

**UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF OHIO
EASTERN DIVISION**

IN RE: WELDING ROD PRODUCTS
LIABILITY LITIGATION

Case No. 1:03-CV-17000

MDL Docket No. 1535

This Document Relates to All Actions

Honorable Kathleen M. O'Malley

**MEMORANDUM IN SUPPORT OF
MOTION TO EXCLUDE ALL TESTIMONY THAT EXPOSURE TO
WELDING FUMES CAUSES PARKINSON'S DISEASE**

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<i>Glastetter v. Novartis Pharmaceuticals Corporation.</i> , 252 F.3d 986 (8 th Cir. 2001)	28
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<i>Hollander v. Sandoz Pharmaceuticals Corp.</i> , 289 F.3d 1193 (10 th Cir. 2002)	6
<i>In re Agent Orange Prod. Liab. Litig.</i> , 611 F. Supp. 1223 (E.D.N.Y. 1985) <i>aff'd.</i> , 818 F.2d 187 (2d Cir. 1987), <i>cert. denied</i> , 487 U.S. 1234 (1988) . . .	20, 22
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<i>Lore v. Lone Pine Corp.</i> 1986 WL 637507 (N.J. Super. Ct. 1986)	30
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<i>Smelser v. Norfolk S. Ry. Co.</i> , 105 F.3d 299 (6 th Cir. 1997)	4
<i>Soldo v. Sandoz Pharms. Corp.</i> , 244 F. Supp. 2d 434 (W.D.Pa. 2003)	20
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<i>Wynacht v. Beckman Instruments, Inc.</i> 113 F. Supp. 2d 1205 (E.D. Tenn. 2000)	7

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Other Authorities

Bernstein, <i>The Admissibility of Scientific Evidence After Daubert v. Merrell Dow Pharmaceuticals, Inc.</i> , 15 Cardozo L. Rev. 2139 (1994)	20
Bernstein, <i>The Breast Implant Fiasco</i> , 87 Cal. L. Rev. 457 (1999)	32-33

Bradford Hill, <i>The Environment and Disease: Association or Causation?</i> , 58 Proc. Royal Neward Sco’y Med. 295 (1965)	12
Cheng, <i>Changing Scientific Evidence</i> , 88 Minn. L. Rev. 315 (2003)	29, 33
Dreessen, Waldemar C. and Senior Surgeon, <i>et al.</i> , U.S. Public Health Service Bulletin No. 298, <i>Health of Arc Welders in Steel Ship Construction</i> , (1947)	14
Dresser, <i>et al.</i> , <i>Breast Implants Revisited: Beyond Science on Trial</i> , 1997 Wis. L. Rev. 705 (1997)	32
Drever, <i>Occupational Health Decennial Supplement</i> . Series DS no.10 ed. London: HMSO; 1995 at (Chapters 4, 5 and App. 7-8) Coggon, <i>et al.</i> , <i>Occupational Mortality of Men</i>	15
Federal Judicial Center, <i>Reference Manual on Scientific Evidence</i> , (2d ed. 2004)	4, 6-7, 9-12, 21, 23, 25
Goldman, <i>et al.</i> , <i>Occupation and PD Onset Age in Movement Disorders Clinic</i> , (Abstract: S08.006, April 27, 2004)	14
Gorell, <i>et al.</i> , <i>Occupational Exposure to Manganese, Copper, Lead, Iron, Mercury and Zinc and the Risk of Parkinson’s Disease</i> , 20 NeuroToxicology 239 (1999)	17
Gorell, <i>et al.</i> , <i>Multiple Risk Factors for Parkinson’s Disease</i> , 217 Journal of Neurological Sciences 169 (2004)	17
Green, <i>Bendectin and Birth Defects: The Challenges of Mass Toxic Substances Litigation</i> , University of Pennsylvania Press (1996)	33
Green, <i>Expert Witnesses And Sufficiency Of Evidence In Toxic Substances Litigation: The Legacy Of Agent Orange and Bendectin Litigation</i> , 86 Nw. U.L. Rev. 643 (1992)	20
Meyers, <i>Focusing: When Less Is More</i> , 28 Litig. 6 (2002)	30
Milham, <i>Occupational Mortality in Washington State 1950-1989</i> , U.S. Department of Health and Human Services, NIOSH, Order No. 00913725 (1997)	16

Nagareda, <i>In the Aftermath of the Mass Tort Class Action</i> , 85 Geo. L.J., 295 (1996)	32
Poulter, <i>Science and Toxic Torts: Is There A Rational Solution To The Problem of Causation?</i> 7 High Tech. L. J. 189 (1992)	20
Racette, <i>et al.</i> , <i>Prevalence of Parkinsonism In A Large Cohort of Welders, Movement Disorders</i> , Vol 19, Suppl. 9, 2004	26
Racette, <i>et al.</i> , Reply from the Authors, 57 Neurology 936 (2001)	25
Racette, <i>et al.</i> , <i>Welding-Related Parkinsonism: Clinical Features, Treatment And Pathophysiology</i> , 56 Neurology 8 (2001)	24
Ravina, <i>et al.</i> , Letter to the Editor, 57 Neurology 936 (2001)	24
Ruskin, <i>Prove it or Lose It: Defending Against Mass Tort Claims Using Lone Pine Orders</i> , 26 Am. J. Trial Advoc. 599 (2003)	31
Saks, <i>et al.</i> , Federal Judicial Center, Annotated Reference Manual on Scientific Evidence, (2d ed. 2004) at 637	8, 10, 23
Sanders, <i>Bendectin on Trial: A Study of Mass Tort Litigation</i> (1998)	33
Sanders, <i>From Science to Evidence: The Testimony On Causation In the Bendectin Cases</i> , 46 Stan. L. Rev. 1, 25 (1993)	20
Sanders, <i>et al.</i> , <i>The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law</i> , 64 Law & contemp. Probs. 107, 110 n.13 (2001)	8
Seidler, <i>et al.</i> , <i>Possible Environmental, Occupational, and Other Etiologic Factors for Parkinson's Disease: A Case Study in Germany</i> , 46 Neurology 1275 (1996)	18
Semchuk, <i>et al.</i> , <i>Parkinson's Disease: A Test of Multifactorial Etiologic Hypothesis</i> , 43 Neurology 1173 (1993)	16
Steiner, <i>The Case Management Order: Use and Efficacy in Complex Litigation and the Toxic Tort</i> , 6 Hastings W.-N.W.J. Envtl. L. & Pol'y 71 (1999)	31

Susser, <i>Causal Thinking in the Health Sciences: Concepts and Strategies in Epidemiology</i> , Oxford University Press (1973)	12
Tanner, <i>et al.</i> , <i>Occupation and Risk of Parkinson's Disease (PD): A Preliminary Investigation of Standard Occupational Codes (SOC) in Twins Discordant for Disease</i> (Abstract: S49.001)	13
Tsui, <i>et al.</i> , <i>Occupational risk factors in Parkinson's Disease</i> , 90 Canadian Journal of Public Health 334-337 (1999)	13
Vieregge, <i>et al.</i> , <i>Long Term Exposure to Manganese in Rural Well Water Has No Neurological Effects</i> , 22 Can. J. Neurol. Sci. 286 (1995)	18
Walker, <i>Scientific Authority: The Breast Implant Litigation and Beyond</i> , 86 Va. L. Rev. 801 (2000)	33
Walrath, Judy, Ph.D., <i>et al.</i> , Nat'l Inst. of Health, Pub. No. 85-2756, <i>Mortality Patterns Among U.S. Veterans by Occupation and Smoking Status</i> (1985)	15
Wright, <i>Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts</i> , 73 Iowa L. Rev. 1001, 1046 (1988)	8

I. INTRODUCTION

Defendants bring this motion pursuant to Federal Rules of Evidence 104(a), 702 and 703 to obtain an order – applicable to all cases in this proceeding – that plaintiffs not introduce at trial any expert testimony that welding fumes cause or accelerate the onset of Parkinson’s Disease (“PD”). The science on this issue is not in dispute. No scientific literature establishes that either welding fume or manganese causes or accelerates the onset of PD. That being so, the Court should not allow plaintiffs to introduce expert evidence to the contrary. Any other result would violate the rule that litigants must employ scientifically reliable expert testimony. *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993) (“*Daubert*”).

PD is a progressive neurological disorder characterized by slowness of movement, resting tremor and rigidity. It affects approximately 1 million Americans, including such public figures as Janet Reno and Michael J. Fox, and it accounts for 75%-80% of all cases of ‘parkinsonism’ – a broad class of clinically and pathologically distinguishable movement disorders sharing some physical symptoms with PD.¹

But for in a very small percentage of cases where genetic factors have been implicated (under 1,000 world-wide), scientists have been unable to identify the cause(s) of PD despite more than one and one-half centuries of research. Remarkably, where medical science has failed, plaintiffs are expected to identify experts who will causally

¹ One such disorder is manganism -- a condition caused by manganese intoxication. In some cases before this Court, the plaintiffs likely will identify experts who will opine that plaintiffs are suffering from manganism and that occupational exposure to welding fumes caused this condition. Although defendants believe that these claims lack the requisite scientific support, this Motion does not address the merit of those claims. Instead, it seeks only to exclude evidence offered in support of claims in which plaintiffs assert that they have, may have, or will contract PD.

relate exposure to welding fumes with the development or early onset of PD.² Such opinions are not based upon reliable scientific evidence, as required by *Daubert* and its progeny and, accordingly, are inadmissible. As discussed in Section VII(C), *infra*, resolution of this medical-evidentiary issue will have widespread application to the welding rod litigation.

II. THIS COURT IS THE ‘GATEKEEPER’ AND MUST EXCLUDE UNRELIABLE EXPERT TESTIMONY.

*“Just as houses are made of stones, so is science made of facts.
But a pile of stones is not a house and a collection of facts is not necessarily science.”*³

Although “[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof” are traditional and appropriate means of attacking “shaky” but otherwise admissible scientific evidence, in *Daubert*, 509 U.S. at 597, the Supreme Court held that Rules 104(a) and 702 of the Federal Rules of Evidence impose a special *gatekeeping* obligation on federal courts, requiring judges to screen proffered expertise to ensure that such evidence is both reliable and relevant before being admitted at trial. *Id.* at 589. Failure to fulfill this gatekeeping obligation is reversible error. *Dodge v. Cotter Corporation*, 328 F.3d 1212 (10th Cir. 2003).

² In a number of constituent cases in the MDL proceeding, including cases originally filed directly in the U.S. District Court for the Northern District of Ohio, the complaints specifically allege that the plaintiff’s “exposure to welding fumes caused him to develop and be diagnosed with symptoms of Parkinson’s Disease, either in the form of Parkinson’s Disease, parkinsonism, or manganism.” Complaint, ¶ 87, *Spafford v. Lincoln Electric Co.*, Civ. A. 1:03-CV-17548 (filed in N.D. Ohio on Dec. 18, 2003); *see also, e.g., Fluckey v. Lincoln Electric Co.*, ¶ 87, Civ. A. 1:04-CV-17342 (filed in D. Minn. on Jan. 30, 2004; plaintiff’s motion to transfer to MDL pending).

³ Jules Henri Poincare (1854–1912), French scientist, author. *Value of Science*, Dover (1958).

Rule 104 (a) provides that “[p]reliminary questions concerning the qualification of a person to be a witness . . . or the admissibility of evidence shall be determined by the court.” Rule 702 provides:

If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or 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.

In cases involving scientific evidence, “evidentiary reliability” is based upon “scientific validity.” *Daubert*, 509 U.S. at 590 n.9. Evidence is scientifically valid if “it is ground[ed] in the methods and procedures of science.” *Id.* While emphasizing that the inquiry envisioned by the Rules of Evidence is “a flexible one,” in *Daubert*, the Supreme Court enumerated a nonexhaustive list of factors for courts to consider when determining whether evidence was scientifically valid or reliable. These include: (1) whether the expert's theory can be or has been tested; (2) whether the theory has been subject to peer review and publication; (3) the known or potential rate of error of a technique or theory when applied; (4) the existence and maintenance of standards and controls; and (5) the degree to which the technique or theory has been generally accepted in the scientific community. *Id.* at 593-94.⁴

Courts, however, were never intended to be limited to the *Daubert* factors in their gatekeeping assessments. Depending on the particular facts of a case, the nature of the

⁴ In addition, courts must “be mindful of other applicable rules,” including Federal Rules of Evidence 703 and 403. *Daubert*, 509 U.S. at 595. Under Federal Rule of Evidence 703, expert opinions may be based on hearsay only if the “facts or data” relied upon are of a type “reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject.” Federal Rule of Evidence 403 allows a court to exclude relevant evidence “if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury.”

issues, the expert's particular expertise and the subject of her testimony, some or all of the *Daubert* factors may apply. *Kumho Tire Co. v. Carmichael* 526 U.S. 137, 138, 119 S.Ct. 1167, 1170 (1999).⁵ The Supreme Court elaborated upon its *Daubert* analysis in *General Electric Co. v. Joiner*, 522 U.S. 136, 118 S. Ct. 512 (1997). In *Joiner*, the Court acknowledged that while the *Daubert* focus is "on principles and methodology," as opposed to the "conclusions that they generate," conclusions and methodology are not entirely distinct from one another:

Although trained experts commonly extrapolate from existing data, nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.

Id. 519.

III. PLAINTIFFS HAVE THE BURDEN OF PRODUCING SCIENTIFICALLY RELIABLE EVIDENCE ESTABLISHING THAT WELDING FUMES CAUSE OR ACCELERATE THE ONSET OF PARKINSON'S DISEASE.

Plaintiffs must produce scientifically reliable expert testimony to establish that exposure to welding fumes is capable of causing or accelerating the onset of PD. As proponents of such evidence, they have the burden of establishing its admissibility "by a preponderance of proof." *Daubert*, 509 U.S. at 593 n.10; *Nelson v. Tennessee Gas Pipeline Co.* 243 F.3d 244, 251 (6th Cir. 2001). Although such scrutiny will "inevitably, on occasion . . . prevent the jury from learning of authentic insights and innovations," *Daubert*, 509 U.S. at 595, the Supreme Court has obviously deemed this less

⁵ Courts also regularly add their own additional factors. See Federal Judicial Center, Reference Manual on Scientific Evidence, 19-21 (2d ed. 2004). Several courts, for example, have also considered whether an opinion has been put to any non-judicial uses and/or whether it was developed solely for the purposes of litigation. See *Smelser v. Norfolk S. Ry. Co.*, 105 F.3d 299, 303 (6th Cir.1997).

objectionable than dumping a barrage of questionable scientific evidence on a jury who would be less capable than a judge of undertaking a critical assessment of its reliability.

IV. PARKINSON'S DISEASE

First described in 1817 by James Parkinson, PD is a slowly progressive degenerative movement disorder. Declaration of C. Warren Olanow M.D., FRCPC, attached hereto as Exhibit 1, at ¶20. PD is associated with a reduction in dopamine due to the degeneration of neurons in the *substantia nigra pars compacta*, the portion of the brain where dopamine is synthesized. *Id.* at ¶21. When the loss of dopamine exceeds a critical threshold, the brain is unable to smoothly coordinate body movements. *Id.* As a result, individuals with PD demonstrate symptoms that include tremors, rigidity, bradykinesia (slowness of movement), impaired righting reflexes, and fixed posture. *Id.*

Medical professionals estimate that PD affects between 1 and 2% of the general population; that approximately one million Americans have already been diagnosed with PD; and that 60,000 new cases will be diagnosed in the United States each year. *Id.* at ¶27. While some individuals have been diagnosed with PD prior to the age of 30, onset of the disease generally occurs between 40 and 70 years of age. As many people now live well into their 80s due to advances in medicine and public health, the prevalence of PD is increasing.

The National Institute for Occupational Safety and Health (NIOSH) estimates that there are more than 400,000 full-time welders in the United States. *Id.* at ¶28. As members of the general population, between 4,000 and 8,000 of these welders will develop PD during their lifetime on the basis of chance, alone. *Id.* at ¶29. Since the number of individuals who perform intermittent welding is substantially greater than

those who weld full-time, the total number of “welders” who will develop PD (on the basis of chance alone) will likewise be substantially greater. *Id.*

V. NO RELIABLE EVIDENCE SUPPORTS A CAUSAL CONNECTION BETWEEN WELDING FUMES AND THE DEVELOPMENT OR ACCELERATED ONSET OF PARKINSON’S DISEASE

A. PLAINTIFFS MUST FIRST ESTABLISH GENERAL CAUSATION BEFORE ESTABLISHING SPECIFIC CAUSATION.

‘General causation’ is concerned with whether an agent (e.g., welding fume) is capable of causing a particular harm (e.g., PD) in a group and not whether the agent caused any particular individual’s harm. Restatement (Third) of Torts: Liability for Physical Harm § 28 cmt. C (Tent. Draft Nov. 3, 2003).⁶ By contrast, ‘specific causation’ addresses the question of whether a particular exposure was the cause of the harm in a specific individual. *Id.*; *see also* Federal Judicial Center, Reference Manual on Scientific Evidence (2nd ed. 2004) at 395-96. One process by which physicians consider specific causation in an individual is called ‘differential diagnosis’ or ‘differential etiology.’ It is a method whereby a medical expert considers the possible causes of a disease and, based on the risk factor for a specific individual, “rules out” each cause until the probable cause is left. *Id.*

Most courts that have considered the issue – including the Sixth Circuit – require that an expert first establish general causation *before* opining that specific causation exists. *See Downs v. Perstorp Components, Inc.*, 126 F.Supp.2d 1090 (E.D.Tenn. 1999), *aff’d*, 26 Fed.Appx. 472, 2002 WL 22000 (6th Cir. 2002); *See also Hollander v. Sandoz*

⁶ Several provisions in the Restatement (Third) of Torts: Liability for Physical Harm (Basic Principles), which has not yet been completed and published, have been tentatively approved by the membership and therefore constitute official ALI policy. Courts have already used these provisions. *See, e.g., SL Service, Inc. v. U.S.*, 357 F.3d 1358, 1362 (Fed. Cir. 2004). These tentative drafts and their provisions are available in Westlaw in the *Forms, Treatises, CLEs and Other Practice Material Directory* and in Lexis in the *Secondary Legal > Restatements > Torts > Restatement 3d, Torts – Drafts Directory*.

Pharms. Corp., 289 F.3d 1193, 1211 (10th Cir. 2002); *Raynor v. Merrell Dow Pharms., Inc.*, 104 F.3d 1371, 1375-76 (D.C. Cir. 1997); *Meister v. Medical Engineering Corp.*, 267 F.3d 1123 (D.C. Cir. 2001); *Jones v. United States*, 933 F. Supp. 894, 900-01 (N.D. Cal. 1996), *aff'd*, 127 F.3d 1154 (9th Cir. 1997); *Bonner v. ISP Tech.*, 259 F.3d 924 (8th Cir. 2001); *In re Breast Implant Litig.*, 11 F. Supp.2d 1217, 1224, 1230 (D. Colo. 1998); *Hall v. Baxter Health Care Corp.*, 947 F. Supp. 1387, 1412-14 (D.Or. 1996); *Haggerty v. Upjohn Co.*, 950 F. Supp. 1160, 1166-67 (S.D. Fla. 1996); *National Bank of Commerce v. Associated Milk Producers, Inc.*, 22 F. Supp. 2d 942, 963 (E.D. Ark. 1998); *Wynacht v. Beckman Instruments, Inc.* 113 F. Supp. 2d 1205 (E.D. Tenn. 2000); *Nelson v. Am Home Prods. Corp.*, 92 F. Supp. 2d 954, 969 (W.D. Mo. 2000); *Cavallo v. Star Enterprise*, 892 F.Supp. 756 (E.D. Va. 1995), *aff'd in part, rev'd in part*, 100 F.3d 1150 (4th Cir. 1996); *Minnesota Mining & Mfg. Co. v. Atterbury*, 978 S.W.2d 183, 193 (Tex. Ct. App. 1998).

In *Cavallo* the court outlined this position as follows:

The process of differential diagnosis is undoubtedly important to the question of “specific causation”. If other possible causes of an injury cannot be ruled out, or at least the probability of their contribution to causation minimized, then the “more likely than not” threshold for proving causation may not be met. **But, it is also important to recognize that a fundamental assumption underlying this method is that the final suspected “cause,” remaining after this process of elimination, must actually be capable of causing the injury. That is, the expert must “rule in” the suspected cause as well as “rule out” other possible causes. And, of course, expert opinion on this issue of “general causation” must be derived from a scientifically valid methodology.**

Id. at 771, (emphasis added).⁷

Both the Reference Manual on Scientific Evidence and the Restatement (Third) of Torts outline the need for establishing general causation.

⁷ Potential causes are not eliminated during a differential diagnosis unless they have previously been identified as general causes. If it were otherwise, physicians and scientists would need to eliminate every agent under the sun as a potential cause, an absurd proposition.

A critical issue courts must address when asked to admit medical testimony on causation is whether the experts must present reliable evidence of general causation before they are permitted to testify as to specific causation. The majority position is that they must do so. They must “rule in” the substance, i.e., show that it is capable of causing the harm under consideration in some people, before they will be permitted to “rule out” other possible causes of the harm. This is the better rule.

Saks, *et al.*, Federal Judicial Center, Annotated Reference Manual on Scientific Evidence, (2d ed. 2004) at 637.

* * *

[E]vidence of general causation is a prerequisite to proof of specific causation. In toxic-substance cases, courts must determine what evidence is necessary to establish a prima facie case of general causation.

Restatement (Third) of Torts, *supra*, at § cmt. C.

See also Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS. 107, 110 n.13 (2001); Richard W. Wright, *Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts*, 73 IOWA L. REV. 1001, 1046 (1988) (“Thus, to prove that a specific condition was a cause of a particular result, one obviously must establish ... that some credible causal generalization links conditions of that type to results of that type.”).

Accordingly, without producing reliable, scientific evidence of general causation, plaintiffs cannot establish specific causation, namely that exposure to welding fumes caused PD in a particular plaintiff.

B. EXISTING EPIDEMIOLOGIC STUDIES DO NOT SUPPORT THE EXISTANCE OF GENERAL CAUSATION BETWEEN EXPOSURE TO WELDING FUMES AND PD.

1. Epidemiology⁸

As *Daubert* recognized, the scientific method involves *testing* hypotheses “to see if they can be falsified; indeed, this method is what distinguishes science from other fields of human inquiry.” 509 U.S. 593. One generally accepted method by which scientists test hypotheses is through epidemiologic studies. Epidemiology studies the distribution of diseases and their causes in human populations. Declaration of David H. Garabrant, M.D., MPH, attached hereto as Exhibit 2, at ¶9. The Reference Manual on Scientific Evidence describes epidemiologic evidence as follows:

Epidemiologic evidence identifies agents that are associated with an increased risk of disease in groups of individuals, quantifies the amount of excess disease that is associated with an agent, and provides a profile of a type of individual who is likely to contract a disease after being exposed to an agent. Epidemiology focuses on the question of general causation (i.e., is the agent capable of causing disease?) rather than that of specific causation (i.e., did it cause disease in a particular individual?). Since the early 1970's, epidemiological evidence has played an increasing role in evaluating the issue of general causation. This is due in large part to the growing number of mass torts, including those relating to asbestos, Bendectin, silicone implants and tobacco products.

Reference Manual on Scientific Evidence at 336-337. Two primary forms of epidemiologic studies are ‘cohort’ studies and ‘case-control’ studies. Garabrant Decl. at ¶¶10-11. In both types of studies, epidemiologists compare groups of individuals. *Id.*

a. Cohort and Case-Control Studies

In a cohort study, the two groups consist of one exposed to the agent of interest and one that has not been exposed (a ‘referent’ group). *Id.* at ¶10. The epidemiologist

⁸ Much of the following discussion about epidemiologic studies, their methodology, results and potential for error is excerpted from the Declaration of David H. Garabrant, M.D., MPH and the Reference Manual on Scientific Evidence. Both sources provide a more detailed explanation that may assist the Court in understanding the existing body of evidence about the relationship between welding fumes and PD.

compares the rate at which the exposed group contracts the disease being studied with the rate that the non-exposed referent group contracts the disease. If the exposed group has a higher rate of disease than the referent group, an association exists. *Id.*

By contrast, in a ‘case-control’ study, the epidemiologist chooses subjects based on whether they have a particular disease, such as PD. *Id.* at ¶11. Individuals with the disease comprise the ‘cases.’ *Id.* Individuals who do not have the disease make up the ‘controls.’ *Id.* The epidemiologist compares the exposure rate among cases to the agent of interest to the exposure rate among the controls. If the rate of exposure among the cases is higher than among the controls, an association exists. *Id.*

b. Establishing the Existence of an Association.

Whether an epidemiologic study establishes that a specific agent, such as welding fumes, is capable of causing a particular disease, such as PD, depends in the first instance on whether the study shows an ‘association’ between exposure to the agent and the disease. Reference Manual on Scientific Evidence at 336-338; Garabrant Decl. at ¶12. An association exists when the two occur together more frequently than one would expect by chance. *Id.*; Annotated Reference Manual on Scientific Evidence, *supra.*, at 488.

c. Assessing the Reliability of an Association.

The absence of an association precludes a finding that a causal relationship exists. On the other hand, the finding of an association does *not* necessarily establish a causal relationship. Garabrant Decl. at ¶12; Reference Manual on Scientific Evidence at 336-37:

However, it should be emphasized that **an association is not equivalent to causation**. An association identified in an epidemiologic study may or may not

be causal. Assessing whether an association is causal requires an understanding of the strengths and weaknesses of the study's design and implementation, as well as a judgment about how the study findings fit with other scientific knowledge.

Id. (emphasis added). Although there may be an association between rising costs of gasoline and the number of teenagers playing lacrosse, no one would seriously suggest that the cost of gasoline has inspired our youth. The association in that scenario would merely be coincidence; two unrelated events occurring simultaneously over time. To determine whether an association supports a causal relationship, epidemiologists must test the association for the existence of bias, confounding and chance. *Id.* at 354; Garabrant Decl. at ¶12.

Bias indicates the existence of a systematic error that affects the entire study and its results. *Id.* at ¶16. A study may be affected by an 'observation bias' (i.e., a bias created by a disparate quality or intensity of measurements between the observed and referent populations) or by a 'selection bias' (i.e., a bias created by disparate criteria for sampling from the exposed and referent populations). The following example illustrates selection bias:

A researcher is investigating an association between petroleum refineries and the development of prostate cancer. Refinery employees who comprise the study's exposed population are identified based upon their complaints of painful urination. The referent population consists of individuals who have been randomly selected from the local telephone directory. The researcher then compares the incidence of cancer in both the observed and referent groups.

Id. The selection of the exposed population is biased because of the criteria employed of painful urination. This is likely to identify a population more likely to have prostate cancer than the remainder of the exposed population, which will produce skewed results.

Confounding occurs when there is some factor, other than the agent being studied, that distorts the association between an exposure and an outcome (e.g., age would be a

confounding factor in a study associating gray hair and death, because gray hair does not cause death, but is differentially associated with age, which does). *Id.* at ¶17. Finally, chance refers to the possibility that a finding of a positive association may represent the result of random error rather than a true association. *Id.* ¶18. Chance is illustrated by a fair coin yielding five heads out of five tosses. Reference Manual on Scientific Evidence at 357.⁹ To assess whether the finding of an association is attributable to a chance occurrence, epidemiologists calculate the ‘statistical significance’ of the study’s results in addition to the study’s ‘confidence interval’. Garabrant Decl. at ¶18. The absence of a statistically significant association precludes the finding of a causal relationship. *Id.*

Even if epidemiologists determine that an association is reliable after evaluating for potential bias, confounding and chance, they will proceed to examine numerous additional factors before concluding that an association demonstrates a causal relationship. These factors include: (1) strength of the association; (2) specificity of the association; (3) replication of the findings; (4) consideration of alternative explanation; (5) biological plausibility; (6) dose-response relationships; (7) consistency with other knowledge; (8) cessation of exposure; and (9) temporal relationship.¹⁰ *Id.* ¶20.

⁹ Because random error can, in part, be attributed to sample size, by enlarging the sample size (e.g., tossing 1,000 coins as opposed to five), the outcome of a study is less likely to be influenced by chance. *Id.*

¹⁰ These factors reflect guidelines proposed by the U.S. Surgeon General in 1964. Cus. Dep’t. of Health Educ., and Welfare, Public Health Serv., Smoking and Health: Report of the Advisory Committee of the Surgeon General (1964). They were further refined in Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. Royal Neward Soc’y Med. 295 (1965). These factors thus have come to be known as the “Bradford Hill” factors. See also Mervyn Susser, *Causal Thinking in the Health Sciences: Concepts and Strategies in Epidemiology* (1973); *In re Joint E. & S. Dist. Asbestos Litig.*, 52 F.3d 1124, 1128-30 (2d Cir. 1995).

2. Epidemiologic Studies Examining the Relationship between Welding Fumes and PD Have Found No Reliable Association.

Over the past 60 years, researchers have conducted numerous epidemiologic studies of the health of welders and the causes of PD. These researchers uniformly have found that there is no statistically significant association between welding fume and PD. Given the absence of a statistically significant association, only one conclusion can be drawn from the existing scientific literature: no evidence supports the proposition that exposure to welding fumes causes or accelerates the onset of PD.

a. The Tsui Study¹¹ Does Not Report an Association

For example, a 1999 case-control study by Dr. Joseph Tsui evaluated whether several occupations, including welding, were associated with an increased risk of PD among the general population of Vancouver, British Columbia, Canada. The occupations of 441 PD cases and 6,659 controls (who did not have PD) were ascertained. Although Dr. Tsui reported associations between PD and numerous occupations, welding was not among those found to be associated with PD. This very large study does not support a conclusion that welders are at increased risk of PD.

b. The Tanner Study¹² Does Not Report an Association

A 2003 case-control study by Dr. Caroline Tanner similarly evaluated whether welding was associated with an increased risk of PD. Dr. Tanner's study consisted of World War II veteran twin pairs where one twin had PD and the other twin did not. Dr. Tanner's study found that while teachers and health-care workers were shown to have an

¹¹ Garabrant Decl. at ¶22(a); Tsui, *et al.*, *Occupational Risk Factors in Parkinson's Disease*, 90 *Canadian Journal of Public Health* 334-337 (1999).

¹² Garabrant Decl. at ¶22(b); Tanner, *et al.*, *Occupation and Risk of Parkinson's Disease (PD): A Preliminary Investigation of Standard Occupational Codes (SOC) in Twins Discordant for Disease* (Abstract: S49.001).

increased risk, perhaps because of their exposure to infectious diseases, individuals who performed welding did not show *any* increased risk of PD.

c. The Goldman / Olanow Study¹³ Does Not Report an Association

Likewise, Drs. Goldman and Olanow, et al. recently surveyed the medical records of 1087 consecutive PD patients seen between 2000 and 2002 at the Parkinson's Institute in Sunnyvale, CA. in order to determine if welding was associated with an earlier age of PD onset. Of the 885 individuals for whom information regarding occupation and age of PD onset was available, 11.8% were teachers; 10.5% were administrative workers; 9.9% were engineers; 9.1% were sales workers; 8.7% were managers; 3% were construction workers; 0.5% were machinists and 0.2% were welders. Although 122 of the 885 (13.8%) had PD onset by the age 50, the mean and median ages of onset were 64.3 and 65 years, respectively. The authors specifically concluded that welding was not associated with an earlier onset of PD in the clinical population they studied.

d. The Dreessen Study¹⁴ Does Not Report an Association.

More than 60 years before the Tanner and Goldman/Olanaw studies, Dr. Waldemar C. Dreessen extensively examined the health of thousands of World War II welders. The Dreessen study presented the results of an investigation by the U.S. Maritime Commission into the health hazards of a group of shipyard arc welders who engaged in mild steel and galvanized steel welding. A total of 4650 individuals from seven different shipyards received physical examinations—one fourth of this group was

¹³ Garabrant Decl. at ¶22(c); Samuel M. Goldman, et al., *Occupation and PD Onset Age in a Movement Disorders Clinic*, (Abstract: S08.006, April 27, 2004).

¹⁴ Garabrant Decl. at ¶22(d); Waldemar C. Dreessen, Senior Surgeon et al., U.S. Public Health Service Bulletin No. 298, *Health of Arc Welders in Steel Ship Construction*, (1947).

comprised of non-welders who served as the control group. A total of 246 workers had done some welding before coming to work at the shipyard. Fume and gas concentrations in the air were measured, and the report stated that mild steel welding fume contains, among other compounds, 2-12% manganese oxide. Although the report primarily addressed respiratory and cardiovascular health issues, significantly, no neurological problems were noted among such a large group of welders who engaged in a significant amount of welding. Dr. Dreessen credited safety and ventilating procedures in the shipyards as a material reason for the welders' lack of diseases of industrial origin.

e. The Walrath Study¹⁵ Does Not Report an Association.

In a 1985 cohort study, Dr. Judy Walrath examined mortality patterns among U.S. Veterans classified by occupation. Despite the high level of welding that took place during World War II, PD was not the cause of death in any of the 771 veterans classified as a welder or flamecutter.

f. The Coggon Study¹⁶ Does Not Report an Association.

In 1995, Coggon, *et al.*, performed a mortality study on a cohort of welders, among other occupations, in England and Wales between 1979 and 1990. Like Dr. Tanner's findings, Coggon found an increased mortality rate among teachers due to PD but did *not* find increased mortality rates associated with any other occupation.

¹⁵ Garabrant Decl. at ¶22(e); Judy Walrath, Ph.D. et al., Nat'l Inst. of Health, Pub. No. 85-2756, *Mortality Patterns Among U.S. Veterans By Occupation and Smoking Status* (1985)

¹⁶ Garabrant Decl. at ¶22(f); Drever, *Occupational Health Decennial Supplement*. Series DS no.10 ed. London: HMSO; 1995 at (Chapters 4, 5 and App. 7-8) Coggon, et al., *Occupational Mortality of Men*.

g. The Milham Study¹⁷ Does Not Report an Association.

Finally, in 1997, Dr. Samuel Milham performed a cohort study of manganese and PD in which he analyzed the death certificates of 588,090 men and 88,071 women who died in Washington State between 1950 and 1989. Dr. Milham found only four cases of PD among welders and flamecutters -- roughly 8 were expected -- and found no statistically significant association between welding and PD.

In sum, although many researchers have studied the issue for decades, none has reported a statistically significant association between welding fume and the onset or acceleration of PD. As such, the medical and scientific communities do not generally accept that exposure to welding fumes causes or accelerates, PD. *See Olanow Decl.* at ¶ 34.

3. Epidemiologic Studies, Examining the Relationship between Manganese and PD, Have Found No Reliable Association.

Not only have epidemiologic studies, examining the relationship between welding and PD, failed to find a reliable association, but, as explained below, epidemiologic studies examining the relationship between exposure to manganese (from any source) and PD, have similarly failed to establish a reliable association.

a. The Semchuk Study¹⁸ Does Not Report an Association.

In 1993, Semchuk, *et al.*, performed a population-based, case-control study of PD in the city of Calgary, Alberta, Canada, to test for an association between the development of PD and family histories and various occupational exposures, including

¹⁷ Garabrant Decl. at ¶22(g); Milham, *Occupational Mortality in Washington State 1950-1989*, U.S. Department of Health and Human Services, NIOSH, Order No. 00913725 (1997).

¹⁸ Garabrant Decl. at ¶23(b); Semchuk, *et al.*, *Parkinson's Disease: A Test of the Multifactorial Etiologic Hypothesis*, 43 *Neurology* 1173 (1993).

manganese. After conducting a univariate analysis, Semchuk did not find any association between occupational exposure to manganese and PD. Rather, she found a positive association between PD and the following four factors: family history of PD, history of head trauma, history of occupational herbicide use, and family history of essential tremor.

b. The Gorell Studies¹⁹ Do Not Report a Statistically Significant Association.

In 1999, J. M. Gorell *et al.*, conducted a population-based case-control study of PD and occupational exposure to numerous toxins, including: manganese, copper, iron, lead, mercury, and zinc. One hundred and forty-four (144) cases and 464 controls provided occupational histories that were reviewed by an industrial hygienist who was blinded to disease status of the cases and controls. Although the authors tentatively found that occupational exposure to manganese in excess of 20 years could have an association with an increased risk of PD, because of the small sample (which relied on only 3 cases and 1 control – none of whom were welders), the authors suggested that a further, more detailed study was necessary.

Notably, in a 2004 follow-up article by Gorell, *et al.*, the authors reported the results of a multivariate logistic regression analysis of their 1999 data. After adjusting for age, sex and race, the authors did not find exposure to manganese to be statistically significantly associated with PD.

¹⁹ Garabrant Decl. at ¶23(e); Gorell *et al.*, *Occupational Exposure to Manganese, Copper, Lead, Iron, Mercury, and Zinc and the Risk of Parkinson's Disease*, 20 *NeuroToxicology* 239 (1999); Gorell, *et al.*, *Multiple Risk Factors for Parkinson's Disease*, 217 *Journal of Neurological Sciences* 169 (2004).

c. The Vieregge²⁰ Study Does Not Report an Association.

In 1995, Vieregge *et al.*, conducted a cohort study to test the neurological impact of chronic exposure to manganese through well water. Individuals studied were over the age of 40 and had principally relied upon their wells for drinking water for at least 10 years. Forty-one (41) individuals whose well water had manganese concentrations of 0.300 mg/l or higher (cases) were compared with 74 individuals whose well water had manganese concentration that never exceeded 0.05 mg/l (controls). All participants were interviewed for medical and occupational history and underwent a complete standardized neurological exam by the same neurologist. The authors did not observe significant differences in the two groups with respect to symptoms set forth by the Columbia University Rating Scale, aiming, steadiness, line pursuit and tapping. As a result, the authors concluded that exposure to manganese in drinking water is not a risk factor for PD.

d. The Seidler Study²¹ Does Not Report an Association.

Finally, in 1996, Seidler, *et al.*, performed a case-control study of PD, enrolling 380 patients from nine German neurologic clinics, 379 neighborhood controls and 376 regional controls. Although five cases and one control reported earlier exposure to manganese, the authors did not report an association between such exposure and PD.

In sum, although numerous epidemiologic studies have looked for a reliable association between manganese exposure and PD, none has been found. As such, the

²⁰ Garabrant Decl. at ¶23(g); Vieregge, et al., *Long Term Exposure to Manganese in Rural Well Water Has No Neurological Effects*, 22 Can. J. Neurol. Sci. 286 (1995).

²¹ Garabrant Decl. at ¶23(c); Seidler *et al.*, *Possible Environmental, Occupational, and Other Etiologic Factors for Parkinson's Disease: A Case Study in Germany*, 46 Neurology 1275 (1996).

medical and scientific communities do not generally accept that manganese exposure causes or accelerates, PD. *See* Olanow Decl. at ¶ 38.

C. IN LIGHT OF THE EPIDEMIOLOGIC STUDIES THAT FAIL TO REPORT AN ASSOCIATION BETWEEN WELDING FUMES AND PD, PLAINTIFFS MUST PRODUCE EPIDEMIOLOGIC DATA IN SUPPORT OF GENERAL CAUSATION.

Where, as here, contrary epidemiologic data exist, plaintiffs *must* produce epidemiologic data in support of causation. *Conde v. Velsicol Chem. Corp.*, 24 F.3d 809 (6th Cir. 1994):

Nineteen epidemiologic studies in humans have found little evidence of long-term adverse health effects from chlordane doses hundreds of times higher than those the Condes were subjected to under a worst-case scenario. Although the Condes cite published critiques of these studies, the critiques only underscore the need for further studies, and do not, as the district court noted, establish causation. Finally, the Condes' reliance on a 1987 draft Technical Support Document prepared by the Environmental Protection Agency, listing chlordane as a probable human carcinogen four years after the Condes' home was treated with Velsicol's termiticide, is misplaced. That document, based primarily on animal studies, concludes that "none of the available epidemiology studies of the chlorinated cyclodienes are adequate to establish either a negative or positive association between chlorinated cyclodiene exposure and carcinogenic risk."

Id. at 813-14 (citation to the district court's opinion omitted); *see also Raynor v. Merrell Dow Pharms., Inc.*, 104 F.3d at 1374 (where epidemiological evidence was to the contrary, it was not methodologically sound to draw causal conclusions from chemical structure, in-vivo animal studies and in-vitro studies); *Meister v. Medical Engineering Corp.*, 267 F.3d. 1123, 1130 (D.C. Cir. 2001) ("[C]hemical, in vitro, and in vivo . . . [s]tudies . . . singly or in combination, are not capable of proving causation in human beings in the face of the overwhelming body of contradictory epidemiological evidence."); *Raynor v. Merrell Dow Pharms., Inc.* 104 F.3d at 1371, 1374; *Lynch v. Merrell - National Lab.*, 646 F. Supp. 856 (D. Mass. 1986), *aff'd* 830 F.2d 1190 (1st Cir.

1987); *Allison v. McGhan Medical Corp.*, 184 F.3d 1300, 1314 (11th Cir. 1999); *Brock v. Merrell Dow Pharms., Inc.*, 874 F.2d 307, 313 (5th Cir. 1989); *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. at 1387.

When epidemiologic evidence exists, it is, all other things being equal, the most probative evidence of general causation. *Soldo v. Sandoz Pharms. Corp.*, 244 F. Supp. 2d 434 (W.D.Pa. 2003); *Pozefsky v. Baxter Healthcare Corp.*, 2001 WL 967608 (N.D.N.Y. Aug 16, 2000); *In re Breast Implant Litigation*, 11 F. Supp.2d 1217, 1224 (D.Colo. 1998) (citing additional cases); *In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1223 (E.D.N.Y. 1985):

A number of sound epidemiological studies have been conducted on the health effects of exposure to Agent Orange. These are the only useful studies having any bearing on causation. All the other data supplied by the parties rests on surmise and inapposite extrapolations from animal studies and industrial accidents. It is hypothesized that, predicated on this experience, adverse effects of Agent Orange on plaintiffs might at some time in the future be shown to some degree of probability. The available relevant studies have addressed the direct effects of exposure on servicepersons and the indirect effects of exposure on spouses and children of servicepersons. No acceptable study to date of Vietnam veterans and their families concludes that there is a causal connection between exposure to Agent Orange and the serious adverse health effects claimed by plaintiffs. Chloracne and porphyria cutanea tarda are the only two diseases that have been recognized by Congress as having some possible connection to Agent Orange exposure, but no proof has been shown of any relationship of these diseases to these plaintiffs.

Id. at 1231, *aff'd*, 818 F.2d 187 (2d Cir. 1987), *cert. denied*, 487 U.S. 1234 (1988).²²

Since Judge Weinstein's opinion in *In re Agent Orange*, courts have tended to favor epidemiological evidence over various types of other evidence, including, but not

²² See also: David E. Bernstein, *The Admissibility Of Scientific Evidence After Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 15 Cardozo L. Rev. 2139, 2166 (1994); Joseph Sanders, *From Science to Evidence: The Testimony on Causation in the Bendectin Cases*, 46 Stan. L. Rev. 1, 25 (1993); Susan R. Poulter, *Science And Toxic Torts: Is There A Rational Solution To The Problem Of Causation?*, 7 High Tech. L.J. 189, 201 (1992); Michael D. Green, *Expert Witnesses And Sufficiency Of Evidence In Toxic Substances Litigation: The Legacy Of Agent Orange And Bendectin Litigation*, 86 Nw. U. L. Rev. 643, 646 (1992).

limited to, animal studies, in vitro research, and structure – activity analysis. Reference Manual on Scientific Evidence at 347.

D. CONTROLLED EPIDEMIOLOGIC STUDIES ARE LIKEWISE ESSENTIAL IN CASES, SUCH AS THIS, INVOLVING DISEASES WITH UNKNOWN ETIOLOGY.

Moreover, in a case, such as this one, that involves a disease with a significant ‘background’ rate, controlled epidemiologic studies are essential to establishing general causation. The ‘background’ rate is the rate at which an adverse health event, such as PD, occurs in an unexposed population. Reference Manual on Scientific Evidence at 388. Because the background rate for the development of PD, for example, is between 1% and 2%, by chance alone, 1%-2% of welders will develop PD. But for a very small percentage of cases where genetic factors have been implicated (under 1,000 worldwide), scientists have been unable to identify the cause(s) of PD since its discovery in 1817. (Olanow Decl. at ¶20). This problem of unidentified risks (often termed “background cases of unknown etiology”) has been recognized in a number of decisions. For example, in *In re Breast Implant Litigation*, 11 F. Supp. 2d at 1230, the court disapproved of a physician’s identification of silicone as the cause of the plaintiff’s disease through differential diagnosis, stating:

As a practical matter, the cause of many diseases remains unknown; therefore a clinician who suspects that a substance causes a disease in some patients very well might conclude that the substance caused the disease in the plaintiff simply because the clinician has no other explanation.

Id. See also *National Bank of Commerce v. Dow Chem., Co.*, 965 F. Supp. 1490 (E.D. Ark. 1996) (rejecting testimony that pesticide caused birth defects where witness acknowledged that causes are unknown for 70%-80% of birth defects), *aff’d.*, 133 F.3d 1132 (8th Cir. 1998); *Whiting v. Boston Edison Co.*, 891 F. Supp. 12 (D. Mass. 1995)

(witness's acknowledgement that 90% of cases of lymphocytic leukemia are of unknown cause cast doubt on "differential diagnosis" of cause); *In re "Agent Orange" Prod. Liab. Litig.*, 611 F. Supp. at 1250 ("Central to the inadequacy of plaintiffs' case is their inability to exclude other possible causes of plaintiffs' illnesses – those arising out of their service in Vietnam as well as those that all of us face in military and civilian life.").

Thus, because a sizeable number of individuals in the general population develop PD as a result of unknown causes and because welders are part of that population, no conclusion concerning causation can be drawn from the mere fact that a welder develops PD. Garabrant Decl. at ¶21.

E. EPIDEMIOLOGIC STUDIES OFFERED BY PLAINTIFFS MUST BE SCRUTINIZED BY THE COURT.

Just as epidemiologic studies are scrutinized by epidemiologists, as outlined above, these studies, when offered as the basis for an expert's opinion, must also be scrutinized by the court before any inferences of causation can be drawn. If the study is found to be unreliable, it cannot form the basis for an opinion on causation. In *General Electric Co. v. Joiner*, 522 U.S. at 145, for example, the Supreme Court affirmed the exclusion of an expert's testimony on causation despite the expert's reliance upon four epidemiologic studies. *See also Newman v. Motorol Inc.*, 78 Fed.Appx. 292, 2003 WL 22407265 (4th Cir. 2003) (opinion based upon epidemiology excluded); *Allison v. McGhan Medical Corporation*, 184 F.3d 1300, 1315-16 (11th Cir. 1999) (four epidemiologic studies were excluded on the basis of "questionable methodology"); *Nelson v. Tennessee Gas Pipeline Co.*, 243 F.3d 244, 253 (6th Cir. 2001) (epidemiologic study held inadmissible for the failure to account for confounding factors); *Deluca v.*

Merrell Dow Pharm., Inc., 911 F.2d 941, 955 (3d Cir. 1990) (systematic bias and sampling error found in an epidemiologic study).

The fact that plaintiffs may produce an epidemiologic study that is published in a peer-reviewed journal will not insulate it from scrutiny. In *Daubert*, 509 U.S. at 593, the Supreme Court stated that “[p]ublication (which is but one element of peer review) is not a *sine qua non* of admissibility; it does not necessarily correlate with reliability.” In *Allison v. McGhan Med. Corp.*, 184 F.3d at 1315-17, the Eleventh Circuit affirmed a trial court’s decision to exclude the testimony of a witness who was relying upon an epidemiologic study, noting that “scrutiny by one’s peers does not insure admissibility”. The Manual on Scientific Evidence echoes this judicial consensus: “In short, ‘Peer review and Publication’ do not themselves establish the ‘reliability’ of the proffered knowledge.” Annotated Reference Manual on Scientific Evidence, *supra*. at 109.

Even if plaintiffs are able to produce an epidemiologic study that employs a reliable methodology, a single unreplicated study does not often establish general causation. “Rarely, if ever, does a single study conclusively demonstrate a cause-effect relationship. It is important that a study be replicated in different populations and by different investigators before a causal relationship is accepted by epidemiologists and other scientists.” Reference Manual on Scientific Evidence at 378.

VI. PLAINTIFFS CANNOT CARRY THEIR BURDEN OF PRODUCING RELIABLE EVIDENCE OF CAUSATION.

A. PLAINTIFFS' OWN EXPERTS CONCEDE THAT NO EPIDEMIOLOGIC DATA ESTABLISHING THAT WELDING FUMES ARE CAPABLE OF CAUSING OR ACCELERATING THE ONSET OF PD EXIST.

In support of their theory that welding fumes cause PD, Plaintiffs are expected to rely on an article authored by Dr. Brad A. Racette, *et al.*, entitled, "Welding-Related Parkinsonism: Clinical Features, Treatment, And Pathophysiology." B.A. Racette et al., *Welding-Related Parkinsonism: Clinical Features, Treatment, And Pathophysiology*, 56 *Neurology* 8 (2001) (Garabrant Decl. at Exhibit D). Dr. Racette's article concludes that "welding *may be* a risk factor for a parkinsonism syndrome that...is clinically indistinguishable from idiopathic PD except for age at onset. We *believe* that welding exposure acts as an accelerant to cause PD." *Id.* at 12 (emphasis added). The next lines of the article temper even this equivocal conclusion:

Further studies are necessary to clarify this important issue. A detailed clinical evaluation of career welders compared with age-matched controls in a proper epidemiologic study will be *essential* to prove the relationship between welding and parkinsonism.

Id. (emphasis added).

Scientists in the field of neurology have called even this suggestion into question, noting, among other things, that because the study was not a case-control study it "cannot be used to infer causation." Ravina *et al.*, Letter to the Editor, 57 *Neurology* 936, 396 (2001). In response to this criticism, Dr. Racette agreed that "our study more closely resembles a cross-sectional study. . . . Nevertheless, cross-sectional studies are, in general, useful for raising the question of the presence of an association rather than testing a hypothesis, which is consistent with our statement that 'welding *may be* a risk

factor for PD.”” Brad A. Racette, Reply from the Authors, 57 Neurology 936, 937 (2001)²³ (emphasis in original) (citations omitted); Neurology 57, Sept. (1 of 2) 2001.

Dr. Racette also has acknowledged the significant limits of his study during litigation. This year, for example, he testified that his 2001 study does *not* establish that exposure to manganese and welding fumes causes or accelerates the onset of PD:

Q. And you -- your study does not prove that exposure to manganese and welding fumes causes, contributes to or accelerates the onset of Parkinson's Disease, correct?

A. My study certainly suggests that it may accelerate the onset of Parkinson's, but it doesn't in any way answer those other questions.

Q. Your study does not definitively prove, though, that exposure to manganese and welding fumes accelerates the onset of Parkinson's?

A. That's correct.

Q. And your study doesn't prove that exposure to manganese or welding fumes causes or contributes to PD?

A. That's correct.

(Racette *Winterman* Dep., 101, 2/3/04; see also Racette *Elam* Dep., 26-28, 76-77, 4/15/03).

Dr. Racette also has confirmed the complete absence of epidemiologic evidence of causation:

Q. Doctor, in your reply to Dr. Sadek, you state that your article Welding-Related Parkinsonism is not an epidemiologic study?

A. Correct.

²³ Cross-sectional studies, in which the presence of both exposure and disease are determined at the same time, typically are not used to identify toxic agents, in part because causation cannot be determined without evidence that the exposure preceded the disease. See Reference Manual on Scientific Evidence at 343.

- Q. Okay. And would you also agree that no epidemiological study concludes that exposure to welding fumes causes a disease indistinguishable from idiopathic PD except for early onset?
- A. I'm not aware from my review of the literature of any epidemiologic study, but that doesn't preclude the studies that I'm not aware.
- Q. But you're not aware –
- A. I'm not aware of any.
- Q. Okay. Are you aware of any epidemiological study which shows that there is a statistically significant increased risk of PD in welders?
- A. I'm not aware of such a study.

(Racette *Elam* Dep., 97, 1/14/03).

Dr. Racette's testimony in 2003 confirms that, as of that date, no one had done the "detailed clinical examination of career welders" that he stated, in the conclusion to his 2001 study, would be "essential to prove the relationship between welding and parkinsonism." Racette *et al.*, *supra*, at 12. By his own admission, therefore, his 2001 study does not provide the necessary scientific evidence required to prove legal causation. Racette *Winterman* Dep., 66-67, (2/3/04). Indeed, Dr. Racette himself acknowledges that a link between welding and PD remains simply a "hypothesis". Racette *Winterman* Dep., 107, 2/3/04.

Although a June 2004 abstract (i.e. abbreviated summary) authored by Racette *et al.* describes a cross-sectional study that purports to find an association between welding and *parkinsonism*, that abstract provides insufficient information from which to draw any conclusions regarding the study's findings, and how they relate to PD. Garabrant Decl. at ¶23.²⁴

²⁴ Racette *et al.*, *Prevalence Of Parkinsonism In A Large Cohort Of Welders*, *Movement Disorders*, Vol 19, Suppl. 9, 2004 (Garabrant Decl. at Exhibit D).

Notably, in a February 2004 deposition, Dr. Paul Nausieda -- an expert who has worked extensively with the plaintiffs in the welding rod litigation, and who appeared on plaintiffs' tutorial submitted to this Court -- confirmed the absence of any published epidemiologic studies concluding that exposure to welding fumes resulted in an increase of PD. Deposition of Paul Nausieda, M.D. (February 25, 2004) at 57. Dr. Nausieda's admission confirms that the plaintiffs cannot produce competent scientific evidence to establish general causation. *See Conde v. Velsicol Chemical Corp.*, 24 F.3d 809, 814 (6th Cir. 1994) (finding no causation where EPA document concluded that none of the available epidemiologic studies concerning chlordane were adequate to establish either a negative or positive association between exposure and carcinogenic risk; and approving district court's finding that plaintiffs' critiques of epidemiologic studies finding little evidence of causation only underscored the need for further study and, therefore, did not establish causation); *Cadarian v. Merrell Dow Pharms., Inc.*, 745 F. Supp. 409, 412 (E.D. Mich. 1989) (no causation where study's authors themselves concluded that the results could not be interpreted without independent confirmatory evidence and could not be used even to "infer" that a particular drug causes a particular malformation, and plaintiffs' expert did not come forward with any independent evidence sufficient to confirm the findings in the study); *Sanderson v. International Flavor and Fragrances, Inc.*, 950 F. Supp. 981, 1004 (C.D. Cal. 1996) ("plausible hypotheses are not 'scientific knowledge' but the building blocks and catalysts of such knowledge").

Given the lack of epidemiologic support, it is not surprising that Dr. Nausieda admitted that the plaintiffs' alleged link between PD and welding fumes is not generally recognized in the scientific community. Nausieda Dep., 59, 2/25/04.

B. PLAINTIFFS MAY NOT ESTABLISH CAUSATION BY RELYING UPON CASE REPORTS.

In the absence of epidemiologic data, plaintiffs may seek to present evidence of isolated ‘case reports’ where an exposed worker reportedly developed PD or an early onset of PD. Case reports describe a single individual or a series of individuals who have coincident exposure and diseases and who are regarded by the observer as unusual in some way. Garabrant Decl. at ¶21. Although case reports can be useful in generating testable hypotheses, alone, they cannot be evaluated for the role of chance, bias or confounding. *Id.* As such, they are not regarded by the scientific community as providing evidence of general causation. *Id.*; see also Restatement (Third) of Torts, *supra.*:

[C]ase reports – reports of an instance of disease of an individual following exposure to a given agent – have been found insufficient by themselves as proof of general causation. See *Siharth v. Sandoz Pharms. Corp.*, 131 F. Supp, 2d 1347, 1361-62 (N.D. Ga. 2001).

In *Glastetter v. Novartis Pharmaceuticals Corporation*, 252 F.3d 986, 989-90 (8th Cir. 2001), the Eighth Circuit held that “[c]ase reports make little attempt to screen out alternative causes for a patient’s condition” and, although they may associate an exposure to one diagnosis of disease, “that association is not scientifically valid proof of causation.” See also *Brumbaugh v. Sandoz Pharm. Corp.*, 77 F. Supp. 2d 1153, 1156 (D. Mont. 1999) (case reports “have been rejected as reliable scientific evidence supporting expert opinion so as to meet the requirements” of *Daubert*); *Hall*, 947 F. Supp. at 1411 (case reports “are universally regarded as an insufficient scientific basis for a conclusion regarding causation because case reports lack controls”); *Cloud v. Pfizer, Inc.*, 198 F.

Supp.2d 1118, 1133-34 (D. Ariz. 2001) (“case reports do not provide reliable scientific evidence of causation”).

In view of the fact that 1-2% of the general population, including welders, will develop PD, case reports are of little value in establishing causation.

VII. EXCLUDING ALL TESTIMONY THAT WELDING FUMES CAUSE PARKINSON’S DISEASE WILL STREAMLINE THIS LITIGATION.

Given the lack of reliable scientific evidence supporting a link between welding fumes and the onset or acceleration of PD, an exclusionary ruling at this point is both necessary and proper. Moreover, it will substantially narrow the disputed issues, to the benefit of the Court and the litigants.

A. THE EXCLUSION OF SUCH TESTIMONY, NOW, IS CONSISTENT WITH THE COURT’S GATE-KEEPING FUNCTION AND IS PROCEDURALLY APPROPRIATE.

*“[T]he Areopagites . . . , finding themselves perplexed with a cause they could not unravel, ordered the parties to appear again in a hundred years.”*²⁵

In *Daubert*, the Supreme Court acknowledged that important differences exist between truth-seeking in the courtroom and in the laboratory: whereas scientific conclusions are subject to perpetual revision, the resolution of legal claims cannot be stalled in anticipation of future scientific investigation and research; instead, courts must resolve legal disputes finally and quickly on the basis of currently available scientific knowledge. *Daubert*, 509 U.S. at 597. As the Supreme Court has said:

The scientific project is advanced by broad and wide-ranging consideration of a multitude of hypotheses, for those that are incorrect will eventually be shown to be so, and that in itself is an advance. Conjectures that are probably wrong are of little use, however, in the project of

²⁵ Edward K. Cheng, *Changing Scientific Evidence*, 88 Minn. L. Rev. 315, citing William Hazlitt, *The Works of Michael de Montaigne: Comprising His Essays, Letters, and Journey Through Germany and Italy 504* (Charles Cotton trans., 1850)

reaching a quick, final and binding legal judgment--often of great consequence--about a particular set of events in the past.

Id. Or as another court has said:

In this case, the Court is mindful of the difficulties facing the plaintiff's experts in conducting their own tests and compiling data. Peer review has also been inhibited since publication has not occurred. The Court notes there is some possibility of future peer review. . . . The Court is aware that the future may shed more light on this matter. Medical science may one day determine with sufficient reliability that a causal relationship exists between a sustained prolonged QT interval and Propulsid but it is not there yet and may never be. A trial court must function in the present assessing evidence that presently exists.

In re: Propulsid Prod. Liab. Litig., 261 F. Supp. 2d 603, 615 (E.D. La. 2003) (citations omitted).

Nor is there any question that it is procedurally proper to exclude, at this time, all testimony that welding fume causes or accelerates the onset of PD. This Court, like other courts, has broad discretion to manage its docket by requiring plaintiffs to present *prima facie* evidence of their claims even *prior* to the commencement of discovery.²⁶ Other courts across the country have exercised that discretion under similar circumstances.²⁷ For example, prior to the commencement of discovery, a case management order may require a plaintiff to submit an affidavit from a physician, scientist, or other expert identifying the precise injury suffered by the plaintiff, the specific substance or product alleged to cause the injury, and the basis for the opinion that the injury was caused by the substance or product. *See, e.g., Lore v. Lone Pine Corp.* 1986 WL 637507 (N.J. Super. Ct., November 18, 1996); *Acuna v. Brown & Root, Inc.*, 200 F.3d 335 at 340 (5th Cir.

²⁶ Rule 16(c)(12) of the Federal Rules of Civil Procedure provides that “the court may take appropriate action with respect to . . . the need for adopting special procedures for managing potentially difficult or protracted actions that may involve complex issues, multiple parties, difficult legal questions, or unusual proof problems . . .”

²⁷ Joyce S. Meyers, *Focusing: When Less is More*, 28 Litig. 6,11 (2002).

2000) (dismissal of 1,600 claims upheld where plaintiffs failed to comply with court's order to establish certain elements of their claims (through expert affidavits) prior to commencement of discovery). Such early evaluation of plaintiffs' evidence is extremely effective in focusing attention on the dispositive scientific and technical issues and in refining those issues. *Id.* It also permits defendants to evaluate their case more accurately when determining such things as whether to move for summary judgment or whether to settle a case before expending tremendous financial resources.²⁸

B. EARLY AND EFFECTIVE GATEKEEPING IS ESPECIALLY IMPORTANT IN A MASS TORT SETTING.

In the context of mass tort litigation such as this MDL, the need for early and effective scientific gate-keeping is even more pronounced. It is a reality that many lawsuits are brought by hundreds of plaintiffs asserting every conceivable injury against every conceivable defendant without regard to actual liability and, oftentimes, without firm scientific bases for their claims.²⁹ Although defendants *ultimately* may prevail against a majority of scientifically meritless claims, that end often comes only after protracted litigation and the expenditure of tremendous financial resources -- both of which defendants should not be required to endure.

The silicone breast implant litigation aptly illustrates this calamity and the need for early and effective scientific gate-keeping. Women began suing implant manufacturers in 1982, claiming that leakage of silicone gel into their bodies was causing or exacerbating systemic illnesses, generally referred to as "atypical connective tissue

²⁸ Scott A. Steiner, *The Case Management Order: Use and Efficacy in Complex Litigation and the Toxic Tort*, 6 Hastings W.-N.W. J. Envtl. L. & Pol'y 71, 85 (1999).

²⁹ William A. Ruskin, *Prove it or Lose it: Defending Against Mass Tort Claims Using Lone Pine Orders*, 26 Am. J. Trial Advoc. 599 at 604.

disease” or “immune system disturbance.” *Hall v. Baxter*, 947 F. Supp. at 1391; *In re Breast Implant Cases*, 942 F. Supp. 958, 959 (E. & S.D.N.Y. 1996); Bernstein, *The Breast Implant Fiasco*, 87 Cal. L. Rev. 457, 461 (1999). In essence, plaintiffs claimed a unique constellation of hundreds of symptoms, including headache, fatigue, joint pain, and confusion--all of which are commonly experienced by the general population. *Hall v. Baxter*, 947 F. Supp. at 1391 n.2.

Armed primarily with anecdotal reports on the adverse health effects of silicone that were, at best, preliminary and only suggestive of a causal relation, several of these plaintiffs successfully prosecuted their cases and accumulated multi-million dollar verdicts against the industry.³⁰ These verdicts, together with an FDA ban of silicone gel breast implants pending epidemiologic review, combined to fuel a litigation explosion. Indeed, plaintiffs ultimately filed more than 400,000 cases in federal and state courts. *Hall v. Baxter*, 947 F. Supp. at 1391; *In re Breast Implant Cases*, 942 F. Supp. at 959.

The first well-regarded epidemiologic study testing the silicone-autoimmune disease relationship was not published until June 1994.³¹ It and subsequent studies concluded that implant recipients had *no* greater risk for developing disease than did the general population.^{32, 33} These findings, however, were made only *after* the threat of

³⁰ Nagareda, *In the Aftermath of the Mass Tort Class Action*, 85 Geo. L.J., 295, 331 (1996) (discussing the case of Maria Stern, who, in 1984, won the first verdict against Dow Corning in the amount of \$1.7 million after alleging that implants were responsible for causing her auto-immune disease).

³¹ Dresser, et al., *Breast Implants Revisited: Beyond Science on Trial*, 1997 Wis. L. Rev. 705, 712-713, citing Sherine E. Gabriel, et al., *Risk of Connective-Tissue Diseases and Other Disorders After Breast Implantation*, 330 New Eng. J. Med. 1697 (1994).

³² Nagareda, *supra*. note 30, at 331 (Roughly one in every one hundred women will develop some form of autoimmune disease. Thus, on the basis of chance alone, one could expect that about 10,000 of approximately 1 million women in the United States with breast implants will develop some form of autoimmune disease.)

thousands of massive verdicts lead to a multi-billion dollar settlement and one of the largest corporate bankruptcies in United States history.^{34, 35}

While the breast implant litigation is a cautionary tale, this Motion is itself a recognition that, at least in this case, the outcome can be different. Here, we are not dealing with tentative or uncertain science. We do not need to wait on the results of further studies. The answers are known. As defendants have demonstrated, numerous epidemiologic studies have consistently shown the lack of an association between either welding fumes or manganese and the onset or acceleration of PD. Given the scientific certainty, allowing such baseless claims to proceed would be an abdication of the Court's gate-keeping responsibilities.

C. THE EXCLUSION OF SUCH TESTIMONY WILL HAVE WIDESPREAD APPLICATION IN THIS PROCEEDING.

Finally, excluding testimony that welding fumes cause or accelerate PD will have widespread application to this case. Such a ruling would dispose of claims where plaintiffs allege that their PD is welding-related. It also would dispose of claims where plaintiffs allege that they are suffering from manganism, or some other form of

³³ In 1998, a group of scientists empanelled by Judge Samuel Pointer, who was presiding over thousands of cases that had been consolidated in the federal system, issued a report consisting of four chapters, each written by one panel member. *See* National Science Panel, *Silicone Breast Implants in Relation to Connective Tissue Diseases and Immunologic Dysfunction* ("National Science Panel Report"). The four Panel members reported that they did not find a sufficient scientific basis to link silicone implants to either connective tissue diseases or immune system dysfunctions. *See* Laurens Walker, *Scientific Authority: The Breast Implant Litigation and Beyond*, 86 Va. L. Rev. 801, 802 (2000).

³⁴ Cheng, *supra* note 25, at 316; *see also* Bernstein, *supra.*, 87 Cal. L. Rev. 457 at 459.

³⁵ The Bendectin Litigation (*In re Bendectin Products Liability Litigation*, 749 F.2d 300, 306 (6th Cir. 1984)) followed much the same course. *See*, e.g. Michael Green, *Bendectin and Birth Defects: The Challenges of Mass Toxic Substances Litigation* (1996); and Joseph Sanders, *Bendectin on Trial: A Study of Mass Tort Litigation* (1998) (Plaintiffs were successful in early litigation, based upon premature, suggestive evidence linking the drug prescribed for morning sickness with birth defects. Courts began dismissing claims only after subsequent, sounder studies, revealed that the drug had no such teratogenic effect.)

parkinsonism, if either the Court or a jury concludes, after weighing the evidence, that the plaintiff has PD. In addition, a ruling from the Court on the narrow evidentiary-medical issue presented by this Motion will streamline the discovery process and the presentation of evidence at trial by narrowing the issues on which expert testimony will be required.

VIII. CONCLUSION.

Pursuant to *Daubert* and its progeny and Federal Rules of Evidence, Rules 104(a), 702 and 703, plaintiffs have the burden of establishing, through scientifically reliable and relevant evidence, that welding fumes cause or accelerates the onset of PD. In light of 1) the dearth of epidemiologic studies finding otherwise, and 2) PD's high background rate of unknown etiology, however, this is a burden that plaintiffs cannot meet.

This Court, in fulfilling its gatekeeping obligation, must reject evidence that is scientifically unreliable. In the context of what promises to be protracted litigation, it is essential that the Court perform its gate-keeping function now.

For the foregoing reasons, defendants respectfully request that this Court exclude any and all testimony that welding fumes cause or accelerate the onset of PD.

Dated: June 17, 2004

Respectfully submitted,

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CERTIFICATE OF SERVICE

I hereby certify that on June 17, 2004, the foregoing was filed electronically. Notice of this filing will be sent to all parties by operation of the Court's electronic filing system. Parties may access this filing through the Court's system.

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