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UNITED STATES COURT OF APPEALS

FOR THE SIXTH CIRCUIT

JEFF TAMRAZ; TERRY TAMRAZ, *Plaintiffs-Appellees,* *v.* LINCOLN ELECTRIC COMPANY; HOBART BROTHERS COMPANY; ESAB GROUP, INC. (08-4015); TDY INDUSTRIES, INC. (08-4016), *Defendants-Appellants.*	Nos. 08-4015/4016

Appeal from the United States District Court
for the Northern District of Ohio at Cleveland.
No. 04-18948—Kathleen McDonald O'Malley, District Judge.

Argued: November 18, 2009

Decided and Filed: September 8, 2010

Before: MARTIN and SUTTON, Circuit Judges; REEVES, District Judge.[*]

COUNSEL

ARGUED: Stephen J. Harburg, SKADDEN, ARPS, SLATE, MEAGHER & FLOM LLP, Washington, D.C., for Appellants. Paul Michael De Marco, WAITE, SCHNEIDER, BAYLESS & CHESLEY CO., L.P.A., Cincinnati, Ohio, for Appellees. **ON BRIEF:** Stephen J. Harburg, John H. Beisner, Jessica D. Miller, SKADDEN, ARPS, SLATE, MEAGHER & FLOM LLP, Washington, D.C., Jonathan D. Hacker, O'MELVENY & MYERS LLP, Washington, D.C., Irene C. Keyse-Walker, Joseph J. Morford, TUCKER ELLIS & WEST LLP, Cleveland, Ohio, for Appellants. John R. Climaco, John A. Peca, Jr., Dawn M. Chmielewski, Patricia M. Ritzert, CLIMACO, WILCOX, PECA, TARANTINO & GAROFOLI CO., LPA, Cleveland, Ohio, Eric C. Wiedemer, KELLEY & FERRARO LLP, Cleveland, Ohio, Elizabeth J. Cabraser, Robert Nelson, LIEFF CABRASER HEIMANN & BERNSTEIN, LLP, San Francisco, California, for Appellees.

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[*]The Honorable Danny C. Reeves, United States District Judge for the Eastern District of Kentucky, sitting by designation.

SUTTON, J., delivered the opinion of the court, in which REEVES, D. J., joined. MARTIN, J. (pp. 21–31), delivered a separate dissenting opinion.

OPINION

SUTTON, Circuit Judge. At issue in this case is the often-elusive line between admissible opinion and inadmissible speculation under Rule 702 of the Federal Rules of Evidence and *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). Several manufacturers of welding supplies appeal a $20.5 million jury verdict based on a doctor's testimony that their products triggered "manganese-induced parkinsonism" in a welder who used them. Because the district court exceeded its discretion in allowing this testimony, we reverse and remand for a new trial.

I.

From roughly 1979 to 2004, Jeff Tamraz worked as an independent-contracting welder in California. Beginning in about 2001, he began to suffer symptoms of Parkinsonism: tremors, drooling, a "masked face" and impaired coordination on his right side. JA 800–03.

In September 2004, Tamraz and his wife Terry sued several manufacturers of welding supplies, alleging that the fumes from their products had caused his condition and that labels on the products had failed to warn of the danger. The case was consolidated with ongoing multidistrict litigation in the Northern District of Ohio. *In re: Welding Fume Prods. Liab. Litig.*, No. 03-cv-17000, MDL No. 1535. The district court selected Tamraz's case for one of several bellwether trials to guide the resolution of the other cases. No. 03-cv-17000, R.2043 (June 6, 2007).

After summary judgment thinned the claims and defendants, Tamraz's case went to trial on three theories of relief (strict-liability failure to warn, negligent failure to warn and fraud by concealment) against five defendants (The Lincoln Electric Company, Hobart Brothers Company, The ESAB Group, Inc., BOC Group, Inc. and TDY

Industries, Inc.). The jury found for Tamraz and against all five defendants on the claims of strict liability and negligent failure to warn, but rejected his claim of fraud by concealment. It awarded Jeff Tamraz $17.5 million in compensatory damages and Terry Tamraz $3 million for loss of consortium. The defendants all filed motions to overturn the verdict under Rule 50 of the Federal Rules of Civil Procedure. The district court denied the challenges of every defendant save BOC Group, against whom the court found insufficient evidence to sustain the verdict. Lincoln Electric, the ESAB Group, Hobart Brothers and TDY Industries appealed.

II.

The manufacturers argue that the district court should not have admitted Dr. Walter Carlini's opinion that the manufacturers' products triggered "manganese-induced parkinsonism" in Tamraz, claiming it did not satisfy the requirements of Rule 702 of the Federal Rules of Evidence. We agree.

A.

The relevant law. Rule 702 says:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to . . . determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed. R. Evid. 702. The rule gives district courts a "gatekeeping role" in screening the reliability of expert testimony, *Daubert*, 509 U.S. at 597, and we review their decisions for abuse of discretion, *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152 (1999).

The relevant science. Doctors now recognize that what James Parkinson described nearly two centuries ago as "the shaking palsy" makes up a family of movement disorders encompassing Parkinson's Disease along with an assortment of other disorders. James Parkinson, *An Essay on the Shaking Palsy* (1817), *reprinted in* 14 J. Neuropsychiatry & Clin. Neurosci. 223 (2002); *see* JA 130–31. The disorders have

different causes, and they have different but overlapping symptoms, including tremors, instability and slowness and rigidity of movement. JA 130. Diagnosing one type of parkinsonism over another is no easy task. JA 140, 553–56.

Two forms of parkinsonism—Parkinson's Disease and manganism—matter here. Parkinson's Disease is the most common type, afflicting more than a million people in the United States alone. JA 131, 607. The typical individual with Parkinson's Disease suffers from a gradual loss of motor function and a tremor when at rest, both usually developing on one side of the body, caused by deterioration of neurons in a part of the brain called (bear with us) the substantia nigra pars compacta. JA 132, 135–36. The causes of Parkinson's Disease range from the obscure to the unknown. As a result, doctors and scientists often define Parkinson's Disease by its undetermined cause—"idiopathic Parkinson's Disease"—"idiopathic" being another way of saying the medical community does not know why a given individual has the disease. In other cases, they use a name other than Parkinson's Disease—such as postencephalitic parkinsonism, drug-induced parkinsonism, or toxin-induced parkinsonism—when they know the cause. JA 131, 150. Over time, as scientists have discovered more genetic and other causes for Parkinson's Disease, the medical profession has defined more sub-classifications of the disease and has had to rely less frequently on "idiopathic" designations. JA 130–32.

Manganism is a form of parkinsonism defined by its cause: overexposure to manganese, a hard and brittle element that resembles iron but is not magnetic. The symptoms of manganism overlap with Parkinson's Disease but include an action tremor instead of a rest tremor, symmetry of symptoms and a distinct gait ("cock walk"). JA 584, 871, 1002–05. The typical manganism patient suffers neuron deterioration in a different part of the brain from the typical Parkinson's Disease patient—medically speaking, the globus pallidus and the substantia nigra pars reticulata, not the substantia nigra pars compacta—and therapies used to treat Parkinson's Disease often do not work with manganism. JA 134–36, 564–67.

The diagnosis of Jeff Tamraz. Every doctor to examine Jeff Tamraz has reached a different conclusion about where his case fits into this puzzle. No one disputes that he suffers from parkinsonism; the question is what kind and from what cause. The first neurologist to see Tamraz, Dr. Michael Siegel, initially diagnosed Tamraz with "an unusual form of Parkinson's disease due to manganese poisoning," JA 805, but then became less sure about the role of manganese after he consulted medical literature on the subject, JA 808–09. He eventually concluded that Tamraz's condition is closer to Parkinson's Disease than to manganism: Tamraz had a rest tremor, asymmetry of symptoms and no "cock walk." JA 809–11, 816–19. Although he could not rule out manganese as the cause of the illness, JA 812, Dr. Siegel believed Tamraz's parkinsonism likely resulted from "factors other than exposure to manganese," JA 817.

Tamraz's second neurologist, Dr. Carlini, the witness at issue here, concluded that Tamraz suffers from "manganese-induced parkinsonism," JA 615, but not in the sense of a manifestation of manganism, as that phrase is sometimes used, *see* JA 600–01. He believed that manganese exposure caused something akin to Parkinson's Disease in Tamraz. JA 600. He found many of the same symptoms that led Dr. Siegel to suspect Parkinson's Disease rather than manganism, JA 616, 619–21, but he noted that scientists recently had discovered genetic or environmental causes for many forms of Parkinson's Disease formerly considered idiopathic, JA 599, 619, and discussed literature raising the possibility that genetics and environmental factors may cause a large fraction of Parkinson's Disease cases. JA 599, 601–02. Dr. Carlini hypothesized that Tamraz might have a genetic predisposition to Parkinson's Disease, and that manganese in lower levels than necessary to cause manganism might nevertheless "trigger" the symptoms of Parkinson's Disease, like "the straw that broke the camel's back." JA 598–99. He did not believe Tamraz has Parkinson's Disease in the strict sense—that manganese in his view caused the disease meant by definition it could not be "idiopathic" Parkinson's Disease—but believed it to be otherwise identical to Parkinson's Disease. JA 599–600.

In addition to Tamraz's treating physicians, the plaintiffs and defendants each hired a doctor to examine Tamraz. Dr. Anthony Lang, the defendants' doctor, testified

that "Parkinson's disease and manganism . . . are quite distinct and different," JA 1300, and concluded, based on his examination of Tamraz, that Tamraz has Parkinson's Disease, not manganism, *see* JA 467. Dr. Paul Nausieda, the plaintiffs' doctor, testified that Tamraz does not have Parkinson's Disease but "a manganese-induced movement disorder," essentially manganism. JA 867–68. He relied on the absence of other explanations for Tamraz's parkinsonism and its early onset and, in contrast to the other doctors, found that Tamraz has some symptoms more indicative of manganism than of Parkinson's Disease. *See* JA 866–73.

B.

The manufacturers do not question one aspect of Dr. Carlini's testimony—that Tamraz suffers from a form of parkinsonism. They dispute his conclusion that manganese exposure caused the illness. L.E.C. Br. at 24. To put the distinction in medical terms, they challenge Dr. Carlini's etiology (what caused the disorder diagnosed?), not his diagnosis (what disorder caused the set of symptoms observed?).

The problem here is that, when Dr. Carlini testified that manganese exposure caused Tamraz's condition, he went beyond the boundaries of allowable testimony under Rule 702. In the video-taped deposition played at trial, Dr. Carlini opined that Tamraz has "manganese-induced parkinsonism" "with a reasonable degree of medical certainty." JA 615. But the etiological component of this conclusion—the "manganese-induced" part—was at most a working hypothesis, not admissible scientific "knowledge." Fed. R. Evid. 702. Because the "knowledge" requirement of Rule 702 requires "more than subjective belief or unsupported speculation," *Daubert*, 509 U.S. at 590, the testimony should have been excluded.

The rest of Dr. Carlini's testimony confirms the speculative nature of this opinion. Under questioning by Tamraz's counsel, Dr. Carlini focused on his diagnosis of parkinsonism and barely explained why he thought manganese caused the disease. He stated only that he diagnosed him with "manganese-induced parkinsonism" because "that seemed the most likely explanation for his early onset parkinsonism," based on his "clinical examination," "Mr. Tamraz's history" "[a]nd just general experience and

knowledge about movement disorders." JA 615. Questioning by the manufacturers' counsel brought to the surface his line of reasoning: (1) Tamraz was exposed to welding fumes presumably containing manganese, JA 613; (2) he developed the symptoms of Parkinson's Disease (though not those of manganism), JA 604, 613–14, 616; (3) scientists have identified genetic factors that cause some forms of otherwise "idiopathic" Parkinson's Disease, JA 599; (4) some literature has hypothesized that toxins combined with genetics may cause other cases of Parkinson's Disease, JA 599, 601; (5) manganese is known to cause manganism, so it would be a possible candidate for triggering Parkinson's Disease as well, JA 601; (6) Tamraz may have the genes for Parkinson's Disease, JA 621; and (7) manganese may have triggered these genes and given Tamraz parkinsonism, JA 615.

That is a plausible hypothesis. It may even be right. But it is no more than a hypothesis, and it thus is not "knowledge," nor is it "based upon sufficient facts or data" or the "product of reliable principles and methods . . . applied . . . reliably to the facts of the case." Fed. R. Evid. 702.

Dr. Carlini acknowledged the speculative jumps involved in steps 4, 5 and 6 of this chain of causation—the steps necessary to his theory that manganese exposure may cause Parkinson's Disease in general. At step 4, he described the literature hypothesizing a link between environmental toxins and latent genetic Parkinson's Disease as "all theoretical." JA 599; *see also* JA 621 ("theoretical writing"). At step 5, he conceded he knew of no studies finding a link between manganese and Parkinson's Disease and that "studies that have looked at that . . . have not found a very strong correlation." JA 602; *see* JA 597, 599, 605, 621; *see also* JA 623 ("Epidemiological studies have failed to find a correlation between manganese and Parkinson's disease."). At step 6, he conceded that "speculation" led him to guess that Tamraz had "an underlying predisposition to Parkinson's disease," JA 621, even though Tamraz has no family history of Parkinson's Disease, JA 613. A negative answer at any one of these steps would defeat his overall theory of causation. The reality that all of them were speculative makes the theory speculative three times over. *Cf. Siharath v. Sandoz*

Pharm. Corp., 131 F. Supp. 2d 1347, 1371 (N.D. Ga. 2001) ("Three scientifically unwarranted 'leaps of faith' exist in this causal chain."), *aff'd*, 295 F.3d 1194 (11th Cir. 2002).

The final step required a leap of faith as well, even ignoring the jumps required to get there. That manganese *could cause* Parkinson's Disease in someone like Tamraz does not show that manganese *did cause* Tamraz's Parkinson's Disease. Parkinson's Disease occurs commonly in the general population and usually without any known cause. Any given case of Parkinson's Disease thus might have occurred regardless of the manganese exposure, making it hard to attribute one case to manganese over all of the other possible causes. *See Bland v. Verizon Wireless, (VAW) L.L.C.*, 538 F.3d 893, 897 (8th Cir. 2008); *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1228–39 (D. Colo. 1998). This attribution is harder still if, as Dr. Carlini hypothesized, Tamraz already had a genetic predisposition toward it, and even more so if, as Dr. Carlini also acknowledged, the base probability of getting parkinsonism from such a predisposition is unknown. *See* JA 601 (acknowledging that he did not know whether someone with a genetic predisposition toward Parkinson's Disease, as he suspected Tamraz had, would have a 90% chance or a 10% chance of manifesting symptoms).

Dr. Carlini never explained how he made this leap—how *this* case stemmed from manganese exposure. When asked how to "tell the difference between a welder with idiopathic Parkinson's disease and a welder . . . tipped into the Parkinson's disease by welding," he answered with tests he *might* do, not tests he had done. JA 602. Asked similar questions twice more, he responded twice more by listing tests he could do, not tests he had done. *See* JA 603 ("[Y]ou would suspect . . . subtle differences . . . which I bet would be possible to tease out if we had . . . some of these more advanced imaging techniques."); JA 622 ("[W]e haven't tried yet. . . . Suppose—let's just take a hypothetical. . . ."). The closest he came to explaining why he suspected that manganese exposure caused Tamraz's parkinsonism is when he noted its "early onset." JA 604, 615. But he also said that roughly ten percent of people with Parkinson's develop symptoms before age 50, JA 607, which, considering the high prevalence of Parkinson's

Disease, does not create an inference that something particularly unusual must have caused it in Tamraz, who was 41 to 44 years old at the onset of his symptoms, JA 467. (Other witnesses suggested that the frequency of early onset is less than ten percent, *see, e.g.*, JA 573, but that has no relevance to *Dr. Carlini's* methodology.) Dr. Carlini's testimony thus suffers from a lack of foundation both for why manganese could cause Parkinson's Disease and why manganism caused this case of Parkinson's Disease.

Under these circumstances, it makes no difference that Dr. Carlini purported to find "manganese-induced parkinsonism" in Tamraz "with a reasonable degree of medical certainty." JA 615. Whatever Dr. Carlini understood by "with a reasonable degree of medical certainty," the phrase—the conclusion by itself—does not make a causation opinion admissible. The "*ipse dixit* of the expert" alone is not sufficient to permit the admission of an opinion. *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997). Minus that one phrase, nothing in his testimony suggests the sort of "knowledge" on this point that the Rules require—only speculation, which is generally inadmissible. "[N]o matter how good" experts' "credentials" may be, they are "not permitted to speculate." *Goebel v. Denver & Rio Grande W. R.R. Co.*, 215 F.3d 1083, 1088 (10th Cir. 2000). Dr. Carlini may be a "distinguished" doctor, and "his conjecture" about causation may be "worthy of careful attention . . . . But the courtroom is not the place for scientific guesswork, even of the inspired sort." *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996).

Rule 702, we recognize, does not require anything approaching absolute certainty. *See Daubert*, 509 U.S. at 590. And where one person sees speculation, we acknowledge, another may see knowledge, which is why the district court enjoys broad discretion over where to draw the line. *See Joiner*, 522 U.S. at 139. Yet, so long as there is a line, some forms of testimony may cross it, and that happened here. Dr. Carlini's opinion contains not just one speculation but a string of them: A suggests by analogy the possibility of B, which might also apply to C, which, if we speculate about D, could eventually trigger E, so perhaps that happened here. At some point, the train becomes too long to pull and the couplings too weak to hold the cars together.

C.

Tamraz resists this conclusion on a number of grounds, all unconvincing. He first turns to Dr. Carlini's use of the phrase "manganese-induced parkinsonism" to describe Tamraz's condition, JA 615, suggesting that Dr. Carlini equates the phrase with manganism, Tamraz Br. at 33 & n.7. If manganism, as its name implies, is caused by manganese, and if Dr. Carlini diagnosed Tamraz with manganism, the thinking goes, the required link between manganese exposure and the disease caused by it has been solved.

The problem is that this argument mischaracterizes Dr. Carlini's testimony. Although some people use "manganese-induced parkinsonism" to refer to manganism, *see, e.g.*, JA 862, Dr. Carlini did not. He used the phrase to mean Parkinson's Disease that happens to have manganese exposure as its cause:

> Q So you use the term manganese-induced parkinsonism to mean the same disease as Parkinson's disease?
>
> A It's the same disease, yes. It's just triggered by manganese. . . . It's a very specific form of the same disease in the sense that I'm implying that it's triggered by a certain environmental toxin as opposed to another.
>
> Q But it's pathologically and clinically Parkinson's disease?
>
> A It's different, yes, than manganism. Exactly. Pathologically it might look exactly the same as sporadic idiopathic Parkinson's disease, yes.

JA 600. "[E]very aspect" of Tamraz's condition, he added, "is consistent with a diagnosis of Parkinson's disease." JA 607. He used a different name for the disease because idiopathic Parkinson's Disease by definition has no cause, so once he assigned a cause to the disease Dr. Carlini also had to assign a name to that "specific form of the same disease": "manganese-induced parkinsonism." JA 600. But the naming did not change the underlying diagnosis. He repeatedly emphasized that he saw none of the symptoms of manganism in Tamraz and that his diagnosis was identical to Parkinson's Disease save for surmise about its cause. *See* JA 599–600, 616; *see also* JA 601 ("He looks like [idiopathic Parkinson's Disease] clinically."); JA 618 ("Tamraz does not have

manganism."). Tamraz's expert, Dr. Nausieda, who used "manganese-induced parkinsonism" nearly synonymously with manganism, acknowledged that Dr. Carlini did not mean the same thing. JA 862.

In denying the manufacturers' motion to exclude Dr. Carlini's causation testimony, the district court noted that the manufacturers "ask[ed] the Court to draw bright lines regarding diagnoses of movement disorders [i.e., between Parkinson's Disease and manganism] that I have already declined to draw." JA 170–71. But it was not just the manufacturers drawing these lines; Dr. Carlini himself called manganism and Parkinson's Disease "very distinctive" diseases and found Tamraz's symptoms lined up with Parkinson's Disease, not manganism. JA 607. Dr. Carlini's opinion cannot escape its own logic.

In conflating "manganese-induced parkinsonism" with manganism, Tamraz conflates diagnosis with etiology, eliding the distinction between Tamraz's disease and what caused it. Diagnosis *and* etiology, however, both were in play in this case. Because Dr. Carlini diagnosed Tamraz with something akin to Parkinson's Disease, not manganism, and because Parkinson's Disease unlike manganism has no standard etiology, Dr. Carlini's etiology must rise or fall on its own.

To use an analogy, chronic shortness of breath may be caused by diseases ranging from emphysema to lung fibrosis to bronchitis to heart disease—which would be the diagnosis. Heart disease, to pick one of these diagnoses, may be caused by diet, smoking, genetics or some combination of the three—which would be the etiology. One could not defend a verdict without linking the etiology to the diagnosis. *Cf. Kelley v. Am. Heyer-Schulte Corp.*, 957 F. Supp. 873, 882 (W.D. Tex. 1997) ("Essentially, this is a bit like saying that if a person has a scratchy throat, runny nose, and a nasty cough, that person has a cold; if, on the other hand, that person has a scratchy throat, runny nose, nasty cough, and wears a watch, they have a watch-induced cold.").

Tamraz likewise conflates a doctor's expertise in diagnosis with a doctor's expertise in etiology, arguing for the reliability of Dr. Carlini's causation testimony because of his "extensive . . . experience" with diagnosing parkinsonism. Appellees'

Response to Rule 28(j) Letter (Nov. 9, 2009). But most treating physicians have more training in and experience with diagnosis than etiology. *See* David L. Faigman, *Judges as "Amateur Scientists"*, 86 B.U. L. Rev. 1207, 1221–22 (2006); Edward J. Imwinkelried, *The Admissibility and Legal Sufficiency of Testimony About Differential Diagnosis (Etiology)*, 56 Baylor L. Rev. 391, 405 (2004); Mary Sue Henefin et al., Reference Guide on Medical Testimony, in *Reference Manual on Scientific Evidence* 439, 471–72 (2d ed. 2000). When physicians think about etiology in a clinical setting, moreover, they may think about it in a different way from the way judges and juries think about it in a courtroom. *See Siharath*, 131 F. Supp. 2d at 1371–73. Getting the diagnosis right matters greatly to a treating physician, as a bungled diagnosis can lead to unnecessary procedures at best and death at worst. *See Bowers v. Norfolk S. Corp.*, 537 F. Supp. 2d 1343, 1361 (M.D. Ga. 2007). But with etiology, the same physician may often follow a precautionary principle: If a particular factor *might* cause a disease, and the factor is readily avoidable, why not advise the patient to avoid it? Such advice—telling a welder, say, to use a respirator—can do little harm, and might do a lot of good. *See* Joe G. Hollingsworth & Eric G. Lasker, *The Case Against Differential Diagnosis:* Daubert, *Medical Causation Testimony, and the Scientific Method*, 37 J. Health L. 85, 98 (2004). This low threshold for making a decision serves well in the clinic but not in the courtroom, where decision requires not just an educated hunch but at least a preponderance of the evidence.

None of this means that physicians may not testify to etiology—we have reversed courts for not allowing such testimony, *see, e.g.*, *Hardyman v. Norfolk & W. Ry. Co.*, 243 F.3d 255, 260–67 (6th Cir. 2001)—only that courts must apply the *Daubert* principles carefully in considering it. "The ability to diagnose medical conditions is not remotely the same . . . as the ability to deduce . . . in a scientifically reliable manner, the causes of those medical conditions." *Gass v. Marriott Hotel Servs., Inc.*, 501 F. Supp. 2d 1011, 1019 (W.D. Mich. 2007), *rev'd on other grounds*, 558 F.3d 419 (6th Cir. 2009). Doctors thus may testify to both, but the reliability of one does not guarantee the reliability of the other.

Nor can Dr. Carlini's testimony be defended as a permissibly admissible "differential diagnosis." A differential diagnosis seeks to identify the disease causing a patient's symptoms by ruling in all possible diseases and ruling out alternative diseases until (if all goes well) one arrives at the most likely cause. *See Hardyman*, 243 F.3d at 260–61. We have accepted this kind of testimony before. *See Glaser v. Thompson Med. Co.*, 32 F.3d 969, 977 (6th Cir. 1994).

The manufacturers, however, do not challenge Dr. Carlini's differential diagnosis, which concluded that Tamraz suffers from parkinsonism similar to classical Parkinson's Disease; they challenge his etiology that manganese caused it. Many courts, including our own, allow experts to employ a rule-in/rule-out reasoning process for etiology as well as diagnosis—essentially, a "differential etiology," though the term seems to be a legal one rather than a medical one. *See McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1252 (11th Cir. 2005); Henefin et al., *supra*, at 444, 481. This court's opinions have used "differential diagnosis" broadly to include what might better be called "differential etiology," but they have not had to distinguish the two concepts because most cases involve just one of them. *See, e.g.*, *Hardyman*, 243 F.3d at 259 n.2 (parties did not dispute the nature of the disease, only what caused it).

Whether we describe Dr. Carlini's causation methodology as "differential etiology" or "differential diagnosis," that does not make it reliable. "[S]imply claiming that an expert used the 'differential diagnosis' method is not some incantation that opens the *Daubert* gate." *Bowers*, 537 F. Supp. 2d at 1360. Calling something a "differential diagnosis" or "differential etiology" does not by itself answer the reliability question but prompts three more: (1) Did the expert make an accurate diagnosis of the nature of the disease? (2) Did the expert reliably rule in the possible causes of it? (3) Did the expert reliably rule out the rejected causes? If the court answers "no" to any of these questions, the court must exclude the ultimate conclusion reached. *See Best v. Lowe's Home Ctrs., Inc.*, 563 F.3d 171, 179 (6th Cir. 2009). Dr. Carlini's opinion fails the last two prongs because, for the reasons already given, his efforts to "rule in" manganese exposure as a

possible cause or to "rule out" other possible causes turned on speculation, not a valid methodology. No matter the label, the testimony does not satisfy Rule 702.

All of this suffices to distinguish the cases on which Tamraz relies to admit "differential diagnosis" testimony. In *Hardyman*, the trial court excluded a doctor's opinion that a railroad brakeman's job activities caused his carpal tunnel syndrome (CTS), finding the testimony unreliable because, although the doctor showed that tasks like those the brakeman performed are known to cause CTS, he cited no studies performed on brakemen and could not quantify how much movement and pressure would lead to how much CTS. 243 F.3d at 261–65. We reversed, holding that the district court demanded too much specificity and too much quantification from the expert. *Id.* at 262, 265. But here the problem is not that Dr. Carlini failed to cite studies about manganese causing Parkinson's Disease in welders or could not quantify how much manganese would lead to how much Parkinson's Disease; the problem is that he failed to cite *any* non-speculative evidence for his conclusion that manganese causes Parkinson's Disease.

Likewise, in *Best*, we reversed the district court for excluding a doctor's testimony that a chemical spill on Best's face caused him to lose his sense of smell. 563 F.3d at 183–84. In that case, Best suffered burns on his skin and irritation to his nasal passages immediately after the incident, and eventually lost his ability to smell altogether. *Id.* at 174. We approved the doctor's method of ruling in the chemical in question by making a careful comparison with similar chemicals he had known to have the same effect. *Id.* at 181. In this case, by contrast, Dr. Carlini analogized only to the "theoretical" possibility of other toxins causing Parkinson's Disease, JA 599, and did not point to any similar elements known to cause Parkinson's Disease. (He did mention one chemical known to cause parkinsonism—the designer drug MPTP—but never attempted to compare it with manganism and did not factor it into his etiology, JA 619.) In *Best*, the doctor also reliably ruled out most alternative causes; the defendant argued that the doctor also should have ruled out another possible factor but did not provide any evidence that this factor could cause the disease. 563 F.3d at 181. Here, though, the

other possibility—unknown (idiopathic) causation—currently accounts for the vast majority of Parkinson's Disease cases, making it impossible to ignore and difficult to rule out. *See Bland*, 538 F.3d at 897. Forecasting today's decision, *Best* cautioned that "[n]ot every opinion that is reached via a differential-diagnosis method will meet the standard of reliability required by *Daubert*." 563 F.3d at 179.

Tamraz invokes the testimony of Dr. Nausieda, who also testified that manganese exposure caused Tamraz's sickness with no objection from the manufacturers. If Dr. Nausieda's testimony passes muster, Tamraz claims, so too should Dr. Carlini's. Tamraz Br. at 35. But we are aware of no authority, and Tamraz points to none, holding that preserving an objection to one witness requires objecting to every similar witness. Although Dr. Nausieda's testimony in some ways overlapped with Dr. Carlini's, moreover, he reached opposite conclusions on the relevant points: He believed that Tamraz suffers from something akin to manganism, not Parkinson's Disease, JA 866–73, and he believed manganese exposure could not cause Parkinson's Disease. JA 951. Perhaps most importantly, even to the extent that Dr. Nausieda's testimony was consistent with parts of Dr. Carlini's, that would not make Dr. Carlini's testimony admissible. The important thing is not that experts reach the right conclusion, but that they reach it via a sound methodology. *See Daubert*, 509 U.S. at 595. Comparisons between methodologies no doubt may be instructive, and an expert may in some circumstances rely on other experts' testimony, *see* Fed. R. Evid. 703—something Dr. Carlini did not do here. But testimony still must be judged by its methodology, not its conclusion.

Dr. Carlini's speculation that Tamraz might have damage to the globus pallidus in his brain—noteworthy because globus pallidus damage characterizes manganism, not Parkinson's Disease, Tamraz Br. at 33 & n.7—is beside the point. Dr. Carlini primarily expected to see cell deterioration in the substantia nigra pars compacta, which Parkinson's Disease characteristically damages. JA 601. That Dr. Carlini guessed Tamraz *also* might have damage in the globus pallidus—and it was nothing more than a guess, *see* JA 601 ("this is all highly speculative obviously")—neither undermines his

diagnosis nor supports his etiology. Globus pallidus damage would be relevant only if Dr. Carlini had actually *detected* globus pallidus damage. That, however, was not the case. There is "too great an analytical gap between the data and the opinion proffered" for the court to admit Dr. Carlini's opinion as testimony. *Joiner*, 522 U.S. at 146.

D.

Having concluded that Dr. Carlini's causation testimony exceeded the permissible boundaries of Rule 702, we must reverse unless we can "say, with fair assurance, . . . that the judgment was not substantially swayed by the error." *Mike's Train House, Inc. v. Lionel, L.L.C.*, 472 F.3d 398, 409–10 (6th Cir. 2006). The error was not harmless.

The emphasis Tamraz put on Dr. Carlini's testimony confirmed its importance. His counsel argued to the jury that "Dr. Carlini alone proves by a preponderance of the evidence that this man has managnese-induced parkinsonism." JA 1407. In their opening argument, their closing argument and again in their rebuttal to the manufacturers' closing argument, counsel for Tamraz played the portion of the video deposition in which Dr. Carlini stated that Tamraz has "[m]anganese-induced parkinsonism" and stated that he held that belief "with a reasonable degree of medical certainty." JA 1317, 1374, 1395. Tamraz's attorney emphasized this supposed certainty: "They are going to tell you he is talking about speculation and conjecture. He is talking about a reasonable degree of medical certainty. . . . That is the standard, and he believes in it." JA 1374–75.

Counsel for Tamraz also leaned heavily on Dr. Carlini's apparent neutrality, calling him a man who "doesn't have a dog in this hunt," JA 1397, and repeatedly mentioning that Dr. Carlini received no payment for his testimony, unlike the manufacturers' only expert, *see* JA 1317, 1374–75, 1399–1400, 1411. They also emphasized Dr. Carlini's treating role: "regardless of what is decided in this case, Jeff Tamraz is going to fly back home, and next month he is going to walk into Dr. Carlini's office for his December scheduled appointment, and they are going to sit down and talk

about how to handle his manganese-induced Parkinsonism. That's the diagnosis." JA 1400–01.

Tamraz argues that Dr. Carlini's testimony could not be harmful because Dr. Nausieda also testified that Tamraz had "manganese-induced parkinsonism," so the jury would have heard the same conclusion even without Dr. Carlini's testimony. But, as shown, Dr. Nausieda and Dr. Carlini meant different things by the phrase "manganese-induced parkinsonism." *Compare* JA 600, *with* JA 862. Dr. Carlini was the only expert who testified that Tamraz had the equivalent of Parkinson's Disease caused by manganese. Without Dr. Carlini, Tamraz would have had to convince the jury that Tamraz suffered from manganism, not Parkinson's Disease. With Dr. Carlini's testimony, however, the jury faced three choices, two of which helped Tamraz: Tamraz won if he had manganism (as Dr. Nausieda testified) *or* Parkinson's Disease caused by manganese (as Dr. Carlini testified); the manufacturers won only if Tamraz had Parkinson's Disease not caused by manganese. Dr. Carlini's testimony thus shifted the primary question from what disease Tamraz had to whether manganese caused it.

The plaintiffs' closing argument accordingly played down the differences between manganism and Parkinson's Disease and played up the causation issue, noting that three out of four neurologists who had examined Tamraz opined one way or another that manganese had caused Tamraz's illness, which "could be a textbook example in law school of preponderance of the evidence. There is more evidence in front of you that his disease was caused by manganese in welding fumes than [that it wasn't]. That is our burden." JA 1407–08; *see also* JA 1375 ("The only neurologist of the four that is telling you that 20-plus years of inhaling manganese . . . has nothing to do with his condition is the one they hired and the one that they paid."). Given the importance of Dr. Carlini's testimony, we cannot say "with fair assurance" that the result would have been the same without it.

Our ruling, however, is a narrow one. The manufacturers here do not challenge the district court's primary *Daubert* ruling on Parkinson's Disease testimony, *In re Welding Fume Prods. Liab. Litig.*, No. 03-cv-17000, 2005 WL 1868046, at *22–37

(N.D. Ohio Aug. 8, 2005), and so we do not decide whether other experts may testify that manganese exposure causes Parkinson's Disease. We simply hold that the causation analysis in Dr. Carlini's deposition fell short of what *Daubert* requires.

We leave it to the able district court judge on remand presiding over this difficult case to decide whether to (1) present Dr. Carlini's deposition minus his attribution of Tamraz's illness to manganese (as the manufacturers requested below, JA 366); (2) exclude Dr. Carlini's depositions altogether if the court determines it cannot or should not sever his purely diagnostic conclusions from his etiological hypothesis (as Tamraz suggested, in the alternative, below, JA 461); (3) allow the parties to redepose Dr. Carlini; or (4) opt for any other amenable solution. All we conclude is that his testimony should not have been admitted as it was.

* * * * *

No one should construe this opinion as criticism of Dr. Carlini, whom the deposition shows to be intelligent and knowledgeable about the subject matter—immeasurably more so than we are. But not everything a knowledgeable person says is "knowledge" under Rule 702, no more than everything a scientist says is "scientific." "[A] district court judge asked to admit scientific evidence must determine whether the evidence is genuinely scientific, as distinct from being . . . speculation offered by a genuine scientist." *Rosen*, 78 F.3d at 318. This causation opinion fell into the latter category and therefore should have been excluded.

The sort of hypothesis Dr. Carlini presented can play a valuable role both in medicine, where, if the costs of action are low, doctors may want to act on hypotheses without further support, and in science generally, where all discoveries start as untested hypotheses. From this perspective, criticizing Dr. Carlini's hypothesis for being speculative would be like criticizing a sapling for being short. Some hypotheses become scientific theories and others do not.

But that is not the issue. The issue is the reliability of his opinion from a *legal* perspective. And what science treats as a useful but untested hypothesis the law should

generally treat as inadmissible speculation. As the Supreme Court has explained, "[t]he scientific project is advanced by broad and wide-ranging considerations of a multitude of hypotheses, for those that are incorrect will eventually be shown to be so . . . . Conjectures . . . are of little use, however, in the project of reaching a quick, final, and binding legal judgment—often of great consequence—about a particular set of events in the past." *Daubert*, 509 U.S at 597. "Law lags science; it does not lead it." *Rosen*, 78 F.3d at 319.

This is an imperfect system, to be sure. Both sides agree that Mr. Tamraz is a good man who suffers from a terrible disease; we now force him to take the chance of prevailing at trial a second time, with less evidence than before. If he does not, yet it turns out ten years from now that manganese causes his disease, that result will seem unfair. But the alternative route—allowing the law to get ahead of science—would be just as unfair. Such an approach would destroy jobs and stifle innovation unnecessarily. *See Joiner*, 522 U.S. at 148–49 (Breyer, J., concurring); *see also, e.g.*, Gina Kolata, *Panel Confirms No Major Illness Tied to Implants*, N.Y. Times, June 21, 1999, at A1 (describing how scientists concluded, after years of litigation, billions in settlements and the bankruptcy of a major manufacturer, that no evidence tied breast implants to health problems). Rule 702 at all events has drawn the line for us, and we must enforce it. *See Daubert*, 509 U.S. at 597. Because this testimony crossed that line, we reverse.

III.

In view of our decision to vacate the jury verdict, we need not reach the other arguments raised on appeal. We note, however, that the contours of the parties's dispute about the propriety of a "sophisticated user" jury instruction have changed in view of intervening law. After trial, the California Supreme Court endorsed and clarified the defense, *see Johnson v. Am. Standard, Inc.*, 179 P.3d 905 (Cal. 2008), and as a result the district court may wish to consider this new authority in determining the propriety of such an instruction at a new trial.

IV.

For these reasons, we reverse and remand for a new trial.

DISSENT

BOYCE F. MARTIN, JR., Circuit Judge, dissenting. The majority finds that the district court abused its discretion in admitting Dr. Walter Carlini's testimony because it "went beyond the boundaries of allowable testimony under rule 702" (*ante* at 6), because it was "speculative" (*ante* at 6), and because his deductions required "leaps of faith." (*Ante* at 8.) Because the majority reached this conclusion by acting as sitting judges rather than under the proper standard of review, I respectfully dissent.

I.

Jeff Tamraz's case is part of a larger multi-district litigation regarding inhalation of manganese fumes by welders. *In re Welding Rod Prods. Liab. Litig.*, 269 F. Supp. 2d 1365 (J.P.M.L. 2003). In April, May, and June 2005, the multi-district litigation court conducted three weeks of *Daubert* hearings to test the methodologies of expert witnesses. As part of the hearing, experts in the neurological community testified regarding the connection between manganese exposure and various forms of parkinsonism. The court also heard argument on the defendants' motion to preclude evidence that manganese exposure causes Parkinson's Disease. The trial court concluded that the evidence proffered was "sufficiently reliable to support the assertion that exposure to welding fumes can cause, contribute to, or acclerate a parkinsonian syndrome that some doctors can diagnose as [Parkinson's Disease] . . . at least in the abstract, as the question is presented here." (Corrected J.A. at 166, Order, Aug. 6, 2005).

Jeff Tamraz worked as a welder from 1979 to 2004. Around 2000-2001, he began experiencing severe neurological symptoms, which eventually became so severe that he could not care for himself. In July 2007, Tamraz brought suit in the United States District Court for the Northern District of Ohio against the five defendants who provided the welding materials that Tamraz used during his welding career. He claimed that his neurological injuries, which manifested symptoms consistent with Parkinson's Disease, were caused by manganese exposure.

Before trial, the defendants moved to exclude parts of the testimony to be presented by one expert, Dr. Carlini. On November 1, 2007, the court ruled on the motion, stating:

> I have read all of the briefs. I have read . . . the two depositions that were taken of Dr. Carlini. I have gone back and reread the Court's *Daubert* opinion, which was on the main MDL docket . . . and I have decided that I am going to deny the defendants' motion.
>
> To a large extent, the defendants' motion asks the Court to draw bright lines regarding diagnoses of movement disorders that I have already declined to draw, and I have already decided that the current state of the science does not require to be drawn. . . . I see nothing about [Dr. Carlini's] methodology that is either flawed or inconsistent with the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used and have described as appropriate diagnostic methods. . . . It is clear that the defendants have fair grounds to attack the somewhat unusual diagnosis that Dr. Carlini renders in this case . . . but that to me goes to the weight and not the admissibility of his testimony.

(Corrected J.A. at 170-173, Tr. of Proceedings, Nov. 1, 2007).

At trial, four experts testified regarding the cause of Tamraz's injury. Tamraz's primary medical expert, Dr. Paul Nausieda, testified that Tamraz suffered from manganese-induced parkinsonism. The defendants' lead medical expert, Dr. Anthony Lang, while testifying that manganese exposure can cause parkinsonism, stated that he did not believe that Tamraz's parkinsonism was caused by manganese exposure. Tamraz's former treating neurologist, Dr. Michael Siegel, first diagnosed Tamraz with an unusual form of Parkinson's Disease due to manganese poisoning. He later revised his opinion to state that, while he could not rule out the possibility than manganese exposure caused Tamraz's injury, Tamraz likely suffered from parkinsonism resulting from factors other than manganese exposure. Finally, Dr. Carlini testified that Tamraz likely had a genetic predisposition to Parkinson's Disease and that exposure to manganese triggered his Parkinson's to develop. The jury found defendants liable and awarded Tamraz a total of $20.3 million in compensatory damages.

Defendants appealed, claiming that the district court erred in admitting Dr. Carlini's testimony because it was speculative and not based upon published literature or scientific studies. The majority agrees and reverses the district court's decision. For the reasons that I will discuss below, I respectfully disagree with the majority's reasoning and conclusions.

II.

As the majority correctly notes, "[t]his court reviews a district court's decision concerning expert testimony for abuse of discretion." *Popovich v. Sony Music Entertainment*, 508 F.3d 348, 359 (2007) (quoting *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 152-53 (1999)). Unfortunately, while paying lip service to the correct standard, the majority actually applies a de novo standard of review. As we have held, "A district court abuses its discretion if it bases its ruling on an erroneous view of the law or a clearly erroneous assessment of the evidence." *Ky. Speedway, LLC v. Nat'l Assoc. of Stock Car Auto Racing, Inc.*, 588 F.3d 908, 915 (6th Cir. 2009) (quoting *Brown v. Raymond Corp.*, 432 F.3d 640, 647 (6th Cir.2005)) (internal quotation marks omitted). "Thus, we will not substitute our own judgment for that of the district court and will reverse an evidentiary decision 'only where we are left with a definite and firm conviction that [the district court] committed a clear error of judgment.'" *In re Scrap Metal Litigation*, 527 F.3d 517, 528 (6th Cir. 2008) (quoting *Conwood Co., L.P. v. U.S. Tobacco Co.*, 290 F.3d 768, 781 (6th Cir. 2002)); *see also Nolan v. Memphis City Schools*, 589 F.3d 257, 265 (6th Cir. 2009) (holding that "[b]road discretion is given to district courts in determinations of admissibility . . . and those decisions will not be lightly overturned.").

"[Abuse of discretion review] requires a reviewing court to be highly deferential when assessing not just a trial court's analysis of each [*Daubert*] factor, but also the trial court's initial selection of which factors are relevant to the case at hand." *Johnson v. Manitowoc Boom Trucks, Inc.* 484 F.3d 426, 430 (6th Cir. 2007). It is within the district court's discretion to determine whether the testimony provided is inadmissible "junk science" or testimony falling within the "range where experts might reasonably differ."

Kumho, 526 U.S. at 153. Thus, we must conduct our review of that decision with great deference.

Here, because the district court found that Dr. Carlini's "methodology . . . [was neither] flawed or inconsistent with the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used and have described as appropriate diagnostic methods", (corrected J.A. at 170-173, Tr. of Proceedings, Nov. 1, 2007), it is far from apparent that the district court should have found Dr. Carlini's testimony to be unreliable. While the district court acknowledged that Dr. Carlini's diagnosis was "unusual", nothing in *Daubert* and its progeny indicates that an unusual diagnosis alone renders a district court's decision to admit an expert's testimony an abuse of discretion. Here, the district court reasonably evaluated Dr. Carlini's testimony in light of a broad range of expert opinions and found that it was sufficiently reliable to be admissible. As it was not a clearly erroneous decision so as to constitute an abuse of discretion, the district court's judgment should be affirmed.

Expert testimony is inherently difficult to evaluate. It is all the more so outside the context of a trial. This is why the application of *Daubert* is flexible. It is why the district court, which is able to hear and evaluate experts first-hand, is given such broad latitude in determining whether testimony is admissible. That same reasoning counsels that this Court interfere only in cases where it is absolutely clear that the testimony is nothing more than "junk science" that the jury cannot be trusted to evaluate. That is not the case here.

In reversing the district court's decision, the majority substitutes their opinion for that of the district court and exercises a standard of review closer to de novo than abuse of discretion. The upshot of the majority's opinion is that they would have found Dr. Carlini's testimony inadmissible had they been the trial judge, which would be acceptable if we reviewed this case de novo. However, the majority does little to explain why the district court's decision to admit Dr. Carlini's testimony was "arbitrary, unjustifiable, or clearly unreasonable", *Plain Dealer Pub. Co. v. City of Lakewood*, 794 F.2d 1139, 1148 (6th Cir. 1986), as they must do if they wish to reverse the district

court's evidentiary conclusions under the abuse of discretion standard. Although they go to great lengths to explain why they are dissatisfied with Dr. Carlini's "thrice-speculative" testimony, they have not shown why admitting expert testimony, which relies on "the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used" is an abuse of discretion. (Corrected J.A. at 170-173, Tr. of Proceedings, Nov. 1, 2007). In my view, the majority has, with long arms and short sight, reached much further than our standard of review permits. For these reasons, I cannot join the majority's opinion.

III.

The majority offers several reasons for reversing the district court's opinion, none of which I find persuasive. They criticize Dr. Carlini's testimony for being "speculative" (*ante* at 6) and for its "leaps of faith." (*Ante* at 8.) They also claim that Dr. Carlini confused etiology with diagnosis. (*Ante* at 11.) They further take issue with the idea of admitting his testimony under a "differential diagnosis" analysis because many incidents of Parkinson's Disease are idiopathic,[1] a cause which, by its very definition, cannot be "ruled out." (*See ante* at 14-15.) I believe that refocusing the question on the underlying issue that *Daubert* and its progeny intended to address—the exclusion of "junk science"—and reviewing the district court's evidentiary decisions through the appropriate abuse of discretion lens, leaves us no choice but to affirm the district court's evidentiary conclusions.

A. Dr. Carlini's Testimony is Admissible under Daubert

The path charting the judiciary's standards for admitting or excluding expert testimony—from the early *Frye* standard to *Kumho's* clarification of *Daubert*—has been a movement towards granting district judges greater discretion in making expert testimony determinations. *See Kumho*, 526 U.S. at 137; *Daubert v. Merrell Dow*

[1]Idiopathic disease are those for which there is no known cause—although Dr. Gregory House may provide the better definition: "Idiopathic, from the Latin meaning we're idiots 'cause we can't figure out what's causing it." *House: Role Model* (Fox television broadcast Apr. 12, 2005).

Pharm., Inc., 509 U.S. 578 (1993); *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923). Today, that discretion is flexible and very broad. *Kumho*, 526 U.S. at 153.

> *Daubert's* role of 'ensur[ing] that the courtroom door remains closed to junk science,' *Amorgianos v. Nat'l R.R. Passenger Corp*., 303 F.3d 256, 267 (2d Cir. 2002), is not served by excluding [medical expert] testimony . . . that is supported by extensive relevant experience. Such exclusion is rarely justified in cases involving medical experts as opposed to supposed experts in the area of product liability. *See generally* Daniel W. Shuman, *Expertise in Law, Medicine, and Health Care*, 26 J. Health Pol. Pol'y & L. 267 (2001) (characterizing the effect of the *Daubert* and *Kumho Tire* cases on claims of medical expertise as '[m]uch ado about little,' while noting that these cases have had a significant effect on toxic tort and products liability litigation).

Dickenson v. Cardiac and Thoracic Surgery of E. Tenn., 388 F.3d 976, 982 (6th Cir. 2004). "As 'gatekeeper,' the trial judge is imbued with discretion in determining whether or not a proposed expert's testimony is admissible, based on whether it is both relevant and reliable." *Johnson*, 484 F.3d at 429 (quoting *Kumho*, 526 U.S. at 147). It is for the district court to determine whether expert testimony is essentially "junk science" rather than testimony falling within the "range where experts might reasonably differ." *Kumho*, 526 U.S. at 153.

One way in which a court may make this determination is by examining the expert's testimony in relation to the factors laid out by the Supreme Court.

> These factors include: (1) whether a theory or technique . . . can be (and has been) tested; (2) whether the theory has been subjected to peer review and publication; (3) whether, with respect to a particular technique, there is a high known or potential rate of error and whether there are standards controlling the technique's operation; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community."

Johnson, 484 F.3d at 430 (internal quotations omitted). Six years after issuing *Daubert*, the Supreme Court clarified that "the factors listed [in *Daubert*] do not constitute a 'definitive checklist or test.'" *Id*. at 429-30 (quoting *Kumho*, 526 U.S. at 150). Our Court has"recognized that the *Daubert* factors 'are not dispositive in every case' and

should be applied only 'where they are reasonable measures of the reliability of expert testimony.'" *In re Scrap Metal*, 527 F.3d at 529 (quoting *Gross v. Comm'r*, 272 F.3d 333, 339 (6th Cir. 2001)). "Rather, the gatekeeping inquiry must be tied to the facts of a particular case, depending on the nature of the issue, the expert's particular expertise, and the subject of his testimony." *Id.*

While Dr. Carlini testified that he was not able to point to a specific study showing that manganese exposure caused Parkinson's Disease, his testimony was supported by his own general experience and knowledge (corrected J.A. at 615, Tr. Testimony of Walter Carlini, Sept. 13, 2007), and theoretical medical writing that explored the connection between manganese exposure and Parkinson's Disease.[2] (*Id.* at 599, Tr. Testimony of Walter Carlini, Sept. 11, 2007). When asked what publications substantiated his claim, Dr. Carlini clarified that "[t]here is a lot of literature out there about the potential—and it's all theoretical—about the potential causes for sporadic parkinsonism. And a lot of literature discusses the combination of environmental factors together with genetic predispositions." (*Id.*) He further stated that "there is a large likelihood that what we now know as sporadic Parkinson's disease, which is not understood very well, is due to a combination of environmental factors together with an underlying genetic predisposition. That's the way the field is moving." (*Id.*) He additionally testified that, "there is [sic] a lot of studies or a lot of thinking out there . . . which conceptualizes sporadic Parkinson's disease as being . . . a combination of environmental factors and genetic predisposition which is how I conceive of manganese-triggered parkinsonism that falls under that rubric." (*Id.* at 600.) He further stated that "there is quite a bit of writing about patients—theoretical writing about patients developing Parkinson's disease due to a combination of genetic and environmental factors." (*Id.* at 621, Tr. Testimony of Walter Carlini, Sept. 13, 2007).

[2]We must evaluate Dr. Carlini's testimony in light of the science available to him at the time. Any findings—positive or negative—regarding the causal connection between manganese and Parkinson's disease made since that time are irrelevant for this analysis. It is clear that he was referring to a then-ongoing debate regarding the causal connection between manganese exposure and Parkinson's Disease. *See* Murry M. Finkelstein, Michael Jerett, *A Study of the Relationships between Parkinson's Disease and Markers of Traffic-Derived and Environmental Manganese Air Pollution in Two Canadian Cities*, 104 ENVTL. RES. 420-432 (2007); *Link Found Between Parkinson's Disease Genes and Manganese Poisoning*, SCI. DAILY (Feb. 2, 2009), *available at* http://www.sciencedaily.com/releases/2009/02/090201141559.htm (last accessed Aug. 17, 2010).

Thus, the connection between manganese and Parkinson's disease, though not agreed upon by every member of the scientific community, was certainly the subject of valid scientific debate and publication at the time of Dr. Carlini's testimony.[3] The district court succinctly explained its decision not to exclude Dr. Carlini's evidence, focusing on his methodology: "I see nothing about [Dr. Carlini's] methodology that is either flawed or inconsistent with the very diagnostic methods that other experts in this case . . . have used and have described as appropriate diagnostic methods." (*Id.* at 170-173, Tr. of Proceedings, Nov. 1, 2007).

While Dr. Carlini's testimony may not have satisfied every *Daubert* factor, it is not necessary that it do so. *Johnson*, 484 F.3d at 429-30 (holding that the factors do not constitute a definitive checklist or test); *see also In re Scrap Metal*, 527 F.3d at 529 (holding that the *Daubert* factors "are not dispositive in every case and should be applied only where they are reasonable measures of the reliability of expert testimony." (internal quotations omitted)). Dr. Carlini's testimony easily satisfied at least one *Daubert* factor because the manganese-Parkinson's Disease theory was the subject of peer review and publication at the time of Dr. Carlini's testimony. *See infra*, n.2.

Furthermore, to the extent that the connection between manganese and Parkinson's Disease could be tested at the time, the then-ongoing studies of individuals exposed to manganese, who later developed Parkinson's Disease, constitutes testing sufficient to satisfy *Daubert*. Therefore, Dr. Carlini's testimony appears to meet one, if not several, *Daubert* requirements. Thus, the district court did not abuse its discretion in admitting it, and the majority errs in so holding.

B. Speculation and Gaps in the Testimony

The majority finds that Dr. Carlini's testimony was speculative, stating without support that the testimony was "no more than a hypothesis, [and is] thus not 'knowledge,' nor is it 'based upon sufficient facts or data' or the 'product of reliable principles and methods . . . applied . . . reliably to the facts of the case." (*Ante* at 7.) I

[3] A quick internet search of scientific studies published in 2007 shows that a considerable number of studies existed at the time, attempting to establish, with varying degrees of success, that manganese exposure among welders could cause Parkinson's Disease.

disagree that Dr. Carlini's testimony was speculative. Based on the record, it seems clear that Dr. Carlini was relying upon scientific studies which tested the causal connection between manganese exposure and Parkinson's Disease. Furthermore, the district court was exercising its broad discretion when it found that Dr. Carlini's methodology was reliable and consistent with the diagnostic methods used by other experts in the case. It seems incredible that the majority—exercising a standard of review that seems closer to de novo than abuse of discretion, and without the benefit of having sat through the hearings and seen the experts—finds Dr. Carlini's testimony to be speculative.

The majority also cites gaps in Dr. Carlini's testimony as a reason to reverse the district court. (*Ante* at 8.) However, the majority's newly-minted requirement that scientific testimony must be without flaws or gaps and have no unprovable inferences or assumptions runs counter to any reasonable understanding of how scientific "truth" is reached. "Scientists disprove things. In the process they filter error from theories and methodology, but they do not prove that the surviving methodologies—those that are left standing or those that are changed to correct errors—are valid." Jan Beyea & Daniel Berger, *Scientific Misconceptions among* Daubert *Gatekeepers: The Need for Reform of Expert Review Procedures*, 64 LAW & CONTEMP. PROBS. 327, 337 (2001). Furthermore, the "theories that survive testing still have components that have never been tested, contain subjective elements, and require that reasonable inferences be made if they are to be used in real world examples." *Id.* At least one other Circuit court has found that "to the extent that [the defendant] asserts there were gaps or inconsistencies in the reasoning . . . such arguments go to the weight of the evidence, not its admissibility." *Campbell v. Metro. Prop. and Cas. Ins. Co.* 239 F.3d 179, 186 (2d Cir. 2001) (the trial court did not abuse its discretion in admitting expert testimony that plaintiffs were suffering from lead poisoning).

Indeed, the most cherished of scientific "truths" are the subject of constant refinement and are frequently overturned by subsequent science. For instance, the 42-year consensus that DNA alone determines heredity was later "dethroned as a universal

principle, albeit after the 1994 article by Black et al. was published." *Id.* at 335. In fact, simultaneously accepted scientific principles are sometimes incompatible, and thus, might fare badly under a strict *Daubert* application.

> Imagine Euclid testifying in a modern day Daubert proceeding: 'Professor Euclid, I understand that one of your postulates is that parallel lines do not meet at infinity. Can you prove this to be true? Have you ever tested this? Isn't it also true that Professor Einstein has proven that your geometry doesn't work in the presence of gravity?'

Id. at 335 n. 42.

While the district court must necessarily draw lines, we must use caution in demanding the type of finality from science that we have come to expect in law. This is especially true when considering cases of newer scientific studies. It seems to me an overly harsh test at the admissibility level to insist upon testimony with no "gaps", when the science itself may be incapable of absolute proof. Do malfeasing defendants get a free pass on the first few victims because there is not yet a sufficient sample set to create scientific studies with no discernable gaps? Do we tell the early victims, "I'm sorry, you had the misfortune of getting sick too soon", and send them home?

The fact that scientists have not reached consensus regarding medical causation does not render reliance on a scientist's theory improper expert testimony, particularly when, as in this case, the expert is relying on studies that appear to have been conducted using standard methodology. Rather, those differences should go to the weight that a jury should give an expert's testimony. *See Best v. Lowe's Home Centers, Inc.*, 563 F.3d 171, 182 (6th Cir. 2009) (finding that "admissibility under 702 does not require perfect methodology. . . . Any weakness in [a "competent, intellectually rigorous physician's"] methodology ["in identifying the most likely cause of the plaintiff's injury"] will affect the weight that his opinion is given at trial, but not its threshold admissibility."). In cases where the state of scientific consensus is difficult to determine, we must defer to the district court. The district court has the distinct advantage of having heard all the experts testify and can weigh the reliability of a given expert's testimony against others

more easily than we can. Our valuations of complicated medical expert issues such as these are made out of context and are therefore more likely to suffer flaws.

Because Dr. Carlini relied on scientific methodology used by other experts in his field, *see infra* at 28, I do not believe that the district court abused its discretion in admitting his testimony. The district court's determination that Dr. Carlini's methodology was sufficiently reliable was certainly not clearly erroneous, so the testimony was admissible. What weight to grant his testimony was a question for the jury, not an appellate court sitting far removed from the trial. Because the majority has not demonstrated that the district court abused its discretion, it errs in reversing the district court's decision.

IV.

For the reasons above, I respectfully dissent.