis”; and (2) “most importantly,” Johnson did not provide “any science behind the MSDS,” such as “the duration or concentration of exposure needed to produce the noted effects” or the scientific literature “relied upon by Airgas for the statements contained in the MSDS.”

We conclude that the district court did not abuse its discretion in disregarding the Airgas MSDS. Dr. Schlesinger failed to come forth with any scientific data to support the MSDS’s warning. He also acknowledged that there is scant scientific evidence of a “cause-and-effect relationship between hydrochloric acid and restrictive lung disease.” Under such circumstances, the Airgas MSDS, standing alone, need not have been accorded any weight. See *Moore*, 151 F.3d at 278 (stating that the district court did not abuse its discretion in finding a MSDS unreliable in part because the expert “did not know what tests Dow [Corning] had conducted in generating the MSDS”).

Johnson next cites a 1993 study of HCl’s effect on nine baboons who were exposed “for fifteen minutes to three concentrations (500 ppm, 5,000 ppm, and 10,000 ppm) of HCl for a one year period.”¹⁰ The study found that one of the nine baboons developed fibrosis after being exposed to a 10,000 ppm concentration of HCl. It ultimately concluded that HCl inhalation did not result in “the development of impaired respiratory/pulmonary function, except at the highest concentration.” Although Johnson was only exposed to a ten to fifty ppm concentration of HCl, Johnson claims that the baboon study is reliable and relevant because: (1) Johnson was exposed to HCl for a much longer time period than the baboon who developed fibrosis; (2) baboons are considered to be an

¹⁰ The acronym “ppm” stands for “parts per million.”

No. 11-50193

animal species that is a surrogate of man; and (3) the study shows that HCl is capable of causing fibrosis.

We have previously recognized the “very limited usefulness of animal studies when confronted with questions of toxicity.” *Allen v. Pa. Eng’g Corp.*, 102 F.3d 194, 197 (5th Cir. 1996) (quoting *Brock v. Merrell Dow Pharm.*, 874 F.2d 307, 313 (5th Cir. 1989)). Accordingly, “studies of the effects of chemicals on animals must be carefully qualified in order to have explanatory potential for human beings.”¹¹ *Id.* Here, the district court found the baboon study unreliable and irrelevant because Dr. Schlesinger did not even attempt to show that there was a “correlation between the duration and length of the baboon exposure and Mr. Johnson’s exposure.” Likewise, Dr. Schlesinger admitted that the respiratory tracts of humans are “pretty unique,” further diminishing the significance of the baboon study. Finally, Johnson’s reliance upon the baboon study was weakened by the fact that there are no other studies of baboons or other animals that corroborate the baboon study’s conclusions. *See id.* In light of *Allen*’s “careful qualification” requirement, we conclude that the district court did not abuse its discretion in rejecting the baboon study. *See also Joiner*, 522 U.S. at 144–45 (finding that the court did not abuse its discretion in rejecting the experts’ reliance on animal studies—which involved the injection of “massive doses” of certain chemicals into infant mice—because the “studies were so dissimilar to the facts presented in th[e] litigation”); *cf. Gulf S. Insulation v. U.S. Consumer Prod. Safety Comm’n*, 701 F.2d 1137, 1146 (5th Cir. 1983) (finding a rat study inconclusive because of the small number of rats tested, the high

¹¹ In *Allen*, we concluded that a study’s finding that ethylene oxide (EtO) caused cancer in rats provided “at best speculative support” for the conclusion that EtO could cause cancer in humans because a different study of mice produced no such results. *Id.* In explaining our conclusion, we adopted the following logic of the appellee’s expert: “Thus, the lack of capacity for the F-344 rat to predict how even the mouse model responds necessarily undercuts confidence that the rat will predict accurately how other species including humans will respond [to EtO exposure].” *Id.*

No. 11-50193

dosages given to the rats, and difficulty in extrapolating those results to humans).

Finally, Johnson contends that he was exposed to amounts of HCl that were between two and ten times the permissible exposure levels set by the Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH). Johnson also references the Acute Exposure Guideline Levels set by the National Research Council (NRC), which provide that Johnson could have been exposed to a “disabling” and possibly “lethal” dose of HCl.

In *Allen*, we addressed the significance of guidelines promulgated by regulatory and advisory bodies:

Regulatory and advisory bodies such as IARC, OSHA and EPA utilize a “weight of the evidence” method to assess the carcinogenicity of various substances in human beings and suggest or make prophylactic rules governing human exposure. This methodology results from the preventive perspective that the agencies adopt in order to reduce public exposure to harmful substances. *The agencies’ threshold of proof is reasonably lower than that appropriate in tort law*, which “traditionally make[s] more particularized inquiries into cause and effect” and requires a plaintiff to prove “that it is more likely than not that another individual has caused him or her harm.”

Allen, 102 F.3d at 198 (emphasis added) (quoting *Wright v. Willamette Industries, Inc.*, 91 F.3d 1105, 1107 (8th Cir.1996)). Thus, *Allen* demonstrates that chemical guidelines are not necessarily reliable in all toxic tort cases. It may be appropriate first to consult the underlying basis for their proscriptions before an expert’s reliance on them can pass *Daubert* muster.

As with the Airgas MSDS, however, Johnson once again does not provide any scientific data or literature to explain how or why the various exposure limits and guidelines were established for HCl. Similarly, Johnson does not argue that the guidelines and exposure limits exist to protect people from

No. 11-50193

developing severe restrictive lung disease and pulmonary fibrosis. Thus, we conclude that the OSHA, NIOSH, and NRC guidelines and exposure limits, standing alone, are insufficient to demonstrate abuse of discretion on the part of the district court. *See also id.* at 195–96 (“[U]nder the circumstances of this case, the fact that EtO has been classified as a carcinogen by agencies responsible for public health regulations is not probative of the question whether Allen’s brain cancer was caused by EtO exposure.”).

In sum, the Airgas MSDS, baboon study, and OSHA, NIOSH, and NRC guidelines do not sufficiently support Johnson’s theory that HCl is known to cause scarring to lung tissue.¹² The district court did not abuse its discretion in dismissing this data as irrelevant and unreliable under *Daubert*.

3.

Johnson also argues that reliable and relevant scientific data concerning exposure to MBTC supports Dr. Schlesinger’s conclusion that MBTC causes scarring to lung tissue. Johnson first references Arkema’s MSDS, which explains that MBTC “CAUSES RESPIRATORY TRACT IRRITATION” and that:

Inhalation and skin contact are expected to be the primary routes of occupational exposure to this material. Based on single exposure animal tests, it is considered to be slightly toxic if swallowed and corrosive to eyes and skin. If swallowed, this material may cause severe internal injury, characterized by pain in the mouth, throat and stomach, vomiting and breathing difficulties.

¹² Johnson also notes that Arkema’s own expert acknowledged that HCl, if inhaled, may cause “acute lung injury or chronic persistent pulmonary function abnormalities,” including “upper airway edema and burns, hypoxia, stridor, pneumonitis and tracheobronchitis.” This admission does not indicate, however, the duration or concentration of HCl exposure that is necessary to cause such ailments. Furthermore, the expert’s admission does not demonstrate that HCl can cause severe restrictive lung disease or pulmonary fibrosis. Finally, assuming it exists, Johnson does not provide any of the underlying data Arkema’s expert may have relied on in forming an opinion regarding the potential dangers associated with HCl exposure. The district court acted within its discretion in discounting this admission as it relates to Dr. Schlesinger’s opinions.

No. 11-50193

Johnson relies on our decision in *Curtis*, 174 F.3d at 669, for the proposition that Arkema's MSDS constitutes scientifically reliable evidence supporting Dr. Schlesinger's causation opinion.¹³

Johnson's reliance on *Curtis* is unavailing. In *Curtis*, we merely found that a MSDS was reliable because it was consistent with a wealth of other reliable information (including a detailed Supreme Court discussion) regarding the potential hazards associated with benzene exposure.¹⁴ *Id.* at 669–70. There is, however, nothing in *Curtis* indicating that material safety data sheets constitute *per se* reliable support for an expert's opinion. To the contrary, in exercising its discretion as a gatekeeper, a court may refrain from treating a MSDS as reliable until it is presented with scientific evidence justifying the relevant statements found within the MSDS. *See Moore*, 151 F.3d at 278 (district court did not abuse its discretion in finding a MSDS unreliable in part because the expert “did not know what tests Dow had conducted in generating the MSDS”).

Moreover, the district court did not abuse its discretion in rejecting the only evidence underlying Arkema's MSDS, namely, one unpublished study performed by Arkema in 1988 concerning MBTC's effect on rats. The study was designed to assess the toxic effects of MBTC when administered by inhalation to rats for six hours per day, five days per week, for four weeks at target concentrations of one, ten, and thirty milligrams per cubic meter. The study did not make any conclusions regarding restrictive lung disease and pulmonary

¹³ Johnson also offers a temporal proximity argument, stating that Johnson's initial symptoms after his exposure to Certincoat were in accord with the warnings in Arkema's MSDS. This temporal proximity issue is addressed in III.4, *infra*.

¹⁴ We also note that the MSDS did not play an important role in *Curtis* as the defendant “d[id] not seriously challenge” the expert's conclusion that “exposure to benzene at levels of 200-300 ppm would cause the injuries suffered by Plaintiffs.” *Id.* at 670. Instead, the primary dispute was over a matter wholly unrelated to the MSDS: whether the expert had shown that the plaintiffs were actually exposed to 200-300 ppm levels of benzene. *Id.* at 670–72.

No. 11-50193

fibrosis, and instead only found that exposure to MBTC had a discernable effect on the lung tissue of rats.¹⁵ The district court determined that the rat study was irrelevant and unreliable because Dr. Schlesinger admitted that “there is no correlation between the durations of exposure” experienced by the rats, on the one hand, and Johnson, on the other. Based on *Allen*’s requirement that animal studies be “carefully qualified in order to have explanatory potential for human beings,” we conclude that the district court did not abuse its discretion in discounting this rat study.¹⁶ *Allen*, 102 F.3d at 197. It follows that the district court did not abuse its discretion in discounting Arkema’s MSDS because its warnings were founded on the rat study.

Johnson also raises the fact that he was exposed to a concentration level of MBTC that was between 100 and 500 times OSHA’s permissible MBTC exposure limit of .1 milligrams per cubic meter. The district court was unpersuaded by the sheer magnitude of, according to OSHA’s exposure limit, Johnson’s over-exposure to MBTC. It found the maximum exposure limit misleading because OSHA set the .1 milligram per cubic meter threshold for all

¹⁵ Specifically, the study concluded that:

Grossly, the incidence of lung discoloration was increased in exposed males and females. Microscopically, amorphous material, (perhaps the test material or monobutyltin dihydroxy chloride, the hydrolysis product of monobutyltin trichloride) and alveolar edema were evident in the lungs of exposed males and females. Other lung changes which occurred with increased incidence and severity in the exposed groups included peribronchial lymphoid cell accumulation and perivascular lymphoid cell infiltrate, extravasated erythrocytes (males only), and accumulation of alveolar macrophages. Dose related responses were shown only by alveolar edema in both sexes and by alveolar erythrocytes in males only.

¹⁶ Johnson was exposed to between ten and fifty milligrams per cubic meter of MBTC. Johnson was, therefore, exposed to amounts of MBTC that were similar in concentration but not duration to the amounts of MBTC involved in the rat study. Nevertheless, Dr. Schlesinger could not adequately explain, as required by *Allen*, why MBTC’s effect on the rats provides a reliable scientific basis for the conclusion that MBTC can cause restrictive lung disease and pulmonary fibrosis in human beings.

No. 11-50193

organotins, not just MBTC. Critically, Dr. Schlesinger conceded that this threshold for organotin exposure was “clear[ly]” not set based on data relating specifically to MBTC. Instead, according to Dr. Schlesinger, the OSHA threshold would be “based on whichever [organotin] they had the most data on in terms of inhalation.” Dr. Schlesinger also conceded that some organotin compounds are more toxic than others. Given Dr. Schlesinger’s concessions, we conclude that the district court did not abuse its discretion in refusing to treat the OSHA exposure limit as reliable scientific evidence. *See Allen*, 102 F.3d at 198 (regulatory “agencies’ threshold of proof is reasonably lower than that appropriate in tort law”).

Accordingly, we hold that Arkema’s MSDS, the rat study, and OSHA’s guidelines do not sufficiently support Johnson’s theory that MBTC is known to cause scarring to lung tissue. The district court did not abuse his discretion in dismissing this data as irrelevant and unreliable under *Daubert*.

4.

Finally, Johnson contends that the strong temporal connection between Johnson’s exposure to Certincoat and subsequent lung injury supports Dr. Schlesinger’s causation conclusion. Johnson states that he “has never smoked and, prior to July 2007, had never suffered a lung injury, never been diagnosed with asthma, never been exposed to a dangerous level of any other toxic chemical, and had no history of lung disease or breathing difficulties.” Moreover, “Johnson had worked in the same plant for over nine years without incident prior to the installation of Arkema’s new chemical hoods in June 2007.”

In *Curtis*, we explained that “temporal connection standing alone is entitled to little weight in determining causation.” *Curtis*, 174 F.3d at 670. “However, a temporal connection is entitled to greater weight when there is an established scientific connection between exposure and illness or other circumstantial evidence supporting the causal link.” *Id.*

No. 11-50193

Our foregoing discussion indicates that there is neither an established scientific connection between exposure to Certincoat and subsequent lung disease nor sufficient circumstantial evidence to indicate a causal link between the same. Therefore, under *Curtis*, the district court acted well within its discretion in according little weight to the temporal connection theory alleged by Johnson.

5.

In conclusion, we hold that the district court did not abuse its discretion in excluding Dr. Schlesinger's expert opinion under *Daubert*. Dr. Schlesinger could not cite to one epidemiological or controlled study of humans indicating that exposure to MBTC or HCl could cause restrictive lung disease and pulmonary fibrosis. *See Allen*, 102 F.3d at 197 ("Undoubtedly, the most useful and conclusive type of evidence in a case such as this is epidemiological studies."). Also, Dr. Schlesinger neither extrapolated from existing data concerning chemicals similar to those in Certincoat nor correlated existing animal studies to Johnson's two exposure episodes. Instead, he relied on blanket statements from presumably credible sources—such as material safety data sheets and advisory guidelines—but failed to present the scientific evidence upon which those statements were founded. *Cf. Joiner*, 522 U.S. at 146 ("[N]othing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert."). Finally, Dr. Schlesinger did not offer evidence that his theory has been generally accepted by the scientific community. The district court's exclusion of Dr. Schlesinger's expert opinion is affirmed.

B.

Johnson next contests the district court's limitation of Dr. Grodzin's opinion, which prevented Dr. Grodzin from expressing his conclusion that MBTC

No. 11-50193

and HCl¹⁷ caused Johnson's lung disease.¹⁸ In reaching his causation conclusion, Dr. Grodzin's research and analysis essentially mirrored Dr. Schlesinger's save for one key distinction: Dr. Grodzin performed a "differential diagnosis" of Johnson. Accordingly, we need only consider whether, given the existence of Dr. Grodzin's differential diagnosis, the district court's exclusion of Dr. Grodzin's opinion constitutes the abuse of discretion.¹⁹

As the Fourth Circuit has observed:

A reliable differential diagnosis typically, though not invariably, is performed after "physical examinations, the taking of medical histories, and the review of clinical tests, including laboratory tests," and generally is accomplished by determining the possible causes for the patient's symptoms and then eliminating each of these potential causes until reaching one that cannot be ruled out or determining which of those that cannot be excluded is the most likely.

Westberry v. Gislaved Gummi AB, 178 F.3d 257, 262 (4th Cir. 1999) (quoting *Kannankeril v. Terminix Int'l, Inc.*, 128 F.3d 802, 807 (3d Cir. 1997)). Many

¹⁷ Like Dr. Schlesinger, Dr. Grodzin's opinion did not address whether tin oxide caused Johnson's lung disease.

¹⁸ Dr. Grodzin currently serves as the Medical Director for the Denton Medical Services Pulmonary Rehabilitation Center. Arkema does not challenge Dr. Grodzin's credentials.

We also note that Johnson sought to admit Dr. Grodzin as an expert so that Dr. Grodzin could offer his medical diagnosis that Johnson suffers from "interstitial lung disease resulting in a severe restrictive condition, and pulmonary fibrosis." The district court ruled that this portion of Dr. Grodzin's opinion satisfied *Daubert's* requirements and was, therefore, admissible. Johnson's appeal only relates to the district court's exclusion of Dr. Grodzin's opinion regarding the cause of Johnson's lung disease.

¹⁹ At the *Daubert* hearing, Johnson informed the magistrate judge that Dr. Grodzin's opinion was derivative of Dr. Schlesinger's opinion with the exception of Dr. Grodzin's differential diagnosis analysis. Accordingly, we need not restate our conclusions in III.A., *supra*, here.

Dr. Grodzin also considered scientific evidence indicating that a prescription drug called Bleomycin can cause pulmonary fibrosis and argued that this evidence supported a similar finding with regard to MBTC and HCl. This theory fails for the reasons stated in III.A.1., *supra*.

No. 11-50193

courts have found that a properly performed differential diagnosis can yield a reliable expert opinion. *See id.* at 262–63.

However, the results of a differential diagnosis are far from reliable *per se*. In *Moore*, for example, after conducting a differential diagnosis, the expert diagnosed the plaintiff with RADS. *Moore*, 151 F.3d at 273; *See also id.* at 288–90 (Dennis, J., dissenting) (noting that the expert had conducted a differential diagnosis of the plaintiff). The expert also concluded that the plaintiff’s RADS was caused by certain chemicals to which the plaintiff was exposed based on his analysis of MSDS warnings, his examination and testing of the plaintiff, and the close temporal proximity between the plaintiff’s exposure and subsequent injury. *Id.* Despite the expert’s differential diagnosis, we held that the district judge did not abuse its discretion in excluding the expert’s causation testimony because he failed to present reliable scientific support showing that the chemicals at issue could actually cause RADS. *Id.* at 278–79.

Furthermore, *Moore* illustrates that an expert may not rely on a differential diagnosis to circumvent the requirement of general causation. *See id.* at 278 (“Dr. Jenkins offered no scientific support for his general theory that exposure to Toluene solution at any level would cause RADS.”); *see also Curtis*, 174 F.3d at 669–70; *Goebel v. Denver & Rio Grande W. R.R. Co.*, 346 F.3d 987, 999 (10th Cir. 2003) (A district court “can admit a differential diagnosis that it concludes is reliable if general causation has been established”). As we explained in *Knight*:

General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual’s injury. Evidence concerning specific causation in toxic tort cases is admissible only as a follow-up to admissible general-causation evidence. Thus, there is a two-step process in examining the admissibility of causation evidence in toxic tort cases. First, the district court must determine whether there is general

No. 11-50193

causation. Second, if it concludes that there is admissible general-causation evidence, the district court must determine whether there is admissible specific-causation evidence.

Knight, 482 F.3d at 351 (internal quotation marks and citations omitted). Thus, before courts can admit an expert's differential diagnosis, which, by its nature, only addresses the issue of specific causation, the expert must first demonstrate that the chemical at issue is actually capable of harming individuals in the general population, thereby satisfying the general causation standard. *See id.*

Here, like in *Moore*, Dr. Grodzin's differential diagnosis is based on the presumption that MBTC and HCl are actually capable of causing restrictive lung disease and pulmonary fibrosis in the general population. Dr. Grodzin has not presented any reliable or relevant scientific evidence to bolster this presumption. Instead, Dr. Grodzin essentially relied on the same scientific evidence and reached the same conclusions as Dr. Schlesinger. As we have explained, the district court did not abuse its discretion in excluding Dr. Schlesinger's opinion, thus negating Dr. Schlesinger's ability to satisfy the general causation requirement. Consequently, the fact that Dr. Grodzin conducted a differential diagnosis does not save his opinion from the same fate as Dr. Schlesinger's opinion. *Cf. Curtis*, 174 F.3d at 670 (“[S]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs’ burden in a toxic tort case.” (emphasis added) (quoting *Allen*, 102 F.3d at 199)). The district court did not abuse its discretion in excluding Dr. Grodzin's causation opinion because, irrespective of the differential diagnosis, Dr. Grodzin is unable to satisfy the general causation requirement.

IV.

After excluding the causation opinions of Dr. Schlesinger and Dr. Grodzin, the district court granted Arkema's motion for summary judgment because

No. 11-50193

Johnson could not “prove the causation necessary to support a claim under Texas law.” Johnson alleges that the district court erred in granting Arkema’s motion for summary judgment because: (1) there is a strong temporal connection supporting causation; (2) the symptoms experienced by other Owens Illinois’ employees provide additional circumstantial evidence of causation; and (3) Arkema’s expert pulmonologist conceded that tin oxide is known to cause scarring of the lung tissues.²⁰

This court reviews a summary judgment *de novo*, applying the same standard as the district court. *Trinity Universal Ins. Co. v. Emp’rs. Mut. Cas. Co.*, 592 F.3d 687, 690 (5th Cir. 2010). “Summary judgment should be affirmed if, viewing the evidence in the light most favorable to the non-moving party, there is no genuine dispute as to any material fact and the movant is entitled to judgment as a matter of law.” *Access Mediquip L.L.C. v. UnitedHealthcare Ins. Co.*, 662 F.3d 376, 378 (5th Cir. 2011) (citations and internal quotation marks omitted). Summary judgment must be entered “against a party who fails to make a showing sufficient to establish the existence of an element essential to that party’s case, and on which that party will bear the burden of proof at trial.” *Celotex Corp. v. Catrett*, 477 U.S. 317, 322 (1986).

A.

Johnson first argues that the strong temporal connection between his exposure to Certincoat and the onset of his symptoms offsets the need to present expert testimony to establish causation. Johnson relies on the Supreme Court

²⁰ In one sentence and without reference to any authority, Johnson also argues that summary judgment was inappropriate because “[s]ix treating physicians in Waco would see Johnson for his lung injury; all six physicians found that Johnson suffers from a lung condition caused by an acute exposure to chemical vapors from Arkema’s hood.” Assuming this argument is not waived, Johnson’s causation theory is nevertheless precluded by the general causation requirement. *See Knight*, 482 F.3d at 351 (The general causation standard concerns “whether a substance is capable of causing a particular injury or condition in the general population”).

No. 11-50193

of Texas’s decision in *Morgan v. Compugraphic Corporation*, which held that “[g]enerally, lay testimony establishing a sequence of events which provides a strong, logically traceable connection between the event and the condition is sufficient proof of causation.” 675 S.W.2d 729, 733 (Tex. 1984). Johnson argues that such a sequence exists in this case because “Johnson (1) had never smoked or had any history of asthma or lung disease prior to exposure, (2) worked within 2–3 feet of Arkema’s machine that was leaking chemical fumes, (3) was exposed to chemical fumes at a level far above the OSHA limit, (4) could see, smell and feel the chemical burning his throat and lungs, (5) suffered classic symptoms of exposure to the chemical, (6) was administered oxygen and transported to the emergency room after 2–3 hours of constant exposure, and (7) despite continuous medical treatment to reduce lung inflammation, suffered permanent scarring to his lung tissue.”

In its 2007 decision in *Guevara v. Ferrer*, the Texas Supreme Court summarized the meaning of *Morgan*.²¹ 247 S.W.3d 662 (Tex. 2007). The court first explained that “[t]he general rule has long been that expert testimony is

²¹ We have previously summarized the *Morgan* decision as follows:

In *Morgan*, the plaintiff suffered from frequent skin rashes and problems with her digestive and nervous systems, which she alleged were caused by her exposure to chemical fumes from a leaking typesetting machine at her workplace. The plaintiff testified that (1) she had always been in good health prior to the installation of the typesetting machine near her desk, (2) she worked with her face two inches from a typesetting machine that was leaking chemical fumes, (3) soon thereafter she experienced problems with breathing and swelling, and (4) after four or five days of constant exposure, she experienced watery eyes, blurred vision, headaches, and swollen breathing passages. The Texas Supreme Court held that this evidence established “a sequence of events from which the trier of fact may properly infer, without the aid of expert medical testimony, that the release of chemical fumes from the typesetting machine caused [the plaintiff] to suffer injury.”

Hamburger v. State Farm Mut. Auto. Ins. Co., 361 F.3d 875, 884–85 (5th Cir. 2004) (citations omitted).

No. 11-50193

necessary to establish causation as to medical conditions outside the common knowledge and experience of jurors.” *Id.* at 665. The court reiterated, however, that “non-expert evidence alone is sufficient to support a finding of causation in limited circumstances where both the occurrence and conditions complained of are such that the general experience and common sense of laypersons are sufficient to evaluate the conditions and whether they were probably caused by the occurrence.” *Id.* at 668–69. Such is generally the case when the lay testimony “establish[es] a sequence of events which provides a strong, logically traceable connection between the event and the condition.” *Id.* at 666 (quoting *Morgan*, 675 S.W.2d at 733).

In the underlying dispute in *Guevara*, the plaintiff had presented evidence at trial of: (1) the decedent’s condition before an automobile accident; (2) the accident itself; and (3) the decedent’s post-accident condition, including his numerous medical treatments.²² *Id.* at 667. The court found that such evidence could establish that the accident caused “basic physical conditions which (1) are within the common knowledge and experience of laypersons, (2) did not exist before the accident, (3) appeared after and close in time to the accident, and (4) are within the common knowledge and experience of laypersons, caused by automobile accidents.” *Id.* The court nevertheless reversed because the evidence

²² The decedent’s treatment and medical expenses included:

among other expenses, the cost of (1) at least two abdominal surgeries; (2) three separate confinements in health care facilities, one of which was for over three months; (3) a great variety and quantity of various pharmaceutical supplies, medicines, and drugs; (4) numerous varied laboratory procedures; (5) extensive treatments for respiratory failure and therapy; (6) physical therapy of various kinds; (7) treatments for kidney failure; and (8) a great assortment and quantity of “central supply” and miscellaneous medical charges.

Id. at 669.

No. 11-50193

was legally insufficient to support a finding that the automobile accident caused all of the medical expenses awarded by the jury:

Non-expert evidence of circumstances surrounding the accident and Arturo's complaints is sufficient to allow a layperson of common knowledge and experience to determine that *Arturo's immediate post-accident condition which resulted in his being transported to an emergency room and examined in the emergency room were causally related to the accident*. Thus, the evidence is legally sufficient to support a finding that some of his medical expenses were causally related to the accident. On the other hand, the evidence is not legally sufficient to prove what the conditions were that generated all the medical expenses or that the accident caused all of the conditions and the expenses for their treatment.

Id. at 669–70 (emphasis added). It remanded the case to the court of appeals for determination of appropriate remittiturs or, if necessary, a new trial. *Id.* at 670.

Here, Johnson's alleged chronic injuries, the severe restrictive lung disease and pulmonary fibrosis, did not develop shortly after the Certincoat exposure incidents but instead manifested in the years following the incidents. In light of *Guevara*, we conclude that this significant gap in time renders the fact-finder unable to evaluate the cause of Johnson's chronic lung disease based solely on its common sense and general experience. We, therefore, agree with the district court's conclusion that Johnson needs the assistance of experts to prove that his Certincoat exposure caused his chronic injuries.

On the other hand, Johnson's acute injuries—which immediately followed his exposure to Certincoat and precipitated an emergency room visit and at least two other doctors' office visits during the summer of 2007—are within those limited circumstances where expert opinion is unnecessary. *See id.* at 669–70; *see also Ballard v. Bunge N. Am Inc.*, 338 F. App'x 447, 448 (5th Cir. 2009) (Owen, J., concurring) (joined by Haynes, J.) (unpublished). Accordingly, the district court erred in granting summary judgment to Arkema regarding

No. 11-50193

Johnson's alleged acute injuries. We therefore reverse and remand, in part, for further proceedings concerning Johnson's alleged acute injuries.

B.

Johnson next argues that "the lower court erred in failing to address the fact that a number of other Owens Illinois employees suffered similar respiratory distress and lung injury as Johnson following similar acute exposures to Arkema's chemicals while working within a few feet of Arkema's hoods." Relying on *Curtis*, 174 F.3d at 669, Johnson argues that the symptoms of his co-workers provide other circumstantial evidence to corroborate his temporal proximity theory.

Johnson's reliance on our *Curtis* decision is misplaced. With regard to the reliability of an expert's causation opinion under *Daubert*, the *Curtis* court found that "a temporal connection is entitled to greater weight when there is an established scientific connection between exposure and illness or *other circumstantial evidence supporting the causal link*." *Id.* at 670 (emphasis added). However, the issue here does not require analysis of our *Daubert* jurisprudence; rather, the question is whether, under Texas law, Johnson can satisfy the element of causation without the assistance of an expert. Johnson has not cited any Texas case law indicating that evidence of similar injuries to others dispenses with the need for expert testimony in this toxic tort case. Accordingly, Johnson's reliance on his co-worker's alleged injuries does not support reversal of the district court's summary judgment.

C.

Finally, Johnson posits that summary judgment was inappropriate because Arkema's expert pulmonologist, Dr. Aris, testified that tin oxide can "cause lung injury and fibrosis." According to Johnson, Dr. Aris also referenced two human case studies indicating that "tin oxide is capable of and does cause the type of lung injury suffered by Johnson." Johnson further notes that other

No. 11-50193

human and animal studies of tin oxide show that it is capable of causing interstitial lung disease.

Even assuming that this evidence suffices to demonstrate that tin oxide is capable of causing restrictive lung disease, thus satisfying general causation, the evidence falls short of satisfying the requirement of specific causation. As discussed in III.B., *supra*, specific causation concerns whether “a substance caused a particular individual’s injury.” *Knight*, 482 F.3d at 351. Johnson does not offer any evidence that the actual amounts of tin oxide to which he was exposed were of a sufficient concentration level to cause his restrictive lung disease and pulmonary fibrosis. *See also Curtis*, 174 F.3d at 670 (“[S]cientific knowledge of the harmful level of exposure to a chemical, plus *knowledge that the plaintiff was exposed to such quantities*, are minimal facts necessary to sustain the plaintiffs’ burden in a toxic tort case.” (emphasis added) (quoting *Allen*, 102 F.3d at 199)). Thus, summary judgment was appropriate on this issue.

V.

For the foregoing reasons, we AFFIRM the district court’s judgment in all respects except as to Johnson’s claims regarding his acute injuries, on which we REVERSE and REMAND for further proceedings.

No. 11-50193

REAVLEY, Circuit Judge, concurring:

I agree that the summary judgment should be reversed, but I disagree with the ruling to deny the trier of fact the testimony of these highly qualified expert witnesses. There are fact issues, primarily the extent of exposure of the plaintiff to the chemical vapors and the diagnosis of his ailment. There may be a disagreement between the experts about what, if anything, it would take for inhalation of these vapors to damage the lungs to the extent of progressive disease. If that bears on the decision of the diagnosis and is in question, the trier of fact needs the assistance of these experts.

It is simply incredible to me to decide that Dr. Schlesinger is an unreliable source for any scientific question in this case. And the same is true of the other expert witnesses appearing in this record. But certainly Dr. Schlesinger is an eminent authority on respiratory toxicology and the study of the adverse effects of exposure to inhaled chemicals. Relying on the diagnosis made by Dr. Grodzin that the patient suffers from restrictive lung disease, Dr. Schlesinger explains the path of physiological response of the lungs inhaling these damaging vapors. And no one will deny that MBTC and hydrochloric acid, if inhaled to some extent, will damage the lungs. The majority in this opinion accept that testimony for “acute injuries,” but decide not to allow proof of any relation to progressive disease.

The district court excluded the testimony of Dr. Schlesinger because he could not cite fully tested and peer reviewed studies proving that hydrochloric acid can cause restrictive lung disease. That significant damage may be done is merely factual information. The possible extent of damage from breathing these chemicals may be at issue and will require the testimony of fully qualified and experienced experts, of which Dr. Schlesinger is surely one. There are no studies to meet the requirement of the district court, and that is not surprising. How would that study be designed and conducted, by

No. 11-50193

obtaining a large population of people to breathe this chemical vapor or that vapor in this volume or that volume, then to have their lung function tested and maybe biopsied? Where would so many persons be found to be subjected to this?

Studies that the district court required may prove helpful in determining the reliability of a particular scientific “theory or technique,” *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 113 S. Ct. 2786 (1993), but that is no checklist and reliability may depend on “the nature of the issue, the expert’s particular expertise, and the subject of his testimony.” *Kumho Tire Company v. Carmichael*, 119 S. Ct. 1167, 1175 (1999). The trial court erred in excluding testimony that is possibly relevant and clearly reliable.