No. 08-4015
(Consolidated for Submission with No. 08-4016)

IN THE
UNITED STATES COURT OF APPEALS
FOR THE SIXTH CIRCUIT

JEFF and TERRY TAMRAZ

Plaintiffs-Appellees

v.

Lincoln Electric Company; Hobart Brothers Company; ESAB Group, Inc.,

Defendants-Appellants

APPEAL FROM THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF OHIO
CASE NO. 1:04-CV-18948, MDL DOCKET NO. 1535

PETITION OF PLAINTIFFS-APPELLEES FOR REHEARING AND REHEARING *EN BANC*

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Plaintiffs-appellees petition for a rehearing and a rehearing *en banc* of the majority panel opinion filed on September 8, 2010 (attached at Addendum A). Plaintiffs submit that *en banc* review is necessary for uniformity because the majority opinion conflicts with (1) Supreme Court and Circuit decisions mandating application of a highly deferential "abuse of discretion" standard when reviewing *Daubert* rulings,[1] (2) this Circuit's decisions governing treating physicians' causation opinions,[2] and (3) this Circuit's "harmless error" jurisprudence.[3] The majority's opinion evinces totally unfounded, preconceived hostility toward treating physicians offering opinions on the causes of their patients' illnesses, even when, as here, the physician settled on his opinion independent of the litigation.

In the *Daubert* context, 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." *Brown v. Raymond Corp.*, 432 F.3d 640, 647 (6th Cir. 2005). In applying this standard, this Court has made clear it "will not substitute our own judgment for that of the district court ...." *In re Scrap Metal Litig.*, 527 F.3d 517, 528 (6th Cir. 2008) (citations omitted). It is impossible to ignore the extraordinary

[1] *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 152-53 (1999), *Johnson v. Manitowoc Boom Trucks*, 484 F.3d 426, 430 (6th Cir. 2009); see Dissent by Martin, J., Op. 21.

[2] *Gass v. Marriott Hotel Serv.*, 558 F.3d 419, 427-28 (6th Cir. 2009); *Best v. Lowe's Home Centers, Inc.*, 563 F.3d 171, 181-84 (6th Cir. 2009) (regarding differential diagnoses); *Dickenson v. Cardiac & Thor. Surg. of East. Tenn.*, 388 F.3d 976, 980-83 (6th Cir. 2004).

[3] *Mike's Train House v. Lionel, L.L.C.*, 472 F.3d 398, 409-10 (6th Cir. 2006); *Maggard v. Ford Motor Co.*, 320 Fed. Appx. 367 (6th Cir. April 7, 2009); *Rush v. Ill. Cent. R.R.*, 399 F.3d 705, 723-24 (6th Cir. 2005).

lengths to which the majority went in substituting its judgment of Dr. Carlini's expert testimony for that of the learned district judge. Worse, in doing so, the majority misjudges Dr. Carlini's opinion by misinterpreting the science, disregarding the record evidence, and engaging in speculative second-guessing of both Dr. Carlini and the district court. It also is impossible to ignore the fact that the majority, after wrongly concluding the district court abused its discretion, reverses based on a "harmless error" analysis that not only highlights an intra-Circuit split (over the proper standard), but, with this complete departure from the Court's "harmless error" cases, in effect creates another intra-Circuit split.

I. In Light Of The Evidence In The Record And Applicable Standards For Assessing Scientific Reasoning, The Majority's Review Of The District Court's *Daubert* Ruling Is Insupportable.

The expert opinion at issue is that of Dr. Walter Carlini, a board certified neurologist who is the treating physician of plaintiff Jeff Tamraz. Dr. Carlini has opined that Tamraz has early-onset parkinsonism, with symptoms closer to Parkinson's disease[4] than those of manganism,[5] and that his condition "most

[4] Dr. Carlini referred to Tamraz's condition by various terms, including "Parkinson's disease," "parkinsonism that looks like Parkinson's disease" or has "a lot of similarities to idiopathic Parkinson's disease," "manganese-induced parkinsonism" (aka "MIP"), and "manganese-triggered parkinsonism." While his choice of nomenclature may have varied, his etiological reasoning – that Tamraz's brain damage was caused by exposure to manganese, a medically-recognized opinion – never wavered. To avoid confusion here, we generally will refer to the condition Dr. Carlini ascribed to Tamraz simply as "PD." *See*, *e.g.*, JA 604(39:20-21) & 615(20:11-12). (Citations to Dr. Carlini's testimony in parentheses pinpoint the pages and lines).

[5] JA 600(26:21-25). As Dr. Carlini testified, manganism, in its classic form, is the most egregious manifestation of brain damage resulting from severe manganese overexposure, JA

likely"[6] was caused or accelerated[7] by his exposure to welding fumes while working without a respirator in tightly confined spaces.[8] The early onset of Tamraz's illness – in his early 40's – is a key factor in his opinion.[9]

The majority opinion states that, in coming to these conclusions, Dr. Carlini speculated "three times over." Op. 7. First, it labels testimony that genetics and environmental toxins can combine to produce PD as his "hypothesis." *Id*. This is wrong on both counts – Dr. Carlini did not conceive this, and it cannot be passed off as "no more than a hypothesis," *e.g.*, having no basis. It aligns with the generally accepted theory, documented in scientific literature since 1996, that "Parkinson's disease (PD) is thought to develop as a result of interactions between genetic susceptibility factors and environmental exposures."[10] While perhaps a "working hypothesis" when Tamraz first encountered manganese in welding fumes decades ago, it has been a generally accepted scientific theory since at least 1996,[11]

598(18:1-7), while Tamraz had lower-dose, longer-term manganese exposure, which, in Dr. Carlini's opinion, was capable of triggering PD. *Id.* at ll. 13-20.

[6] JA 615(21:1-3).

[7] JA 596(8:8-14).

[8] JA 613(13:1-5).

[9] JA 601(27:14-17), 604(42:19-22).

[10] Sandy *et al*., *CYP2D6 allelic frequencies in young-onset Parkinson's disease*. Neurology 47(1): 225-230 (1996).

[11] *Id*.

with no backtracking.[12] Defendants' expert at the two-week *Daubert* hearing, Dr. Warren Olanow, espoused it.[13] And defendants' expert at the *Tamraz* trial, Dr. Anthony Lang, co-authored an article several months before Dr. Carlini's testimony, a "state of the art review by leaders in Parkinson's research,"[14] which stated: "It is clear that PD ... is ***most likely*** caused by a combination of genetic and environmental factors."[15] Yet, when Dr. Carlini adopts that theory,[16] it becomes a "hypothesis."[17]

Second, the majority maintains it took two more "speculative jumps" for Dr. Carlini to identify manganese exposure as the environmental factor that brought about Tamraz's "early-onset" parkinsonism. Op. 7. But Dr. Carlini did not have to make speculative jumps; rather, he engaged in probabilistic scientific reasoning, determining that Tamraz "most likely" had a genetic susceptibility to PD, activated

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[12] Litvan *et al*., *The etiopathogenesis of Parkinson disease and suggestions for future research: Part I*. J. Neuropathol. Exp. Neurol. 66(4): 251-257 (2007).

[13] JA 591:6-18.

[14] Litvan, *supra,* at 329.

[15] *Id.* at 329-336, cited at JA 448 (emphasis added). *Compare with* JA 599(21:11) (Dr. Carlini: "there is a large likelihood").

[16] JA 600(23:5-12), 621(46:16-19), 622(50:20-24).

[17] Of course, as with any scientific theory, not everyone agrees with it, but that does not justify the majority's "hypothesis" label; nor is it correct in interpreting Dr. Carlini's description of the scientific literature supporting his opinion as "theoretical writing" as if he were suggesting a lack of support. To scientists, the terms theory and theoretical writings do not connote a lack of support, often quite the opposite (*e.g.*, the theory of relativity is not a hypothesis). *See* discussion in *Daubert v. Merrell Dow Pharms.*, 509 U.S. 579, 590 (1993).

when he was exposed to manganese in confined spaces.[18] In line with accepted scientific thought, he viewed Tamraz's "early-onset" PD as evidence of genetic susceptibility, an inference defense expert Dr. Lang deemed reasonable.[19] Yet, when Dr. Carlini makes that inference, it becomes a "guess." Op. 7.

The majority also treats as guesswork Dr. Carlini's conclusion that manganese – a known neurotoxin – was the most likely activating or accelerating agent of Tamraz's illness. The majority fails to recognize the process of probabilistic scientific reasoning that led him to this conclusion. First, he ruled in welding fumes – specifically the manganese in them – as the activating or accelerating environmental agent because "manganese would be a highly likely suspect for an environmental factor based on the way it causes problems in the brain."[20] Second, his recognition that manganese damages all regions of the brain relevant to PD, not just the regions associated with manganism following severe manganese poisoning,[21] tracks the reasoning of a wider group of authoritative

[18] JA 601(28:2-4); *see* Henefin *et al.*, Reference Guide on Medical Testimony, *Reference Manual on Scientific Evidence* 439, 471 (2000) ("Physicians use both causal and probabilistic reasoning in determining both internal and external causation in regard to a particular illness.").

[19] *See* Dr. Lang's Report, p. 3, cited at JA 448 (inferring "the possibility of a genetic form of the disorder" from his "relatively young age of onset").

[20] JA 601(30:16-18).

[21] JA 596(8:15-25), 598 (18:13-20), 601(28:7-16).

researchers and analysts in the neurological and neurotoxicological communities.[22] Third, he explained that manganese was more likely than any other neurotoxin to have been the activating or accelerating environmental agent "because we know that manganese all by itself can cause a form of parkinsonism if it's in sufficient quantity."[23] Fourth, he concluded that Tamraz's exposure to manganese over time in his job, at levels lower than the intoxicating doses that can cause manganism,[24] most likely was the neurotoxic "hit" that "set off" his genetic susceptibility, tipping him into PD.[25]

The majority mentions "early onset" only dismissively, Op. 8-9; but, in Dr. Carlini's view, it solved the mystery of why Tamraz developed PD. Although the majority tosses this factor aside – as if Tamraz's highly unusual PD in his early 40's could only warrant an "idiopathic" PD designation – it should be noted that the majority cites no scientific basis, whether inside or outside the record, for

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[22] Racette *et al*., *Welding-related parkinsonism: Clinical features, treatment, and pathophysiology*. Neurology 56(1):8-13 (2001); Martin, *Manganese neurotoxicity: Connecting the dots along the continuum of dysfunction*. NeuroToxicology 27(3): 347-349 (2006); Weiss, *Economic implications of manganese neurotoxicity*. NeuroToxicology 27(3): 362-368 (2006); See also testimony of Dr. Louis at *Daubert* hearing, Vol. I, p. 108.

[23] JA 599(19:18-21); Racette, *supra*, at 12 ("welding exposure" as "an accelerant to cause PD").

[24] JA 599(20:1-5) ("Because in manganism all you need is manganese; you don't need any genetic predisposition. If you have enough manganese, you can poison somebody. But you don't need that much manganese to cause the other kind of manganese-induced or manganese-triggered Parkinson's.").

[25] JA 599(19:4-8), 601(28:23-25).

embracing idiopathic PD, to the exclusion of another more probable designation. Thus, with due respect, the majority has engaged in precisely what it accuses Dr. Carlini of – speculating.[26] In contrast, the district court well knew, when it admitted Dr. Carlini's testimony in 2007, that there already was scientific support in the record buttressing Dr. Carlini's interpretation of "early onset" as a critical, indeed the decisive, factor in his etiological determination. This exchange between the district court and defendants' own expert at the 2005 *Daubert* hearing demonstrates the acceptance enjoyed by Dr. Carlini's "early onset" interpretation:

> THE COURT: … [W]hen you are weighting all of these factors in your effort to make a differential diagnosis, at what point or of what weight could you give age of onset?
>
> DR. OLANOW: I think age of onset is important. Quite candidly, if I see a young onset patient, it makes me think of genetic causes because the evidence suggests that most cases that are genetic in origin are young onset, like in Parkin cases. Recently there have been some genes discovered that have made a lot of news because the age of onset is relatively old, but the alpha synuclein, the Parkin, the large majority tend to occur at a very young age of onset. So if I see a young age of onset, it strongly supports a genetic background. (JA 591:6-18)

Thus, when Dr. Carlini presented a strikingly similar interpretation of "early onset" two years later, it was only natural and reasonable for the district court to observe:

> ... I see nothing about his methodology which is either flawed or inconsistent with the very diagnostic methods that other experts in this

[26] Based on articles not found in the record – including one by two lawyers who are not experts on physicians – the majority further speculates that Dr. Carlini takes more care with diagnoses than with etiology. Op. 11. But even defendants presented evidence showing parkinsonisms' etiology matters in prescribing treatment. JA 134-37, 141-43. This highlights the majority's inclination to resolve the case based on its own viewpoints, rather than the record.

> case, both the plaintiffs[’] and the defendants’ experts alike, have used and have described as appropriate diagnostic methods. (JA 171:8-12.)

After confusing Dr. Carlini’s probabilistic reasoning with speculation, the majority proceeds to fault him for flaws it perceives in his thinking: for failing to consider that PD occurs “usually without any known cause,” Op. 8; failing to consider that Tamraz could have developed PD “regardless of the manganese exposure,” *id*.; failing to do brain tests to confirm manganese caused it, *id*.; and not using manganese-induced parkinsonism synonymously with manganism. *Id.* at 10.

The district court reasonably accounted for all of these factors, whether in its unusually thorough *Daubert* hearing and ruling, JA 128-66, or in explaining why Dr. Carlini’s testimony passed muster. JA 167-73. To require ruling out all unknown agents is to set an impossible burden on Tamraz and his experts. Indeed, the Reference Guide on Medical Testimony, on which the majority relies, deems it unnecessary to do so.[27] Dr. Carlini did consider, and ruled out, whichever parkinsonisms he could, given the data at hand. JA 614(15-16). As for other environmental agents or alternative causes of PD, defendants did not ask Dr. Carlini if he had considered them, so it would be unfair to assume he did not.[28]

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[27] Henefin *et al*., at 476 (“Accordingly, the common statement that ‘alternative causes of disease must be ruled out’ before causation is attributed can be more accurately refined to say that ‘the role of other causes must be adequately considered.’”).

[28] JA 171 (“[By the district court:] I think it is telling that the defendants have not proffered an affidavit from any expert indicating that there is anything wrong with Dr. Carlini’s methodology, or that there is anything faulty with respect to his qualifications.”).

The majority's observation that "[a]ny given case of Parkinson's Disease thus might have occurred regardless of the manganese exposure" has no bearing on whether Carlini's methodology for diagnosing PD in this case, or the district court's appraisal of it, was flawed.[29] It is a statement that could be made in practically every toxic tort case. Dr. Carlini's methodology, like that of every causation expert in this case, was designed merely to offer an opinion whether manganese caused Tamraz's parkinsonism, not whether unknown persons with unknown genetic predispositions and a lifetime of unknown environmental exposures might get the disease sometime.

Based on her earlier two-week exploration of every facet of the relevant science, JA 138, 164, 165-75, the district judge appreciated that, while Dr. Carlini did not use terms like "MIP" and "manganism" as others have, his etiological opinion was reliable, making his "word choice" immaterial.[30] Indeed, as the judge aptly observed, Dr. Carlini was a treating physician who did not testify like a

[29] Nor does it matter that Dr. Carlini could not answer when asked if someone with a genetic predisposition toward the disease has a 90% or 10% chance of developing it. JA 601. As Dr. Carlini logically explained with reference to Tamraz, "You can't say for sure." *Id.* at 622. This was the only proper answer because it depended on information – *e.g.*, a subsequent environmental exposure – that the question did not supply and that could not be. *Id*. at 601. In any event, he accounted for accelerated onset in his opinion. As for the brain testing he did not perform, the district court knew full well that ethical testing does not definitively classify parkinsonisms. JA 151; *see* also JA 313(36) (Dr. Carlini: "While the patient is alive?").

[30] The majority stresses word choice, particularly "manganism," though Dr. Carlini said Tamraz does not have it. JA 317(54:11-12), and it accuses Tamraz of "conflating 'manganese-induced parkinsonism' with manganism ...." Op. 11. *But see* JA 535:16-17 (defense counsel's opening statement: "When I say manganism, I mean manganese-induced Parkinsonism.").

"practiced litigation expert." *Id.* Ironically, this fact works against him in the majority opinion, contrary to standards for treating physician testimony established only last year in *Gass*, 558 F.3d at 427-28, and *Best*, 563 F.3d at 181-84. Thus, the majority's opinion will make it more difficult, not less, for treating physicians to render admissible causation opinions, placing greater emphasis, not less, on polished litigation experts. Ultimately, as the dissent notes, Op. 28-31, this opinion pushes the Court into new *Daubert* territory, requiring absolute scientific certainty that will be impossible to establish, not just in this case and not just by this expert.

II. The Majority Opinion Highlights An Intra-Circuit Split On The Proper "Harmless Error" Standard, Then Opens Another By Departing From This Circuit's Prior Evidentiary Error Precedents.

A. The full Court should decide the proper "harmless error" standard.

The majority follows a line of Sixth Circuit authority that presumes the evidentiary error materially affected the jury verdict, which conflicts with another line of Sixth Circuit authority that presumes such error did *not* materially affect the verdict. [31] A circuit's "harmless error" standard involves more than just semantics.

[31] *Compare* Op. 16 (citing *Mike's Train House* ("The harmless-error standard provides that if one cannot say, with fair assurance, .. that the judgment was not substantially swayed by the error, it is impossible to conclude that substantial rights were not affected."), *Maggard*, *supra*, 320 Fed. Appx. 367, and *Rush*, *supra*, 399 F.3d at 723-24, *with Stockman v. Oakcrest Dental Ctr.*, P.C., 480 F.3d 791, 804 (6th Cir. 2007) (an evidentiary error justifies nullification of "a jury verdict where the error so altered the total mix of information submitted to the jury that it was substantially likely to have affected the verdict"), *Cetlinski v. Brown*, 91 Fed. Appx. 384, 389 (6th Cir. 2004) ("even if the trial court abuses its discretion, a new trial is not required unless 'substantial rights' of a party are affected" and placing the "burden of showing harmful prejudice . . . on the party seeking the new trial"); *see also Hillside Productions, Inc. v. County Of Macomb*, Case Nos. 08-2268, 2232, 2001 WL 2545592, at *9 (6th Cir. June 15, 2010).

Often, it is the difference between affirmance and reversal. Unless the full Court articulates the proper standard Sixth Circuit panels must use in conducting "harmless error" inquiries in evidentiary error cases, this Circuit's "harmless error" pronouncements in such cases will remain inconsistent. Granting a rehearing *en banc* is the logical way to achieve uniformity on this key point.

B. The majority's conclusion this could not be harmless error cannot be reconciled with the case cited or other Sixth Circuit authority.

Regardless which "harmless error" standard applies, the majority opinion contradicts prior Circuit law holding that, when wrongly-admitted evidence augments or duplicates properly-admitted evidence, the error is harmless.[32] In *Mike's Train House*, the only "harmless error" case the majority cites, the Court held that allowing an engineering expert to testify was not harmless error because he "was the only expert to testify" in support of the plaintiff's liability theory. 472 F.3d at 410. "Without his testimony," the Court explained, "the jury would not have learned of [plaintiff's liability theory], nor would they have heard testimony from any other expert witness regarding" that theory. *Id*.

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[32] *See, e.g., Rush*, 399 F.3d at 723-24 (error in admitting evidence harmless because it was cumulative); *United States v. Martinez*, 588 F.3d 301, 312 (6th Cir. 2009) (error in admitting evidence harmless because it merely "bolster[ed] the testimony of the Government's primary expert"); *Morales v. American Honda Motor Co., Inc.*, 151 F.3d 500, 514 (6th Cir. 1998) (error in excluding evidence harmless because it was not necessary to rebut the plaintiff's argument); *United States v. Smith*, 736 F.2d 1103, 1107 (6th Cir. 1984) (error in admitting evidence harmless because evidence was duplicative).

Relying on *Mike's Train House*, the majority cites two reasons why "we cannot say 'with fair assurance' that the result would have been the same without" Dr. Carlini's testimony. Op. 17. First is "[t]he emphasis Tamraz put on Dr. Carlini's testimony," noting that plaintiff highlighted his "treating role." *Id.* at 16. In fact, however, both sides emphasized Dr. Carlini's testimony equally. Defendants embraced it from the outset because they believed his differential diagnosis would help the jury accept defendant's theory that Tamraz had PD, not manganism.[33] As a reflection of their faith in his differential diagnosis, defense counsel designated significant portions of his videotaped testimony and spent as much of their closing argument touting his testimony as Tamraz's counsel did, including replaying a considerable portion of the videotape (twice as much as Tamraz replayed),[34] every bit of which defendants obviously perceived as friendly to their causation theory.[35] No doubt because of that perception, defendants also reminded the jury Dr. Carlini was Tamraz's "treating physician." Tr. 2759:8-10.

[33] As defendants said of Dr. Carlini's testimony: "Dr. Carlini proved sufficiently competent and qualified both to diagnose Mr. Tamraz with Parkinson's disease and to rule out a diagnosis of Manganism. A board certified neurologist, Dr. Carlini intelligently discussed the two disease entities and the features described in the literature to be diagnostic of each.... He arrived at his diagnosis of Parkinson's disease after interviewing Mr. and Mrs. Tamraz, examining Mr. Tamraz and applying his experience and clinical judgment.... Most circuits have held that such a differential diagnosis provides a valid foundation for admitting an expert opinion...." JA 364.

[34] *Compare* Tr. 2743-44 & 2826 *with* Tr. 2760-61, 2762-65.

[35] Defense counsel's closing argument demonstrates their affirmative reliance on Dr. Carlini's testimony, reliance born of their belief it bolstered their causation theory insofar as he maintained Tamraz did not have manganism and did have PD. *See* Tr. 2759, 2760, 2761, 2765, 2784 ("Dr. Carlini agreed, Mr. Tamraz had none of that.... Dr. Carlini admitted that Mr. Tamraz

The fact that both sides equally emphasized how much Dr. Carlini's testimony helped their respective cases suggests the jury most likely viewed it as equiponderant, not, as the majority surmises, one-sided in Tamraz's favor. In assessing how much the "emphasis" on Dr. Carlini's testimony "swayed" the jury, *Mike's Train House*, 472 F.3d at 410, the majority wrongly gives undue weight to the impact Tamraz's efforts to exploit his testimony must have had on the jury, while giving ***no*** weight to the jury impact of similar efforts by defendants. The fact the parties' efforts stood in equipoise should have led the panel to conclude those efforts were self-canceling and, thus, that the purported error was harmless.

The majority next cites the difference between Dr. Carlini's causation testimony and that of Dr. Nausieda, as evidence the purported error could not be considered harmless. Op. 17. In its view, admitting Dr. Carlini's testimony in addition to Dr. Nausieda's allowed Tamraz to offer the jury two distinct diagnoses, both "of which helped Tamraz" – in essence requiring defendants to fight a two-front war where "the primary question" had shifted "from what disease Tamraz had to whether manganese caused it." *Id.* The majority's logic is flawed for several reasons. First, its focus on the purported difference between the two

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had none of those symptoms.... Dr. Carlini admitted that Mr. Tamraz had none of this.... [Dr. Carlini] couldn't say that Jeff Tamraz had manganism. He couldn't say he had manganese-induced Parkinsonism .... Dr. Carlini diagnosed Parkinson's disease .... In his deposition, we went through line by line, symptom by symptom, manganese-induced Parkinsonism. And he said Jeff Tamraz doesn't look anything like that description either....").

physicians' diagnoses is a puzzling retreat from the majority's earlier formulation of the "primary question": what caused Tamraz's disease? *Id*. at 6 (distinguishing "Dr. Carlini's etiology (what caused the disorder diagnosed?)" from "his diagnosis (what disorder caused the set of symptoms observed?)"). Second, the record belies attempts to distinguish Dr. Nausieda's opinion from Dr. Carlini's on the operative question before the jury – etiology. Both physicians identified manganese as the cause of Tamraz's illness, which both called MIP. JA 758, 863-66. Third, any notion that the verdict somehow resulted from the jury latching onto the distinction between Dr. Nausieda's opinion and that of Dr. Carlini is implausible, if for no other reason than Dr. Nausieda testified both he ***and*** Dr. Carlini (whose testimony he reviewed) found the very same disease with the very same cause.[36]

The operative question for the jury was, did manganese cause Tamraz's parkinsonism or didn't it? Tamraz presented more than a day of testimony by Dr. Nausieda and 90 minutes of testimony by Dr. Carlini to show that manganese caused it. *See*, *e.g.*, J.A. 742-795, 826-1034, 1036-1088. Defendants presented a day of testimony by Dr. Lang to show it did not. *See*, *e.g.*, JA 1299-1306. In contrast to *Mike's Train House*, 472 F.3d at 410,[37] hypothetically removing Dr.

[36] J.A. 862-63, 865 (Dr. Nausieda testifying that Tamraz had manganese-induced parkinsonism and that he and Dr. Carlini were "all in on the same page" with respect to that diagnosis); J.A. 758 (Dr. Carlini agreeing his "diagnosis" was "manganese-induced parkinsonism").

[37] The Court employed the same reasoning in *Maggard*, holding that improperly excluding expert testimony offered by the defendant could not be harmless error because, in its absence, there was "only one plausible version of the events leading to [the plaintiff's] injury," *i.e.*, the

Carlini's testimony from the mix still would leave the jury with Dr. Nausieda's exhaustive testimony that manganese caused Tamraz's illness. Until this case, the Court uniformly found such error to be harmless. *See* n.32, *supra*.[38]

This trial lasted eleven days. The jury deliberated eight more. To accept that Dr. Carlini's barely 90-minute videotaped testimony tipped the scales in Tamraz's favor, such that its admission could not be harmless error, one must believe it made no difference to the jury that defendants themselves touted Dr. Carlini's testimony, not to blunt its impact, but to show Tamraz has PD, not MIP or manganism; or that Dr. Lang testified that manganese did not cause his illness; or that Dr. Nausieda testified that manganese did cause it. Under the majority's analysis, none of these factors could conceivably have made enough difference to the jury to support a "harmless error" finding. Abundant Sixth Circuit authority and common sense both dictate otherwise, and not a single case from this Circuit supports the majority's "harmless error" calculus.

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plaintiff's. 320 Fed. Appx. at 376. That factor, also found *Mike's Train House*, is not present here. *See also Nemir v. Mitsubishi Motors Corp.*, 381 F.3d 540, 558-60 (6th Cir. 2004).

[38] A Seventh Circuit decision coincidentally involving manganese-induced parkinsonism, *Jones v. Lincoln Elec. Co.*, 188 F.3d 709 (7th Cir. 1999), is also instructive here. The Seventh Circuit determined the district court abused its discretion under *Daubert* in permitting a defense expert to testify beyond his expertise, *id*. at 724, but found harmless error in part because the plaintiffs rebutted the improper expert testimony with their own expert's testimony. *Id*. at 726. The testimony of defense expert Dr. Lang disputing any connection between manganese and Tamraz's illness (*see*, *e.g.*, JA 1300-01) also supports a finding of harmless error.

CONCLUSION

Therefore, Tamraz respectfully requests a rehearing by the full Court.

Respectfully submitted,

/s/ Paul M. De Marco
Paul M. De Marco

CERTIFICATE OF SERVICE

I certify that on October 1, 2010, the foregoing document was served on all parties or their counsel of record through the CM/ECF system if they are registered users or, if they are not, by placing a true and correct copy in the United States mail, postage prepaid, to their addresses of record.

/s/ Paul M. De Marco