

IN THE CIRCUIT COURT OF OHIO COUNTY, WEST VIRGINIA

CHESTER EARL CARTER, et. al<sup>1</sup>,

Plaintiffs,

v.

CIVIL ACTION NO. 01-C-145

CSX TRANSPORTATION, INC.,

Defendant.

**COPY**

### MEMORANDUM OF OPINION

(Findings of Fact and Conclusios of Law  
/Gentry Hearing 2008)

The following Findings of Fact and Conclusions of Law are filed following the hearings conducted by this Court in conformity with Gentry v. Mangum, 195 W. Va. 512, 466 S.E.2d 171 (1995), regarding the methodological reliability of the

---

<sup>1</sup>The relevant plaintiffs are all those with pending solvent cases against CSX Transportation, Inc. in the State of West Virginia. The lead plaintiff for reference has always been the first of this group taken in alphabetical order. Plaintiff Chester Earl Carter was dismissed after the subject *Daubert/Gentry* hearing was ordered but, for ease of reference, his name has remained as the lead plaintiff. Defendant states that the remaining plaintiffs subject to Defendant's *Daubert/Gentry* motion are John Childers, Charles R. Clemons, Marvin Ferrell, Phillip Knipp, Jack C. Little, John C. Schneider and Kenneth Tschop, Jr.

hypothesis of permanent brain damage caused by long-term, low-dose occupational exposure to certain organic solvents.<sup>2</sup>

#### INTRODUCTION

1. This matter comes before the Court in an action captioned *Carter v. CSXT*, Nos. 00-C-205M, 01-C-145; 03-C-231; and 03-C-233. Plaintiffs in *Carter* and other similar actions pending against CSXT in this Court bring claims under the Federal Employers' Liability Act, 45 U.S.C. § 51 et seq. ("FELA"). Plaintiffs allege that while employed by CSXT, they were occupationally exposed to certain cleaning agents known as organic chlorinated solvents, and suffer as a result from a form of permanent brain dysfunction sometimes called "toxic encephalopathy."

2. The Court held an evidentiary hearing to evaluate the hypothesis propounded by plaintiffs that long-term, low-dose occupational exposure to certain solvents can cause toxic encephalopathy. This hearing was the culmination of several prior hearings at which plaintiffs were permitted to introduce expert testimony in support of the proposed hypothesis, but in

---

<sup>2</sup> The use of the phrase "low-dose occupational exposure" is used here to refer to the types and durations of exposures alleged by the plaintiffs in the above-captioned lawsuits. The particular allegations vary somewhat from plaintiff to plaintiff, but all involve exposures to solvents in an amount less than that required to produce an acute reaction of anoxia or hypoxia.

which this Court continued to express concern for the reliability of the evidentiary support behind that hypothesis.

3. The Court and the parties agreed upon a procedure for the present proceeding, whereby each side would have an opportunity to present evidence on the current state of science regarding the hypothesis. CSXT initially offered evidence at a hearing held on May 1, 2008. On that date, CSXT proffered the expert testimony of Dr. Kirk Frey, a neurologist. The Court then directed plaintiffs to come forward with expert scientific evidence supportive of the hypothesis, expressing a particular interest in hearing from a neurologist.

4. On January 14, 2009, plaintiffs produced expert testimony from Dr. Douglas Linz, an occupational medicine specialist (not a neurologist), via videotaped deposition testimony. At that same hearing, CSXT presented rebuttal evidence from Dr. James Albers, a neurologist.

5. The Court's findings and conclusions are as follows:

#### **I. LEGAL STANDARD**

6. For expert scientific evidence to be admissible, it must be both reliable and relevant. *Gentry v. Mangum*, 195 W. Va. 512, 518, 466 S.E.2d 171, 177 (1995). The admissibility of expert scientific evidence is governed in West Virginia by Rules

104 and 702 of the West Virginia Rules of Evidence.<sup>3</sup> Rule 104(a) "requires the proponent of the testimony to show by a preponderance that the evidence is admissible." *Gentry*, 195 W. Va. at 522 n.8, 466 S.E.2d at 181, n.8. Rule 702 requires that evidence proffered by a scientific expert must "assist the trier of fact" and that the expert must be qualified based on "knowledge, skill, experience, training, or education." W. Va. R. Evid. 702.

7. In evaluating the admissibility of scientific evidence under Rules 104 and 702, courts are to act as a "gatekeeper" to determine "whether the expert testimony reflect scientific knowledge, whether the findings are derived by scientific method, and whether the work product amounts to good science," in addition to ensuring that the proffered evidence is relevant to the issues before the court. *Gentry*, 195 W. Va. at 515, 466 S.E.2d at 174; see also *State v. Lockhart*, 208 W. Va. 622, 629, 542 S.E.2d 443, 450 (2000) (same).

8. Recent West Virginia opinions have affirmed the court's gatekeeper function:

[T]he trial court's initial inquiry must consider whether the testimony is based on an assertion or inference derived from the scientific methodology. Moreover, the testimony must be relevant to a fact at

---

<sup>3</sup> Rule 702 of the West Virginia Rules of Evidence is identical to the corresponding rule under the Federal Rules of Evidence.

issue. Further assessment should then be made in regard to the expert testimony's reliability by considering its underlying scientific methodology and reasoning. This includes assessment of (a) whether the scientific theory and its conclusion can be and have been tested; (b) whether the scientific theory has been subjected to peer review and publication; (c) whether the scientific theory's actual or potential error is known; and (d) whether the scientific theory is generally accepted within the scientific community.

*Jones v. Recht*, 221 W. Va. 380, 385 655 S.E.2d 126, 131 (2007).<sup>4</sup>

9. In essence, the court's role is to evaluate proffered evidence in order to keep "junk science" out of the courtroom. See, e.g., *id.* at 390, 136 (Davis, C.J. concurring in part, dissenting in part). A well-qualified expert must use "a methodology that is grounded in something more than rank speculation or imagination (like reading tea leaves, detecting auras, and the like)" in order to meet the threshold for

---

<sup>4</sup> In *Jones*, the Supreme Court of Appeals reversed exclusion of a neurologist's testimony, finding that instead of excluding the expert's testimony in its entirety, the court should attempt to demarcate and exclude the testimony of the neurologist that trespassed into areas of biomechanics in which the neurologist acknowledged that he was not qualified to provide. Here, in contrast to the issue in *Jones*, the primary question is one of methodology rather than qualification. Moreover, the *Jones* case involved testimony on widely accepted neurological conditions, rather than on an unproven theory of causation.

admissibility. *In re Flood Litig.*, 222 W. Va. 574, 668 S.E.2d 203, 211 (2008).<sup>5</sup>

10. Reliable scientific methodology, as opposed to junk science, is grounded in a reliable "process or method used to derive [the] principles and theories." *State v. Henning*, 212 W. Va. 128, 133, 569 S.E.2d 204, 209 (2002). It is based not merely upon the quantity of supporting published materials, but upon their quality. "[M]ere publication of an article is not the end of the peer review process." *Black v. Rhone-Poulenc, Inc.*, 19 F. Supp. 2d 592, 600 (S.D. W. Va. 1998); see also *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 593 (1993) ("Publication (which is but one element of peer review) is not the sine qua non of admissibility; it does not necessarily correlate with reliability.")

11. Reliable scientific methodology also requires that a hypothesis is falsifiable. As Justice Blackmun explained in *Daubert*, "[s]cientific methodology today is based on generating a hypotheses and testing them to see if they can be falsified." *Daubert*, 509 U.S. at 593 (quoting Michael D. Green, *Expert*

---

<sup>5</sup> The court in *In re Flood* reversed post-trial exclusion of a "standard methodology used in the engineering profession" to determine the effects of waterflows on land structures. 668 S.E.2d at 211-12. Again, unlike in *Jones* and *In re Flood*, the present issue involves speculative methodology used to support a novel causation hypothesis.

*Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation*, 86 NW. U. L. REV. 643, 645 (1992)). For the method to be reliable, there must be a way to prove the hypothesis false.

12. The scientific methodology does not necessarily have to be generally accepted, but it must be scientifically "valid" and "trustworthy." *Gentry*, 195 W. Va. at 521, 466 S.E.2d at 180; *see also Henning*, 212 W. Va. at 133, 569 S.E.2d at 209.

13. In sum, the "expert's opinion is admissible if the basic methodology employed by the expert in arriving at his opinion is scientifically or technically valid and properly applied." *Jones*, 221 W. Va. at 384, 655 S.E.2d. at 131 (emphasis added).

14. Under this legal framework, the issue presently before this Court is whether plaintiffs' proposed hypothesis - that of low-dose, solvent-induced toxic encephalopathy - is sufficiently reliable and relevant; is based upon scientifically valid and properly applied methodology; is falsifiable; and is valid and trustworthy.

15. On each of these points, it is plaintiffs who bear the burden of persuasion. W. Va. R. Evid. 104(a); *Gentry*, 195 W. Va. at 522 n.8, 466 S.E.2d at 181 n.8; *see also Daubert*, 509 U.S. at 592 n.10.

## II. BACKGROUND

### A. Events Leading Up To The Present Hearing.

16. The May 2008 and January 2009 hearings summarized in these findings of facts and conclusions of law are not the first this Court has conducted on the plaintiffs' proffered low-dose, solvent-induced toxic encephalopathy hypothesis. Rather, these most recent hearings were intended to address lingering questions this Court has raised about the reliability of the plaintiffs' theory.

17. Trials involving numerous plaintiffs have been conducted by the Court since the first *Daubert/Gentry* hearing in 1997 and the subject scientific hypothesis has been central to all of them. Trials included *Richard Damron and Ronald Damron v. CSXT*, Civil Action No. 99-C-414, *Harold Daniels v. CSXT*, Civil Action No. 99-C-280, *James Morrison v. CSXT*, Civil Action No. 01-C-145 (*Bierman Group* consolidated case number), *Arnold L. Dummitt, Chester R. Kibbey and Gary Jenkins v. CSXT*, Civil Action No. 01-C-145 (*Bierman Group* consolidated case number) and *Dummitt and Kibbey v. CSXT*, Civil Action No. 01-145 (re-trial following a hung jury). Defense verdicts were ultimately rendered in each case except that of Gary Jenkins. There, the Court granted the Defendant's motion for summary judgment. In each trial the parties presented expert testimony and the Court admitted scientific evidence relevant to the subject hypothesis.

18. In 1997, in *Allen v. CSXT*, No. 96-C-35, this Court held a *Daubery-Gentry* hearing on this same hypothesis. Plaintiffs proffered expert testimony from Dr. Douglas Linz, but did not offer any supporting testimony from a neurologist. The court in *Allen* recognized that the hypothesis was fiercely debated, but found it admissible. However, the court noted that "quite possibly in this case the reach of the law may exceed the grasp of medicine." *Allen*, Feb. 2, 1998, Order at p. 3. (A copy of the Opinion and Order is attached hereto as Exhibit 1).

19. Over the course of the *Allen* hearing, and in several subsequent FELA trials before this Court, the Court became aware of significant developments in the body of science related to the hypothesis at issue which called into question its reliability, as well as the reliability of the expert testimony previously proffered in its support.

20. One such development occurred in September 8, 2003, when NIOSH published a request for application ("RFA") entitled "Neurological Indices of Long Term Solvent Exposure in Workers." This September 8, 2003 RFA specifically set forth the limitations of previous studies related to the plaintiffs' hypothesis:

The limitations of previous studies are now better understood, and include selection bias, inadequately quantified exposure assessments, use of nonspecific neurological outcome variables, inclusion of individuals with neurological outcomes from

nonworking-related causes, and lack of sensitivity and specificity of neurodiagnostic tools.

21. NIOSH RFA, at p. 2. NIOSH's RFA further critiqued the significant biases and methodological flaws of the very studies plaintiffs had relied upon to support their theory (and continue to rely upon).

The neurological symptoms and objective findings of workers with long-term exposures to organic solvents are not specific to occupational exposures. They can often be attributed to other causes, such as medical conditions and other nonworking-related factors. Some case reports of neurologically impaired workers have used nonspecific symptoms (a measurement of effect) as surrogates for exposure, and individuals with nonworking-related causes of impairment have not been routinely excluded. Some studies have compared neurologically impaired workers who had a history of long-term or heavy solvent exposure with healthy unexposed workers. Nonworking-related causes in the differential diagnosis of the neurological conditions were not routinely ruled out, as would be warranted in medical practice. Such studies are biased to show that exposed persons are neurologically impaired. They do not prove that organic solvent exposure caused the neurological condition. Cross-sectional studies have not clearly established an association between long-term solvent exposure and chronic neurological impairment, and have not shown evidence of dose-response relationships.

NIOSH RFA, p. 2 (emphasis added). Indeed, it was precisely because the existing studies were too flawed to reliably support the theory of long-term solvent-induced encephalopathy that NIOSH sought new study proposals to test the theory.

22. In 2004, plaintiffs were again before this Court propounding their low-dose, solvent-induced toxic encephalopathy

hypothesis in *Bierman v. CSX Transportation*, No. 01-C-145. Plaintiffs called Dr. Alan Ducatman, an occupational medicine doctor, and Dr. Lisa Morrow, a neuropsychologist, but again did not offer any testimony from a neurologist. Plaintiffs' experts conceded that objective physiological evidence of their causation hypothesis was not available, but suggested objective evidence would soon be forthcoming in the form of a new research study. The evidence to which they referred was a study by Dr. Marc Haut. See *Bierman v. CSX*, No. 01-C-145, Testimony of Dr. Lisa Morrow, Gentry Hearing Transcript dated September 20, 2004 at 436-37, 482 (noting her ongoing work on a brain imaging study with Dr. Marc Haut and others and that "[w]e have some positive findings").

23. In July 2006, Dr. Marc Haut, Dr. Alan Ducatman, Dr. Lisa Morrow, and others published a paper entitled, "*Corpus Callosum Volume in Railroad Workers With Chronic Exposure to Solvent*" ("the Haut study") in the *Journal of Occupational and Environmental Medicine*. See *J. Occup. Environ. Med.* June 2006; 48(6); 615-24. Based on the Haut study's stated conclusions, this new study appeared as though it might provide plaintiffs with long promised support for their causation hypothesis beyond the sources discredited by NIOSH.

24. CSXT subpoenaed the underlying data from the Haut study so that it could evaluate its conclusions and methodology.

On November 8, 2006, the West Virginia attorney general moved to quash CSXT's request. On December 12, 2006, this Court denied that motion, thereby enabling CSXT to obtain the underlying Haut study data. In February 2007, CSXT provided that data to one of its neurology experts, Dr. Kirk Frey, for independent analysis.

**B. This Court Orders The Present Hearing.**

25. In May 2007, plaintiffs and CSXT were again before this court in another solvents related case in the re-trial of *Dummitt & Kibbey v. CSX*, No. 01-145. This Court expressed renewed concern with the reliability of the plaintiffs' scientific evidence, stating, "[t]his is very, very weak. It gets weaker as I try these cases." *Dummitt & Kibbey v. CSX*, No. 01-145, Hearing Transcript dated May 17 and 19, 2007 at 17. This Court concluded, based on its experience in *Dummit/Kibbey* and the course of the many other prior cases, that plaintiffs' hypothesis to date had insufficient "reliable relevant scientific evidence to support it." *Id.* at 25. "It is flawed science." *Id.* at 5. The Court decided to revisit the relevance and reliability of plaintiffs' proposed hypothesis with a new *Daubert/Gentry* hearing. *Id.* at 5-6, 17. At that time, the Court ordered the hearings which are the basis of these findings of fact and conclusions of law.

26. Specifically, the purpose of these new hearings was to evaluate the validity of the Haut study - the data plaintiffs

had pointed to as the promised new support for their hypothesis - and to determine if this new data advanced plaintiffs' hypothesis, or whether the "law exceeds the grasp of medicine" for the long-term, low-dose solvent exposure hypothesis.

27. In the present hearings, defendant produced neurologists Dr. Frey and then Dr. Albers (as a rebuttal witness). Plaintiffs produced Dr. Linz, an expert in occupational medicine, who had testified previously in the 1997 *Daubert-Gentry* hearing in *Allen*. Despite this Court's expressed interest in receiving testimony from a neurologist on the plaintiffs' side, plaintiffs have yet to produce a neurologist.

### III. THE APPROPRIATE METHODOLOGY

#### A. Plaintiffs Fail To Produce A Neurologist Or Reliable Neurological Evidence In Support of Their Hypothesis.

28. Although the hypothesis in question involves allegations of brain injury, plaintiffs have failed to produce a neurologist, or any competent neurological evidence, in support of their theory.

29. As plaintiffs expert explained, "Encephalopathy is a - a brain disorder." (Deposition of Dr. Douglas Linz dated October 29, 2008 ("Linz Dep.") at 19). Logically, the field dedicated to studying brain injuries - neurology - would be a relevant and important discipline from which to seek expert

opinions on the cause of purported brain injuries. As this Court stated, "what I'm looking for and was looking for [is] whether or not the plaintiffs could put on a neurologist so there could be legitimately a basis, a scientific basis, upon which this matter could go to the jury." (Tr. II at 99.) Yet, despite this Court's prompting, plaintiffs never offered testimony from a neurologist.

30. Instead, plaintiffs called an expert in occupational medicine. In the past, plaintiffs have called experts in occupational medicine, family practice and neuropsychology. This absence of neurology testimony in support of plaintiffs' theory is not necessarily indicative of a categorical divide between the fields of neurology and neuropsychology, but rather of plaintiffs' failure to meet their burden of proof. As discussed above, it is plaintiffs here who bear the burden of showing by a preponderance of the evidence that their proffered hypothesis is sufficiently relevant and reliable, and based upon the appropriate methodology, to be admissible. *Gentry*, 195 W. Va. at 522 n.8, 466 S.E.2d at 181, n.8. Their lack of neurological evidence to support what they allege is a brain injury syndrome begs the question of whether any reliable evidence for this syndrome exists.

**B. Evidence-Based Medicine Is The Methodology That Should Be Applied In Evaluating The Evidence That Is Before The Court**

31. Plaintiffs bear the burden of showing that their proffered evidence is based on reliable methodology. *State v. Henning*, 212 W. Va. 128, 133, 569 S.E.2d 204, 209 (2002). The appropriate methodology for reviewing scientific evidence to determine whether a cause and effect relationship exists between an exposure and a disease is "evidence-based medicine."

32. Evidence-based medicine is derived from the "Bradford Hill criteria." These criteria consider, among other things, the strength of a purported association, the reproducibility from study to study, the specificity of the association, the reproducibility from study to study, the specificity of the association, the temporal relationship of the association, the biological gradient (or dose response), and whether it can be measured experimentally. Hill, A.B. *The Environment and Disease: Association or Causation? President's Address*, Proceedings of the Royal Society of Medicine Vol. 58, 295-300 (1965); *Reference Manual on Scientific Evidence*, 2d ed., Federal Judicial Center, 2000, at 375-376. These highly regarded criteria are embraced by organizations such as the World Health Organization, the National Academy of Science, and the United States Environmental Protection Agency.

33. Plaintiffs' experts agree, as do defendant's, that evidence-based medicine is the appropriate methodology to evaluate a causation hypothesis, such as that currently before the Court. (Tr. II at 30); see also Linz Dep. at 54 (noting the Hill criteria require that "cause needs to come before the effect. It needs to be reproducible, it needs to be seen in . . . multiple different circumstances. It's not just a one time phenomenon. Needs to be predictable.").

34. The experts also agree that case control studies can generate a hypothesis, but not answer one. (Linz Dep. at 120; Tr. II at 29.)

#### IV. THE EVIDENCE PRESENTED

##### A. The Haut Study.

35. One of the reasons this Court called the present hearing was to determine whether the Haut study advanced plaintiffs' hypothesis, thus providing plaintiffs with some potentially reliable data (as opposed to the studies criticized by NIOSH and called into question by this Court).

36. However, the Haut study, as a result of its very design, as well as certain key shortcomings detailed below, fails to support plaintiffs' hypothesis.

37. The Haut study focused on one structure of the brain - the corpus callosum. The corpus callosum is *not* a part of the

brain involved with memory function. (Tr. I at 141; Linz Dep. at 115.)

38. The study was originally designed to use Positron Emission Tomography ("PET") technology to study results, but the actual study conducted used Magnetic Resonance Imaging ("MRI") results instead. Using MRI images of subjects' brains, Haut measured the size of the corpus callosum in two different groups of individuals by using a manual delineation, or "tracing" technique. (Tr. I at 78-79.)

39. The study subjects were divided into two groups, one group that reported exposure to organic solvents, and one group that did not. Exposure data was collected from questionnaires given to the study subjects, and was therefore based upon the subjects' own recollection and reports. (Tr. I at 61.) These test subjects likely included some former litigants. (Tr. II at 26-27; Linz Dep. 68, 93-94.)

40. The Haut study measured differences in corpus callosum size in one group of individuals compared to another. The study therefore did not - and could not - measure changes in individual brains. Plaintiffs' expert Dr. Linz agrees, noting "the fact that they concluded that the corpus callosum was smaller . . . in the exposed versus the unexposed group doesn't answer the question as to whether or not that was an acquired trait after adulthood." (Dr. Linz Oct. 6, 2008 Dep. at 151).

41. Haut reported finding a statistically significant difference in corpus callosum total area and anterior area between exposed and unexposed. (Tr. I at 83.) He hypothesized that solvent exposure was the cause of the reported difference. (Tr. I at 84.)

42. Yet, plaintiffs' and defendant's experts agree that the design of the Haut study was hypothesis generating, not hypothesis testing. (Linz Dep. at 120; Tr. II at 29.) In other words, the study was not designed in such a way as to be capable of proving cause and effect.

**B. Dr. Kirk Frey's Analysis Of The Haut Data.**

43. After this Court denied the West Virginia Attorney General's motion to quash CSXT's subpoena, the underlying data used by Haut in his study was provided to Dr. Kirk Frey, a neurologist with expertise in radiology, nuclear medicine and brain imaging (Tr. I at 60), in February 2007. Dr. Frey conducted his own critical evaluation of the Haut data - using state of the art methods - and concluded that the data (1) did not provide evidence of statistically significant brain tissue loss; and (2) did not support the hypothesis that chronic exposure to solvents causes changes in the brain.

44. Dr. Frey worked with Neuralyse, Inc. to conduct an independent analysis of the Haut data based upon reliable and reproducible methods. The laboratory Dr. Frey and Neuralyse

used was the Montreal Neurologic Institute lab of Dr. Alan Evans "international leaders in developing approaches and software tools for the analysis of brain shape, structure and volume, particularly as it pertains to magnetic resonance imaging data." (Tr. I at 88.)

45. The MRI scans used by Haut were coded so that those reviewing the scans in the laboratory could not identify a given scan as belonging to a reportedly exposed or unexposed individual. (Tr. I at 87.) The laboratory used a computer program to measure the size of the corpus callosum, eliminating the potential human error and variability involved with hand tracing, and also permitting three dimensional measurement of these three dimensional structures. "It's a completely automated technique so it does not require anyone to make a judgment of where the white matter [types of brain structure] is or attempt to trace it on a computer screen. It's done automatically." (Tr. II at 20.)

46. Dr. Frey and his team also took the data one step further, and measured the other white-matter brain structures in the scans, instead of just the corpus callosum. (Tr. I at 119.) Based on this analysis, Neuralyse, Inc. generated its own report on August 29, 2007. The method used by Dr. Evans and Neuralyse is peer reviewed and reproducible. (Tr. I at 96-97, 110.)

47. At the May 2008 hearing before this Court, Dr. Frey explained the results of the re-analysis of the Haut data. Dr. Frey and Neuralyse found only a 1.25% difference in corpus callosum size between the exposed and unexposed groups. (Tr. I at 118.) There was no statistically significant finding of difference. "[N]othing is statistically significantly different between the groups." (Tr. I at 116, 118.)

48. Further, Dr. Frey and Neuralyse found no evidence of brain tissue loss. Whereas Haut measured only the corpus callosum, Dr. Frey and his team also measured the other white-matter brain structures in the scans. (Tr. I at 119.) If the corpus callosum had in fact become smaller in size over time, the loss of brain structure would produce a measurable increase in fluid around the brain to compensate for this loss. (Tr. I at 85-86, 121.) "[I]f loss of brain structure, be it the corpus callosum or the frontal lobe or both, has occurred since maturation of the brain, then there should be secondary changes in the data to show that." (Tr. I at 85.) Yet, there was no evidence of such changes. In conclusion, Dr. Frey's reanalysis found:

- No support from the data that solvent exposure caused demyelination (break down) of white matter in the brain (Tr. I at 119);
- No evidence of frontal-lobe volume change (Tr. I at 123);

- No difference in frontal lobe volumes between exposed and unexposed groups (Tr. I at 124); and
- No difference between groups in cerebrospinal fluid volume (Tr. I at 124-25).

49. In sum, Dr. Frey did not find any support for either Dr. Haut's finding of a statistically significant difference in corpus callosum total area and anterior area between exposed and unexposed groups, or for Dr. Haut's stated conclusion that solvent exposure was the cause of the reported difference. (Tr. I at 83-84.)

**C. Dr. Frey's Unchallenged Critique Of The Haut Study.**

50. At the May 2008 hearing, Dr. Frey enumerated the Haut Study's limitations and design flaws and explained why Haut's study reached inaccurate results different from those reached in the re-analysis. Dr. Frey's critique of the Haut study went unchallenged by plaintiffs, and their expert, Dr. Linz, stated that he neither analyzed the Haut data, nor relied upon it to form his conclusions. (Linz Dep. at 78.)

51. To begin with, Haut designed his study around PET technology, but then applied the study to MRI technology. These technologies are not the same. (Tr. I at 44.)

52. In addition, both defendant's and plaintiffs' experts agree that cross-sectional studies - like the kind conducted by Haut - cannot document changes in the brain. (Tr. I at 85, 131-132.) Such studies can only measure difference, not change.

(Linz Dep. at 114.) "[T]o confidently interpret the difference [Haut] reports between the solvent-exposed and the control individuals as a change, there would have to either be evidence over time that a 'within subject difference' occurred. You'd have to MRI them all at the end of high school and again after employment for some period of time and document that, within an individual brain, a difference occurred. Well, that's not what was done." (Tr. I at 131.)

53. Next, Haut studied the corpus callosum, a portion of the brain with no function in memory, though plaintiffs' alleged symptoms involve memory loss. (Tr. I at 141; Linz Dep. at 115.) Also, in measuring the brain volume, Haut excluded certain portions when calculating the total. If reviewed by a neuroscience publication, this unexpected approach would have required additional explanation. (Tr. I at 115.)

54. The Haut study also failed to make use of the state of the art technologies available, instead using a manual tracing method prone to human error. (Tr. I at 127-128.) "[T]he only way of establishing the precision of the manual method would be to have the same individual repetitively measure individual brains to see how consistently they get an answer. And that is not done." (Tr. I at 129.) Haut's method also forced two dimensional tracing upon a three dimensional structure, an

inconsistency that is not present when using a computer that can match the dimensions. (Tr. I at 127-128.)

55. The study results are questionable due to the significant selection bias in the choice of study subjects. The study subjects all reported being symptomatic; a better comparison would have been drawn to a random selection of all workers allegedly exposed to solvents, regardless of symptoms. (Tr. I at 135.) Indeed, some of the subjects were likely former litigants (Tr. II at 26-27; Linz Dep. 68, 93-94), which further fuels the possibility of exaggerated reports of exposure.

56. The study was also unable to accurately measure the exact type and dose of exposure. (Tr. I at 73.) Rather, the alleged exposure levels were based upon the recollections of individuals claiming brain damage and possible memory loss. (Tr. I at 139; Linz Dep. at 112.)

57. Moreover, Dr. Haut changed his hypothesis *after* he had reviewed the results of the study. It is not valid scientific methodology to use data to both form a new hypothesis while simultaneously using it as a basis to prove that very hypothesis. (Tr. I at 34, 133.) "This is an approach ones takes in an exploratory fashion, collects some data, interrogate the data to see what it might be showing us. But any result you achieve in this way cannot be proven by those data because it's circular." (Tr. I at 133.)

**D. Dr. Frey's Further Testimony That The Solvent-Induced Toxic Encephalopathy Hypothesis Is Not Supported By Reliable Methodology.**

58. In addition to his unchallenged critique of the Haut study, Dr. Frey also provided testimony with respect to the scientific reliability of the plaintiffs' hypothesis generally. Dr. Frey testified that a diagnosis of solvent-induced toxic encephalopathy is not widely accepted in his field of neurology. (Tr. I at 59.)

59. More critically, toxic encephalopathy does not have common signs and symptoms providing sufficient predictive value to be a true syndrome. (Tr. I at 145.) "To be a syndrome, we have a description that is sufficiently detailed that any of us who are experts in the appropriate medical area should be able to identify in a given patient. So we have a reliable and common set of signs and symptoms that are agreed upon as sufficient to define who has and who does not have the condition, the syndrome." (Tr. I at 144.)

60. Plaintiffs have not demonstrated a reliable method to show that plaintiffs' reported symptoms are due to an actual neurotoxic effect, rather than just behavioral manifestations in those who believe they have an injury. (Tr. I at 144-45, 151.)

61. In sum, Dr. Frey testified that plaintiffs' theory is not supported by reasonable, reliable data. (Tr. I at 145-46.)

**E. Plaintiff's Expert Testimony In Support Of The Hypothesis.**

62. In support of the hypothesis at issue, plaintiffs proffered the testimony of Dr. Douglas Linz, an occupational medicine specialist. (Linz Dep. at 97.) Dr. Linz is neither a neurologist or a neuropsychologist in training. (Linz Dep. at 95, 97.) Dr. Linz appeared via video deposition testimony played to the court at the January 2009 hearing.

63. Dr. Linz's testimony, although intended to support plaintiffs' theory of general causation, demonstrated instead the utter lack of any clear or consistently applied methodology for assessing the causation hypothesis. Specifically, and as set forth in more detail below, Dr. Linz

- does not rely on the Haut study in support of his conclusions and fails to rebut Dr. Frey's critique of that study;
- admits that key aspects of his theories are unproven in the scientific literature;
- admits to relying on self-reports of potential litigants alleging brain injury and medical loss to assess past exposure to solvents;
- admits that there is no distinct pattern of symptoms associated with toxic encephalopathy and that the symptoms on which he bases his diagnoses are non-specific and could relate to a variety of other conditions;
- claims to rely on the World Health Organization ("WHO") criteria to diagnose toxic encephalopathy, and yet fails to consistently apply those criteria in specific cases; and

- diagnoses toxic encephalopathy even when neuropsychological testing (the only testing he relies on for purposes of diagnosis) shows no objective signs of abnormalities.

Dr. Linz's failure to properly apply a scientifically reliable methodology to diagnose toxic encephalopathy leaves the plaintiffs with no admissible testimony or evidence in support of their theory of general causation.

64. Although one of the intended purposes of the hearing was to ascertain whether the Haut study furthered plaintiffs' theory, Dr. Linz did not review any of the data upon which the Haut study was based, nor did he review any of Haut's deposition testimony, or speak to Dr. Haut. (Linz Dep. at 11, 82, 115.)

65. Dr. Linz admits that the Haut report does not effect "in any way" his belief that there is a causal link between solvent exposure and toxic encephalopathy, explaining that his opinion on the existence of solvent-induced toxic encephalopathy does not depend upon the Haut report. (Linz Dep. at 78).

66. Plaintiffs therefore offered no evidence to rebut Dr. Frey's criticism of the Haut study. Because there is no evidence demonstrating that the Haut study constitutes reasonably reliable evidence or an appropriate evidence-based methodology - indeed, the evidence demonstrates the opposite - it must be excluded as a matter of law. *Gentry*, 195 W. Va. at 521, 466 S.E.2d at 180; *see also Henning*, 212 W. Va. at 133, 569 S.E.2d at 209.

67. Instead of relying on the Haut study or providing new evidence, Dr. Linz relies on the same literature and methodology this Court has found to be "very, very weak." *Dummitt & Kibbey v. CSX*, No. 01-145, Hearing Transcript dated May 17 and 19, 2007 at 17. Dr. Linz testifies that his diagnostic methodology is supported by this scientific literature and that his belief that long-term, low-dose solvent exposure can cause toxic encephalopathy is generally accepted in the scientific and medical community. (Linz. Dep. at 55-64.) However, he admits that the criteria for determining a causal connection between a solvent exposure and alleged solvent-induced toxic encephalopathy has not significantly changed since 2004. (Linz. Dep. at 55.)

68. He further agrees that the current literature in support of CTE [toxic induced encephalopathy] does not prove any causation mechanism to a reasonable degree of scientific certainty. (Linz Dep. at 107.) Dr. Linz admits he can't describe the mechanism for how solvents purportedly cause brain damage "because we don't know the mechanism for certain." (Linz Dep. at 27.) Despite not being able to explain a causation mechanism, Dr. Linz concludes that solvent exposure can cause damage to brain at time of exposure, resulting in symptoms that occur later in life. (Linz. Dep. at 90-92.) Dr. Linz admits

90-92.) Dr. Linz admits that this "delayed onset" theory is an unproven hypothesis. (Linz Dep. at 129-30.)

69. In reaching his conclusions, Dr. Linz relies on exposure histories provided by workers themselves. (Linz Dep. at 34.) He has "never seen baseline testing for people." (Linz Dep. at 51.) Dr. Linz admits that it may be problematic to rely on the memories of patients with memory complaints. (Linz Dep. at 112-13; see also Linz Deposition dated October 6, 2008 at 44.) He also concedes that his patients likely include former litigants. (Linz Dep. at 68, 93-94.)

70. Despite acknowledging that his analysis depended upon the accuracy of self-reports made by potential litigants suffering from alleged brain injury and memory loss, Dr. Linz suggests that that the Court should disregard the self-reporting bias in the potential litigants' work histories because he is somehow able to screen for that bias.

Q. Are you saying that a worker could not fool you, for lack of a better word, by coming in to see you after talking with his co-workers who you may have seen, who has the same lawyer perhaps, as the co-workers you've seen, who may have sat - sat through a solvent trial, which is an experience that I've had, other plaintiffs come in to watch -"

A. Correct.

(Linz. Dep. at 173.) Dr. Linz's own confidence in his intuitive powers is not a basis for a reliable scientific methodology.

71. Dr. Linz's methodology involves taking medical and work histories, conducting a physical exam, neuropsychological tests and emotional behavioral assessment performed by neuropsychologist. (Linz Dep at 41-49.) However, there is no distinct pattern or group of symptoms associated with toxic encephalopathy. Dr. Linz agrees that is a fair statement that the "symptoms, themselves, are non-scientific, [and] could relate to a variety of potential conditions." (Linz Dep. at 101.)

72. Dr. Linz states that each patient's "neurological examination is usually entirely normal." (Linz Dep. at 145.) He therefore diagnoses patients as suffering from toxic encephalopathy even where no neurologic signs or physiological abnormalities exist, based upon his own neuropsychological evaluation. (Linz. Dep. at 145.) (As this court noted in *Dummitt & Kibbey*, based on the evidence presented in that case and in many prior solvents cases, "there's been no neurological correlation between the exposure and the condition." *Dummitt & Kibbey v. CSX*, No. 01-145, Hearing Transcript dated May 17 and 19, 2007 at 22.)

73. Dr. Linz states that the criteria he uses to make a diagnosis are derived from World Health Organization ("WHO") criteria (Linz. Dep. at 50, 100-01), although the WHO criteria are merely a description of outward symptoms, not a methodology

for evaluating a neurological disorder (Tr. II. at 37.) Based on the WHO criteria, Dr. Linz diagnoses different severity levels of chronic toxic encephalopathy starting with Type 1 as the least severe. (Linz. Dep. at 50-51.)

74. Dr. Linz states that a pattern of symptoms combined with occupational history is what permits him to make a diagnosis. Because there are no physiological signs or distinctive symptoms, he cannot predict whether someone is suffering from solvent exposure without first being told the person's occupational history. (Linz. Dep. at 147-49.) His diagnosis of solvent exposure as a cause of encephalopathy rests on a self-report of solvent exposure.

75. Dr. Linz admits that based on symptoms alone, there is no way to distinguish between different encephalopathies. (Linz Dep. at 147-49.) Dr. Linz concedes that the "brain can be adversely affected by a wide variety of different circumstances" (Linz Dep. at 47), and agrees that the symptoms he associates with toxic encephalopathy could relate to a variety of potential conditions. (Linz Dep. at 101.) Indeed, Dr. Linz is unwilling to fully address how one would rule out early onset Alzheimer's as a potential alternative explanation for some patients' reported symptoms. (Linz Dep. at 146-47.)

76. Despite being unable to rule out alternative causes, Dr. Linz maintains that his neuropsychological testing method

can diagnose solvent-induced toxic encephalopathy. Yet, Dr. Linz admits that he has diagnosed chronic toxic encephalopathy in patients who do not demonstrate neuropsychological deficits. (Linz Dep. at 151, 154; Tr. II at 42.) In fact, his definition of "Type 1" toxic encephalopathy is that of a person who gives self-reports of solvent exposure, self-reports of symptoms such as "memory problems, attentional [sic] deficits, you know, depression" and who have neuropsychological test scores "within a general range of what we would anticipate . . . from the general population." (Linz. Dep. at 51.) He admits that he makes "Type 1" diagnoses even though "in terms of objective abnormalities to support the reporting of symptoms and so forth, there are none." (Linz. Dep. at 51; emphasis added.) See also Linz Dep. at 154 ("I've diagnosed [a plaintiff] as having Type 1 chronic toxic encephalopathy which, by definition, does not have objective evidence.") Moreover, although the WHO criteria for Type 1 toxic encephalopathy state that symptoms should fully reverse themselves within 6 months after exposure ends (Tr. II at 46), Dr. Linz diagnoses patients with Type 1 encephalopathy even when their symptoms did not begin until years after exposure ended and/or whose symptoms have worsened over time. (Linz Dep. at 136-37.)

77. The WHO criteria for "Type 2" encephalopathy requires an objective finding of abnormalities, either a sustained

personality change or impairment of intellectual function, such as memory and concentration loss. (Linz Dep. at 49; see also Opinions of James W. Albers *Solvent-Induced Toxic Encephalopathy* (Jan. 9, 2009) at slides 4, 72). Yet, Dr. Linz has diagnosed Type 2 encephalopathy in one individual who had only one abnormal test result, surrounded by a balance of otherwise normal scores, and despite "superior" scores on memory tests. (Tr. II at 42; Linz Dep. at 159-61.)

78. Thus, Dr. Linz does not apply his own chosen methodology - that of neuropsychological questioning instead of physical neurological test results - in a consistent manner.

79. Dr. Linz's own testimony reveals that the very methodology he relies upon to support plaintiffs' causation hypothesis cannot rule out alternative causes, or even establish exposure as a cause without already being told that it has occurred. Accordingly, Dr. Linz's methodology fails the basic requirements of *Gentry/Daubert* because his theory and conclusion cannot be tested in a reproducible way, or with a known rate of error. See *Jones*, 221 W. Va. At 385, 655 S.E.2d at 131. His method is not falsifiable. See *Daubert*, 509 U.S. at 593 (good science must be capable of proving a hypothesis false); see also *Dummitt & Kibbey v. CSX*, No. 01-145, Hearing Transcript dated May 17 and 19, 2007 at 17 ("it becomes a self-fulfilling prophecy. A person works for the railroads, exposed to

solvents, and they complaint of something that may touch upon some brain sickness, and it's therefore solvent-induced encephalopathy. That's not true science.")

80. Moreover, Dr. Linz does not apply his own methodology in a consistent way. He will, in effect, make a diagnosis absent any supporting methodology. (Linz. Dep. at 145.) His definition of a "Type 1" diagnosis is one in which abnormal neuropsychological test findings are absent - although the test he says he employs requires otherwise. (Linz. Dep. at 51.) For an expert's opinion to be admissible, "the basic methodology employed" must be not only scientifically valid, but also "properly applied." *Jones*, 221 W. Va. at 384, 655 S.E.2d. at 131 (emphasis added). This failure to apply any methodology, save total reliance on patient self-reports, further renders his opinion inadmissible.

**F. Dr. James Albers' Rebuttal Testimony.**

81. Following the presentation of Dr. Linz's video deposition at the January 2009 hearing, defendant offered rebuttal testimony from Dr. James Albers, a neurologist. Dr. Albers testified briefly with regard to the Haut study, and then offered his evaluation of Dr. Linz's opinions and of the evidence relating to the long-term, low-dose solvent exposure theory at issue.

82. Because one of the original purposes of this hearing was to evaluate the validity of the Haut study, Dr. Albers offered his critique of the study, despite the fact that Dr. Linz did not rely on the Haut study findings in his analysis. (Linz Dep. at 11, 82, 115.) Dr. Albers reviewed the Haut paper, and Dr. Frey's reanalysis of the Haut data, although Dr. Albers did not conduct his own analysis of the underlying Haut data. (Tr. II at 15.)

83. Based on his review, Dr. Albers concluded that the Haut report was subject to a number of flaws, including (1) the suggestion that it could measure "change" in brain volume when its design only permitted measurement of differences between two groups (Tr. II at 16); (2) the use of a "substantially old" and "primitive" tracing technique instead of the "up-to-date, current technique" employed by Dr. Frey's team (Tr. II at 19, 23); and (3) the reliance upon individual subjects' memories for exposure data (Tr. II at 25), amongst other issues. Dr. Albers concluded that the Haut study provided no evidence of brain damage causally related to chronic occupational exposure to solvents. (Tr. II. at 22.) Indeed, the MRI results were all "entirely normal." (Tr. II at 16.)

84. Dr. Albers next testified regarding Dr. Linz's conclusions and methodology. Dr. Albers expressed concern over Dr. Linz's reliance on the memories of subjects as the basis for

his exposure data when the subjects themselves complained of memory problems. (Tr. II at 39.) As Dr. Albers' explained, Dr. Linz's method relies upon having "a history that is provided to him that is believable." (Tr. II at 36.) The reliance on subjects' self-reporting is additionally complicated because of potential litigation bias, which "can be a major factor, particularly when you're talking about something that's not quantifiable and we have no direct measurement of it." (Tr. II at 26-27.)

85. One reason Dr. Linz must base his opinions on individuals' reports and recollection is because there are no biological markers or physiological signs of long-term occupational solvent exposure. Dr. Albers asserts that when applying Dr. Linz's methodology, there is "[n]othing you can test other than the neuropsychological testing." (Tr. II at 38.)

86. Dr. Albers also noted that Dr. Linz's methodology did not rule out other possible causes. Yet, other possible causes exist. Indeed, Dr. Albers testified that complaints about memory problems become prominent as people age, and that a recent Mayo Clinic study suggests that "symptoms that we would associate with early dementia - namely memory complaints - appear to be rising. In their study, it seemed to be more prominent arising more rapidly in men. And that this mild

mental decline with age is much more common than previously thought." (Tr. II at 54.)

87. Dr. Albers further testified that other explanations may exist for the reported symptoms Dr. Linz describes, and without an objective way to rule out alternative causes, his evidence does not support any one causation hypothesis over another. "It is a basic tenet of verifying a diagnosis; it must escape someone's ability to suggest an alternative. If by definition you can't falsify it, it makes it very difficult to deal with this as a real entity." (Tr. II at 35.)

88. Dr. Albers also critiqued Dr. Linz's assertion that various literature supports his opinion and methodology. "Science is not consensus. [It] doesn't matter how many people like me give an opinion; science is hypotheses that can be tested. . . . Just the fact that these items are in textbooks doesn't necessarily mean that they're correct. And when we are discussing causation, I think the evidence based upon good science is what we should be looking at." (Tr. II. at 32-33.) Upon Dr. Albers' own review of the literature, he concluded that there was "absolutely not" evidence-based support for the "progressive syndrome" hypothesis that Dr. Linz subscribes to. (Tr. II at 58-60, 66-67, 71.)

89. Even if Dr. Linz's methodology were reliable - and it is not, as established above - Dr. Linz does not even apply his

own methods as described. According to not just Dr. Albers, but the basic principles of scientific reliability as applied by the courts, a method must be "properly applied" in addition to being "scientifically or technically valid." *Jones*, 221 W. Va. at 384, 655 S.E.2d. at 131. Yet, when Dr. Linz makes diagnoses of solvent-induced toxic encephalopathy in subjects who show no abnormalities on the very tests Dr. Linz relies upon, his work becomes not only incapable of falsification, but departs from any clear methodology altogether. Dr. Linz "certainly does not practice what he was preaching to us." (Tr. II at 43.)

90. Due to these serious flaws, Dr. Albers concluded that "the methodology that [Dr. Linz] uses . . . is really contrary to the basic tenets of evidence-based medicine." (Tr. II at 30.) Dr. Albers further concluded that there is insufficient scientific evidence to support the hypothesis that long-term chronic occupational exposure to solvents causes permanent brain damage. (Tr. II at 71-72.)

#### CONCLUSION

91. Plaintiffs have failed to produce relevant and reliable evidence to support their theory of injury as required for admissibility pursuant to Rules 104 and 702 of West Va. Rules of Evidence. *See, e.g., Lockhart*, 208 W. Va. at 629, 542 S.E.2d at 450. The Haut study, the "new" data promised by

plaintiffs, does not provide any reliable evidence to further plaintiffs' hypothesis. Plaintiffs' testifying expert did not even rely on the study to reach his conclusions. Plaintiffs also did not provide any competent neurological evidence to support their alleged neurological condition.

92. Instead, plaintiffs rely on an occupational medicine expert whose opines that long-term, low-dose occupational exposure to certain organic solvents can cause brain damage. This opinion is based upon literature he admits does not prove a causation mechanism to a reasonable degree of scientific certainty, and upon the reported recollections of former workers (including litigants) who claim to suffer from memory problems. It is not based on any neurological signs of disease. It is not based on any unique or commonly occurring symptoms. It is not based upon a method that permits other potential causes to be ruled out. Indeed, Dr. Linz's "diagnoses" solvent-induced encephalopathy when he is told that solvent exposure occurred, even in the face of contrary neuropsychological evidence. This amounts to no methodology at all. Rather, this is a self-fulfilling proposition is more akin to "reading tea leaves" than to a reasonably reliable methodology. *In re Flood Litig.*, 222 W. Va. 574, 668 S.E.2d 203, 211 (2008). Or, as this Court said in *Dummitt & Kibbey*, "the more you look at it, it [is] just - it

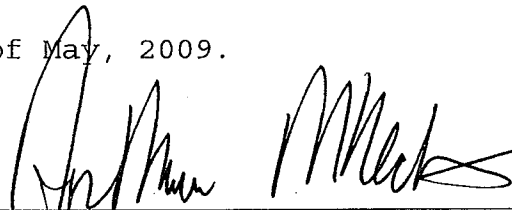
is gossamer. It's illusive." *Dummitt & Kibbey v. CSX*, No. 01-145, Hearing Transcript dated May 17 and 19, 2007 at 18.

93. With respect to the hypothesis that long-term, low-dose, occupational solvent exposure can result in permanent brain damage, the "reach of the law" has truly exceeded the "grasp of the medicine." *Allen*, Feb. 2, 1998, Order at p. 3. Plaintiffs' experts should not be permitted to testify to a hypothesis, the foundation of which rests on unreliable evidence and an unreliable, and inconsistently applied, methodology.

The Defendant's *Motion in Limine* to exclude the scientific testimony of the Plaintiffs is hereby **GRANTED**, with the objection of the Plaintiffs saved to the Court's ruling.

It is so **ORDERED**.

Enter this 19<sup>th</sup> day of May, 2009.



ARTHUR M RECHT, CHIEF JUDGE

A copy of this Memorandum Of Opinion, Findings of Fact and Conclusions of Law has been sent to all counsel as follows:

JOHN W MCTIERNAN ESQUIRE  
CAROSELLI BEACHLER MCTIERNAN & CONBOY  
20 STANWIX STREET 7<sup>TH</sup> FLOOR  
PITTSBURGH PA 14222

MARK T WADE ESQUIRE  
THE WADE LAW OFFICE  
901 WESTERN AVENUE STE 206  
PITTSBURGH PA 15233

JAMES W TURNER ESQUIRE  
HUDDLESTON BOLEN  
P O BOX 2185  
HUNTINGTON WV 25772-2185

IN THE CIRCUIT COURT OF BROOKE COUNTY, WEST VIRGINIA

STEVEN ALLEN, ROBERT CALLIHAN, :  
MICHAEL COBB, PAUL DENNISON, :  
VIRGIL GEARY, VERTIS HADLEY, :  
RONALD HOWARD, RONALD HUMM, :  
BILLIE JOE LEMLEY, RONALD :  
MARCUM, JOHN MCCLUNG, JAMES :  
MCCOY, JOEL MERRITT, JANICE :  
MILLER, RUSSELL MOORE, LARRY :  
MORRIS, WILLIAM RALLS, ROBERT :  
SHEPHERD, CECIL SULLIVAN, :  
PHILIP WILEY, AND CLARENCE :  
WILLIAMS, :

Plaintiffs, :

v. :

CSX TRANSPORTATION, INC., :

Defendant. :

CIVIL ACTION NO. 96-C-35  
1 through 21.

OPINION AND ORDER

This matter involves twenty-one (21) cases instituted against CSX Transportation, Inc. (herein CSX), under The Federal Employer Liability Act, 45 USC §51 (1939)<sup>1</sup> alleging that each plaintiff has suffered permanent, irreversible damage to the brain and/or nervous system due to long-term exposure to low doses of certain solvents.

CSX disputes that there exists reliable and relevant evidence that low dose exposure to certain solvents over long periods of

---

<sup>1</sup>State Courts have concurrent jurisdiction along with Federal Courts of claims brought under The Federal Employers Liability Act 45 USC §51 (1939). Syllabus Pt. 4, McGraw, et al. v. Norfolk & Western Railway Company, et al., (Case No. 23996, December 15, 1997).

EXHIBIT # 1

time can cause permanent, irreversible damage to the brain and/or nervous system.

Because of the critical nature of the validity of the science of the case, this Court considered it necessary to conduct an *in limine* hearing within the boundaries shaped by the West Virginia Supreme Court of Appeals in Gentry v. Mangum, 195 W.Va. 512, 466 S.E.2d 171 (1995)<sup>2</sup>.

Consequently, this Court conducted a hearing over a period of two days to receive testimony concentrating upon a two-part analysis in regard to the twin issues of scientific reliability and relevance by:

(1) First, determining whether the proffered expert testimony reflects scientific knowledge, that is, whether the findings are derived by scientific method, and whether the work product amounts to good science; and

(2) Second, whether the scientific testimony is relevant to the task at hand. See Syllabus Pt. 2, Gentry v. Mangum, *Supra*.

Throughout the entire period of time that this Court received the number of prominent experts within the areas of occupational medicine, epidemiology, neurology, neuropsychology, and toxicology,

---

<sup>2</sup>Procedural issues in a matter instituted pursuant to The Federal Employer Liability Act are determined by State procedural rules and substantive issues are determined by the provisions of the statute and interpretive decisions of The Federal Employer Liability Act given by the Federal Courts. See Syllabus Pt. 2, McGraw v. Norfolk & Western Railway Co., *Supra*.

two recurring themes emerged. First, that quite possibly in this case the reach of the law may exceed the grasp of the medicine, and second, that there is reliable and relevant evidence that to a "reasonable probability" there is a general causal relationship between a given physical condition (encephalopathy) and the exposure to certain solvents. See Syllabus Pt. 3, Hovermale v. Berkeley Springs Moose Lodge No. 1483, 165 W.Va. 689, 271 S.E.2d 335 (1980).

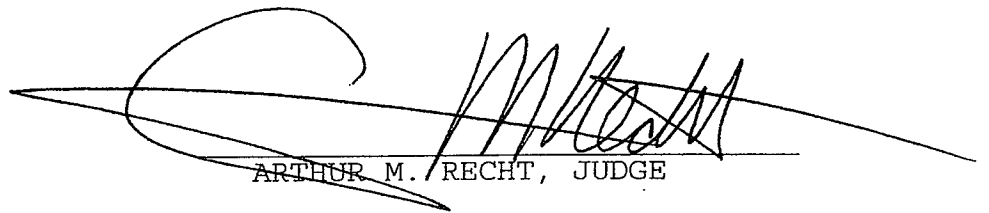
This Court is satisfied after a review of the entire record of the hearings conducted on November 13 and 14, 1997, that the science within the proffered evidence is valid enough to be reliable and relevant. This Court in its gate-keeping function is not to decide whether the proffered evidence is right, but only that it is reliable. While certainly there is a fierce debate among competing professionals, there is sufficient scientific knowledge that will assist the triers of fact to support the hypothesis that low doses with long term exposure to certain solvents can cause encephalopathy. Whether any of the plaintiffs in this case, on a case specific analysis, can sufficiently carry their burden of proof remains to be determined. The science that was presented may not be right but it is reliable.

Accordingly, the motion *in limine* to exclude the scientific testimony of the plaintiffs is denied with the exception of the defendants saved to the Court's ruling.

It is so **ORDERED**.

Copies of this Opinion and Order shall be mailed to the plaintiffs in care of Mark T. Coulter, Esquire, Robert Peirce & Associates, 2500 Gulf Tower, 707 Grant Street, Pittsburgh PA 15219 and Carl N. Frankovitch, Esquire, Frankovitch, Anetakis & Colantonio, 337 Penco Road, Weirton, WV 26062 and upon the defendants in care of Stephen M. Houghton, Esquire and Richard D. Klaber, Esquire, Dickie, McCamey & Chilcote, Two PPG Place Suite 400, Pittsburgh PA 15222-5402.

Entered this 2<sup>nd</sup> day of February 1998.



ARTHUR M. RECHT, JUDGE