IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF IOWA
CENTRAL DIVISION

RENE JUNK, as Parent and Next Best Friend of T.J., a minor, Plaintiff, vs. TERMINIX INTERNATIONAL COMPANY LIMITED PARTNERSHIP; THE DOW CHEMICAL COMPANY; DOW AGROSCIENCES, LLC; and JIM BRENEMAN, an individual, Defendants.	No. 4:05-cv-0608-JAJ **ORDER**

This matter comes before the Court pursuant to Terminix International Company Limited Partnership's ("Terminix") May 12, 2008, Motion to Strike Plaintiff's Expert Witnesses and The Dow Chemical Company's and Dow AgroSciences LLC's (collectively "Dow AgroSciences") May 12, 2008, Motion in Limine to Exclude Expert Causation Testimony of Cynthia M. Bearer, M.D., Ph.D., and Mohamed Abou-Donia, Ph.D. and for Summary Judgment. [Dkt. Nos. 140, 143]. Plaintiff Rene Junk, as Parent and Next Best Friend of T.J., a minor ("Junk"), disclosed Dr.Bearer as an expert witness. For the reasons set out below, the portions of Terminix's and Dow AgroSciences' motions relating to Dr. Bearer are denied.

I. FACTUAL HISTORY[1]

In February of 1992, Plaintiff Rene Junk became pregnant with her first child. During Rene Junk's pregnancy, Terminix applied the pesticide Dursban L.O. ("Dursban") to cracks and crevices in the interior of the Junk home to treat spider infestation. Dursban,

[1] The Factual Background is based on the facts as alleged in the June 12, 2007, report of Dr. Mohamed Abou-Donia, Ph.D.

a trademark of Dow AgroSciences, contains the organophosphate chlorpyrifos. Following Tyler Junk's birth on August 28, 1992, Terminix continued to regularly apply Dursban to the Junk home. The last application of Dursban to the Junk home occurred on September 15, 1994.

On June 25, 1992, doctors discovered a chorioangioma, or a large tumor, in Tyler Junk's umbilical cord. Throughout her pregnancy, Rene Junk suffered a number of symptoms, including nausea, vomiting, diarrhea, and skin rash. On August 25, 1992, three days prior to Tyler Junk's birth, doctors discovered that he suffered from an enlarged heart and tachycardia, or a rapid heart rate. On August 28, 1992, Rene Junk gave birth to Tyler Junk. Tyler Junk suffered from tachycardia and had an enlarged heart and liver at the time of birth. The birth was approximately two months premature. Following the birth of her son, Rene Junk was diagnosed with pulmonary edema.

Throughout the first months of his life, Tyler Junk suffered from fussiness, loss of appetite, difficulty with breathing, and a runny nose. From the time that Tyler Junk was approximately six months old, Rene Junk observed that he appeared to exhibit symptoms of developmental delay. Tyler Junk was later diagnosed with cerebral palsy, and currently suffers from significant developmental delay.

On October 3, 2005, Rene Junk, on behalf of her minor son, filed a lawsuit against Dow AgroSciences, Terminix, and other defendants in Iowa state court. [Dkt. 1]. Junk alleged that her son suffered physical, neurological, and psychological injuries as a result of exposure to chlorpyrifos, which is contained in Dursban. On November 4, 2005, Defendants filed a notice removing the lawsuit to the United States District Court for the Southern District of Iowa. [Dkt. 1].

II. PROCEDURAL HISTORY

On August 10, 2006, this Court issued the initial scheduling order and discovery plan in this matter. [Dkt. 56]. After granting an extension, the Court set Junk's initial expert disclosure deadline on June 18, 2007. [Dkt. 80]. Junk disclosed several expert witnesses, including Dr. Bearer. Dr. Bearer is a neonatologist who received her Ph.D. in biochemistry from Case Western Reserve University in 1977 and her M.D. from The Johns Hopkins University in 1982. [Junk App., p. 1]. She is the attending neonatologist and director of the neonatology training program at Rainbow Babies & Children's Hospital in Cleveland, Ohio. [Junk App., p. 1]. Dr. Bearer is an associate professor in pediatrics, neurosciences, and environmental health sciences at Case Western Reserve University. [Junk App., p. 1]. She is board-certified pediatrics and neonatal/perinatal medicine. [Junk App., p. 6]. From 1998 to 2001, Dr. Bearer served on the Scientific Advisory Board, Environmental Health Subcommittee of the Environmental Protection Agency ("EPA"). [Junk App., p. 7]. Dr. Bearer was selected by the EPA to provide an independent review of a 2007 EPA summary report regarding children's environmental health research. [Junk App., p. 625].

Dr. Bearer submitted a preliminary report dated June 15, 2007, and a supplemental report dated July 6, 2007, regarding the medical causation of Tyler Junk's condition. [Junk App., pp. 34-41]. Dr. Bearer was deposed by Dow AgroSciences and Terminix on January 23, 2008. On May 12, 2008, Terminix and Dow AgroSciences filed motions to exclude the expert causation testimony of Dr. Bearer on the grounds that it did not meet the requirements of Federal Rule of Evidence 702. [Dkt. Nos. 140, 143]. Junk timely responded to these motions. [Dkt. Nos. 161, 164]. Dr. Bearer executed a declaration dated May 28, 2008, that was included in Junk's appendix in support of Junk's response to Dow AgroSciences' and Terminix's motions to exclude her expert causation testimony. [Junk App. pp. 958-75]. On July 2, 2008, Dow AgroSciences timely replied. [Dkt. 201].

On July 7, 2008, this Court held a motion hearing in which the parties addressed Dow AgroSciences' and Terminix's motions regarding the exclusion of Dr. Bearer's causation testimony. [Dkt. 203]. Counsel for Junk, Terminix, and Dow AgroSciences were present at the hearing. At the hearing, the Court reserved ruling on the motions.

III. STANDARD OF ADMISSIBILITY

Federal Rule of Evidence 702, along with the tenets of Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579 (1993), govern the admissibility of expert testimony in federal court. Rule 702 states:

> If scientific, technical, or otherwise 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.

FED. R. EVID. 702. "District courts must ensure that all scientific testimony is both reliable and relevant." Marmo v. Tyson Fresh Meats, Inc., 457 F.3d. 748, 757 (8th Cir. 2006) (citing Daubert, 509 U.S. at 580; Fed. R. Evid. 702). "To satisfy the reliability requirement, the proponent of the expert testimony must show by a preponderance of the evidence both that the expert is qualified to render the opinion and that the methodology underlying his conclusions is scientifically valid." Id. at 757-58 (citing Daubert, 509 U.S. at 589-90). The Daubert factors for reliability are

> (1) whether the expert's methodology has been tested;
> (2) whether the technique has been subjected to peer review and publication;
> (3) whether the technique has a known or knowable rate of error; and

> (4) whether the technique has been generally accepted in the proper scientific community.

Bonner v. ISP Technologies, Inc., 259 F.3d 924, 929 n.3 (8th Cir. 2001) (quotation omitted). To satisfy the relevancy requirement, expert testimony must "provide[] information beyond the common knowledge of the trier of fact." Kudabeck v. Kroger Co., 338 F.3d 856, 860 (8th Cir. 2003) (citation omitted).

In determining whether expert testimony should be admitted, the requirements of Daubert and its progeny direct district courts to concentrate on the second tenet listed in Rule 702 - the principles and methods utilized by the expert. "The focus, of course, must solely be on principles and methodology, not on conclusions they generate." Bonner, 259 F.3d at 929 (quoting Daubert, 509 U.S. at 594-95). "An expert opinion must be supported appropriate validation – i.e., good grounds based on what is known." Glastetter v. Novartis Pharmaceuticals Corp., 252 F.3d 986, 988-89 (8th Cir. 2001) (quoting Daubert, 509 U.S. at 590).

In assessing the reliability and relevancy of proffered expert evidence, the Court "should be conscious of two guiding, and sometimes competing, principles." Westberry v. Gislaved Gummi AB, 178 F.3d 257, 261 (4th Cir. 1999).

> On the one hand, the court should be mindful that Rule 702 was intended to liberalize the introduction of relevant expert evidence. And the Court need not determine that the expert testimony a litigant seeks to offer into evidence is irrefutable or certainly correct. As with all other admissible evidence, expert testimony is subject to being tested by "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof." On the other hand, the court must recognize that due to the difficulty of evaluating their testimony, expert witnesses have the potential to "be both powerful and quite misleading." And, given the potential persuasiveness of expert testimony, proffered evidence that has a greater potential to mislead than to enlighten should be excluded.

Id. (internal quotation and citations omitted).

The Eighth Circuit has stated that a properly-conducted differential diagnosis is presumptively admissible under Daubert. Bland v.Verizon Wireless L.L.C., No. 07-3010, 2008 WL 3474178, at *4 (8th Cir. August 14, 2008) (citation omitted). See also Glastetter, 252 F.3d at 989 (citing Turner v. Iowa Fire Equipment, Co., 229 F.3d 1202, 1208 (8th Cir. 2000)) ("Because a differential diagnosis is presumptively admissible a district court may exercise its gatekeeping function to exclude only those diagnoses that are scientifically invalid."). "A 'differential diagnosis [is] a technique that identifies the cause of a medical condition by eliminating the likely causes until the most probable cause is isolated.'" Bland, 20008 WL 3474178, at *4 (quoting Turner, 229 F.3d at 1208). "In performing a differential diagnosis, a physician begins by 'ruling in' all scientifically plausible causes of the plaintiff's injury. The physician then 'rules out' the least plausible causes of injury until the most likely cause remains." Kudabeck, 338 F.3d at 860-61 (quoting Glastetter, 252 F.3d at 989). "The final result of a differential diagnosis is the expert's conclusion that a defendant's product caused (or did not cause) the plaintiff's injury." Glastetter, 252 F.3d at 989 (citation omitted).

IV. ANALYSIS

To prove causation in a toxic tort case, Junk must show both that "the alleged toxin is capable of causing injuries like that suffered by the plaintiff in human beings subjected to the same level of exposure as the plaintiff, and that the toxin was the cause of the plaintiff's injury." Bonner, 259 F.3d at 928 (citing Wright v. Willamette Indus., Inc., 91 F.3d 1105, 1106 (8th Cir. 1996) ("We agree . . . that a plaintiff in a toxic tort case must prove the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover.")). "In other words, the plaintiff must put forth sufficient evidence for a

jury to conclude that the product was capable of causing her injuries, and that it did." Bonner, 259 F.3d at 928. This is known as general causation and specific causation. See also, Knight v. Kirby Inland Marine, Inc., 482 F.3d 347, 351 (5th Cir. 2007) ("General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury.") (quotation omitted).

The Eighth Circuit of Appeals has stated the requisite level of proof a plaintiff must produce regarding exposure and dosage in a toxic tort case. The Court of Appeals stated:

> To prove exposure levels, plaintiffs need not produce a mathematically precise table equating levels of exposure with levels of harm. Rather, a plaintiff need only make a threshold showing that he or she was exposed to toxic levels known to cause the type of injuries he or she suffered.

Mattis v. Carlon Elec. Prods., 295 F.3d 856, 860-61 (8th Cir. 2002) (quotation and citation omitted). The Court of Appeals also stated that "[i]t is sufficient for a plaintiff to prove that she was exposed to a quantity of the toxin that 'exceeded safe levels.'" Bonner, 259 F.3d at 931 (citation omitted). See also, Bednar v. Bassett Furniture Mfg. Co., Inc., 147 F.3d 737, 740 (8th Cir. 1998) ("The Bednars did not need to produce a 'mathematically precise table equating levels of exposure with levels of harm' in order to show Marian's level of exposure to gaseous formaldehyde, but only 'evidence from which a reasonable person could conclude that [the] defendant's emission has probably caused' the harm about which they complain.") (citation omitted).

A. General Causation

In this general causation analysis, "[T]he fundamental question underlying [Dr. Bearer's] testimony . . . is whether the chemicals [Rene and Tyler Junk] were exposed to and the type of exposures they experienced cause [severe neurodevelopmental delay]." Knight, 482 F.3d at 351. In opposing the admission of Dr. Bearer's general causation opinion, Dow AgroSciences focuses on the term "cerebral palsy," contending that Junk

presents no evidence linking chlorpyrifos exposure and "cerebral palsy." However, the Court finds that Dow AgroSciences limiting of the scientific inquiry to a connection between chlorpyrifos exposure and "cerebral palsy" to be too narrow. Tyler Junk suffers from severe neurodevelopmental delay with neurologic deficits. Thus, the proper inquiry in the general causation analysis is whether in utero and postnatal exposure to chlorpyrifos is capable of causing severe developmental delay.

Dr. Bearer is well-qualified to provide an opinion regarding the capability of in utero and postnatal chlorpyrifos exposure to cause neurological harm. Dr. Bearer has over twenty years of experience as a neonatologist and is board-certified in pediatrics and neonatal/perinatal medicine. [Junk App., pp. 1, 6]. For three years, she served on the Scientific Advisory Board, Environmental Health Subcommittee of the EPA. [Junk App., p. 7]. Dr. Bearer has served on numerous national advisory groups addressing the effect of environmental factors on children's health. [Junk App., p. 7-8]. Furthermore, Dr. Bearer was selected by the EPA to provide an independent review of a 2007 EPA summary report regarding children's environmental health research. [Junk App., p. 625].

Dr. Bearer's opinion that in utero and postnatal exposure to chlorpyrifos can cause neurological harm is scientifically reliable. In addition to her experience, Dr. Bearer based her general causation opinion on a number of peer-reviewed articles and studies documenting the effects of in utero and prenatal chlorpyrifos exposure on animals and humans. [Dr. Bearer's Decl. ¶¶ 12(h)-(k), 12(p), 12(v), 24(hh) (animal studies); ¶ 12(l)-(o), 12(q)-(t), 12(w)-(y)(human studies)]. One of the most recent scientific articles cited by Junk provides a helpful summary of the research results to date regarding the effects of chlorpyrifos exposure.

> Although, [chlorpyrifos] has been shown to be relatively safe in adult animals, newly discovered evidence indicates that chlorpyrifos is a developmental neurotoxicant in the fetus and is thus harmful (Garcia, et al., 2003). In animals and cellular models, chlorpyrifos inhibits neural cellular replication (Qiao

> et al., 2001), interferes with cellular differentiation (Crumpton et al., 2000), evokes oxidative stress, alters neurotransmission (Dam et al., 1999; Bloomquist et al., 2002; Karanth et al., 2006; Slotkin and Seidler, 2007) and induces neurobehavorial changes (Ricceri et al., 2006). Additionally, animals exposed to [chlorpyrifos] in utero or as juveniles display motor and cognitive delays (Moser, 2000). In humans, elevated levels of chlorpyrifos in umbilical cord plasma are inversely associated with birth weight and length in children born to minority women (Whyatt et al., 2004). The literature indicates that chronic [chlorpyrifos] exposure is associated with decreased birth weight and length. In addition, lower birth weights have been specifically been documented among African Americans infants (Rauh et al., 2006; Perera et al., 2003, 2005) exposed to chlorpyrifos *in utero*. Finally, [chlorpyrifos] exposure is associated with alterations in developmental and psychomotor indices in Mexican-American children (Eskenazi et al., 2007) and with immunological abnormalities (Thrasher et al., 2002).

Marilyn D. Saulsbury et al., *Characterization of chlorpyrifos-induced apoptosis in placental cells,* Toxicology, 2008 Feb. 28; 244(2-3): 98-110. Dr. Bearer specifically cites seven articles to support her opinion that "prenatal/postnatal exposure to chlorpyrifos is associated with neurodevelopmental delay in both animals and humans." [Dr. Bearer Prelim. Rep., p.1; Dr. Bearer Supp. Rep., p. 2]. Each of these articles demonstrates the mechanisms by which in utero and/or postnatal chlorpyrifos exposure cause neurological harm in either animals or humans.

Dow AgroSciences contends that this evidence does not support Dr. Bearer's opinion on general causation because it does not "fit" and is not relevant.[2] At its root,

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[2] Dow AgroSciences contends that, "What Plaintiff has done in her opposition brief is drop mention of dozens of studies unrelated to T.J.'s specific injuries (and in many cases, directly contrary to any symptoms T.J. has), hoping that the equivalent of a document dump will so overwhelm the Court that it will get lost in the confusion. The Court must not lose sight of the principles of 'fit' and relevancy. DAS has never maintained that chlorpyrifos cannot cause harm. Of course it can; all substances can cause harm at certain levels . .

(continued…)

Dow AgroSciences' argument is that no study demonstrates in utero and postnatal chlorpyrifos exposure can cause the exact type of neurodevelopmental delay experienced by Tyler Junk. However, Junk's inability to point to one study that unequivocally demonstrates chlorpyrifos exposure can cause that exact type of neurodevelopmental delay does not preclude the admission of Dr. Bearer's general causation opinion. "[T]here is no requirement 'that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.'" Kudabeck, 338 F.3d at 862 (quoting Heller v. Shaw Indus., Inc., 167 F.3d 146, 155 (3rd Cir. 1999)). The scientific evidence that Junk provides, including peer-reviewed animal and human studies, demonstrates the mechanisms by which in utero and postnatal chlorpyrifos exposure can cause neurologic harm. Based on her experience, the peer-reviewed animal and humans studies, and the published literature, Dr. Bearer can "reliably conclude" that in utero and postnatal chlorpyrifos exposure is capable of causing severe developmental delay. Thus, the Court finds that her opinion on general causation is admissible.

B. Specific Causation

To meet her burden regarding specific causation, Junk offers Dr. Bearer's opinion that "Tyler Junk's neurodevelopmental delay is the result of his exposure to Dursban both in utero and in the early years of his life." [Dr. Bearer Decl., ¶ 27]. Dr. Bearer conducted a differential diagnosis to reach this conclusion. [Dr. Bearer Decl. ¶ 10]. Dr. Bearer based her opinion on (1) the medical records, (2) the medical and scientific literature, (3) other materials she reviewed, and (4) and the exposure analysis of Dr.

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[2](…continued)
. What DAS has always contended is that exposure to chlorpyrifos is not capable of causing the particular type of injuries (described as cerebral palsy by T.J.'s clinical providers) that T.J. has . . ." [Dkt. 201, pp. 17-18].

Richard Fenske, Ph.D.[3] [Dr. Bearer Decl. ¶ 27; Dr. Bearer Depo., pp. 58-60]. In Tyler Junk's case, besides chlorpyrifos exposure, Dr. Bearer considered as possible causes of his condition genetic abnormality, hypoxia, prematurity, and chorioangioma. [Dr. Bearer Prelim. Rep., p. 1; Dr. Bearer Supp. Rep., pp. 1-2; Dr. Bearer Decl., ¶¶ 18-20].

1. "Ruling In" Toxic Causality

In conducting her differential diagnosis, Dr. Bearer stated that she "ruled in" in utero and postnatal exposure to chlorpyrifos as a "scientifically plausible cause" of Tyler Junk's injury. To prove exposure in a toxic tort case, a plaintiff must provide "evidence from which a reasonable person could conclude that [the] defendant's [action] has probably caused the harm about which they complain." National Bank of Commerce of El Dorado v. Associate Milk Producers, Inc., 191 F.3d 858, 862 (8th Cir. 1999) (quotation omitted). Furthermore, differential diagnosis assumes that general causation has been proven for the list of possible causes. Ruggiero v. Warner-Lambert Co., 424 F.3d 249, 254 (2nd Cir. 2005) (citations omitted). To rule in chlorpyrifos exposure as a scientifically plausible cause of Tyler Junk's injuries, Dr. Bearer stated, "For Tyler Junk, the exposure level was sufficient to cause injury, including neurodevelopmental delay." [Dr. Bearer Decl., ¶15].

Dow AgroSciences contends that Dr. Bearer failed to properly rule in exposure to chlorpyrifos as a scientifically plausible cause of Tyler Junk's injuries because she lacked data regarding Rene and Tyler Junk's exposure levels. However, Junk is only required to show that Tyler Junk was exposed to a quantity of chlorpyrifos that exceeded safe levels. Bonner, 259 F.3d at 931. See, e.g., Bednar, 147 F.3d at 739-40. Dr. Bearer's opinion regarding Tyler Junk's exposure meets that threshold and is scientifically reliable. In her deposition and declaration, she states that she relied on the exposure analysis of Dr.

[3] Junk has designated Dr. Fenske as an expert in exposure analysis and offers his testimony regarding the chlorpyrifos exposure of Rene and Tyler Junk. The admissibility of Dr. Fenske's expert testimony pursuant to Rule 702 has not yet been determined by the Court.

Fenske to conclude that Tyler Junk was exposed to a quantity of chlorpyrifos that exceeded safe levels. [Dr. Bearer Depo., pp. 58-60; Dr. Bearer Decl., ¶ 16]. Dr. Fenske estimated Tyler Junk's exposures based on the amounts of Dursban applied to the Junk home and concluded that his exposure exceeded safety levels. [Dr. Bearer Decl., ¶ 16; Dr. Fenske Rev. Rep., p. 6]. Dr. Bearer's reliance on Dr. Fenske's exposure analysis is consistent with her clinical practice, as she testified that she usually relies on other experts to analyze exposure. [Dr. Bearer Depo., pp. 16, 104-05]. For these reasons, the Court finds that Dr. Bearer's opinion regarding exposure meets the threshold for toxic tort cases and is scientifically reliable under Daubert.[4]

In her report, Dr. Bearer explained how she ruled in chlorpyrifos exposure to be the cause of Tyler Junk's condition.

> [P]renatal/postnatal exposure to chlorpyrifos is associated with developmental delay both in animals and in humans (3-9). The animal studies suggested that chlorpyrifos reduces neuronal proliferation in the central nervous system and blocks the neurotrophic effects of acetylcholine (10). The window of vulnerability for organophosphates is likely to extend from the embryonic period into postnatal life (4). Exposures during the spurt in brain growth, which in human pregnancies begins during the third trimester, may be particularly deleterious (5, 6, 10-13). Some of the effects of chlorpyrifos appear to be delayed and emerge later as behavioral anamolies (14).

[Dr. Bearer Prelim. Rep., p. 1; Dr. Bearer Supp. Rep., p. 2].

Dr. Bearer explained further in her supplemental report her method of "ruling in" chlorpyrifos exposure as the cause of Tyler Junk's condition.

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[4] The Court has yet to determine the admissibility of Dr. Fenske's expert testimony regarding Rene and Tyler Junk's exposure to chlorpyrifos. Dr. Bearer's expert causation opinions will be reexamined to the extent that they are based on Dr. Fenske's exposure analysis when the admissibility of the expert testimony of Dr. Fenske is before the Court.

> Dursban was applied in the Junk household 17 times between Tyler's conception in early 1992 and September 1994. Tyler experienced multiple exposures to chlorpyrifos, Dursban's active ingredient, *in utero* and after birth, as outlined on pages 2-4 of Dr. Abou-Donia's report. After birth, Tyler's exposure to chlorpyrifos likely included inhalation, skin and ingestion exposure, as further noted by Dr. Abou-Donia on page 4 of his report. Chlorpyrifos is directly toxic to the nervous system. Chlorpyrifos is capable of teratogenic effects, as detailed on pages 5-7 of Dr. Abou-Donia's report.
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> Symptoms and conditions experienced by Tyler and Rene Junk are indicative of toxic exposure to chlorpyrifos. For example, during pregnancy, Rene experienced nausea, diarrhea, rash, vomiting and spotting following Dursban application. Ultrasound on July 6, 1992 noted large chorioangioma in umbilical cord. On August 25, 1992 hemangioma was noted. Following Tyler's birth, Renee [sic] was diagnosed with pulmonary edema.
>
> For Tyler Junk, Dursban exposure was sufficient to cause injury, including neurodevelopmental delay referred to as cerebral palsy. The toxic effect of chlorpyrifos is amplified in infants, and the effect is even more pronounced for a fetus *in utero*. Although it may be impossible to quantify precisely the amount of Dursban to which Tyler Junk was exposed, it is more likely than not that the multiple exposures, *in utero* and after birth, caused Tyler's neurodevelopmental delay.

[Dr. Bearer Supp. Rep., p. 2].

In her deposition, Dr. Bearer laid out the scientific principles by which she determined Tyler's condition was caused by chemical exposure.

> I determined that an exposure had occurred to an agent that had been reported to cause the same effect that I saw in Tyler. That it was a significant exposure in terms of number of times the house had been sprayed, the EPA warning that any kind of indoor exposure exceeds the level of concern. I determined that the exposure had occurred during the time when Tyler's

> brain was developing, so it was during the critical period of time. I also did not think that there was any other causes for his neurodevelopmental delay.

[Dr. Bearer Depo., pp. 129-30].

The Court finds that Dr. Bearer sufficiently went through the process to rule in chlorpyrifos exposure as a scientifically plausible cause of Tyler Junk's condition. "Under Daubert, expert opinions employing a differential diagnosis must be based on scientifically valid decisions as to which potential causes should be 'ruled in' and 'ruled out.'" Ervin v. Johnson & Johnson, Inc., 492 F.3d 901, 904 (7th Cir. 2007) (citation omitted). Dr. Bearer's methods for determining toxic causality of Tyler Junk's condition mirror those outlined in the Reference Manual on Scientific Evidence.

> An expert who opines that exposure to a compound caused a person's disease engages in deductive clinical reasoning. In most instances, cancers and other diseases do not wear labels documenting their causation. The opinion is based on an assessment of the individual's exposure, including the amount, the temporal relationship between the exposure and the disease, and other disease-causing factors. This information is then compared with scientific data on the relationship between exposure and disease.

Reference Manual on Scientific Evidence, 422-23 (Federal Judicial Center, 2d. ed. 2000). Dr. Bearer assessed Tyler Junk's exposure levels by utilizing Dr. Fenske's report. [Dr. Bearer Depo., pp. 58-60; Dr. Bearer Decl., ¶ 16]. She analyzed the temporal relationship between chlorpyrifos exposure and Rene and Tyler Junk's symptoms and relied on Dr. Abou-Donia's report regarding the timing of applications and symptoms. [Dr. Bearer Supp. Rep., p. 2; Dr. Bearer Depo., pp. 60-63, 156; Dr. Bearer Decl., ¶ 14]. Dr. Bearer compared the information about Tyler Junk with the scientific data relating to in utero and postnatal exposure to chlorpyrifos and fetal neurologic harm. [Dr. Bearer Supp. Rep., p. 2; Dr. Bearer Decl. ¶¶ 11-16]. Dr. Bearer's "ruling in" of chlorpyrifos exposure as a scientifically plausible cause of Tyler Junk's injury meets the requirements of Daubert.

2. "Ruling Out" Other Plausible Causes

Dr. Bearer stated that she considered and "ruled out" genetic abnormality, hypoxia, prematurity, and chorioangioma as other possible causes of Tyler Junk's injury. [Dr. Bearer Prelim. Rep., p. 1; Dr. Bearer Supp. Rep., pp. 1-2; Dr. Bearer Dec., ¶¶ 17-20].

a. Genetic Abnormality

In her two reports and in her deposition, Dr. Bearer stated that she excluded genetic abnormality as a possible cause of Tyler Junk's condition because his treating physicians did not suspect it as a cause and no genetic tests were sent. [Dr. Bearer Prelim. Rep., p. 1; Dr. Bearer Supp. Rep., p. 1; Dr. Bearer Depo., p. 163]. In her declaration, Dr. Bearer further clarified that no medical evidence exists to establish that Tyler Junk's condition is consistent with a genetic abnormality or was caused by a heritable genetic factor. [Dr. Bearer Decl., ¶ 17].

b. Prematurity

In her deposition, Dr. Bearer excluded Tyler Junk's premature birth as a possible cause of his condition. [Dr. Bearer Depo., p. 136]. Dr. Bearer stated, "In 32 week infants with very transient respiratory distress, we don't see cerebral palsy as being a sequela. He has the same chance as a term baby for having CP." [Dr. Bearer Depo., p. 136]. In her declaration, Dr. Bearer explained that a correlation between clinical findings and the medical records is the first step in determining whether prematurity is associated with injury. [Dr. Bearer Decl., ¶ 19(dd)]. Dr. Bearer stated that Tyler Junk's prematurity does not correlate with any clinical findings sufficient to conclude that his prematurity was the cause of his condition. [Dr. Bearer Decl., ¶ 19(dd)]. Based on the lack of correlation between Tyler Junk's medical records and clinical findings, Dr. Bearer ruled out prematurity as a possible cause of Tyler Junk's neurodevelopmental delay.

Dr. Bearer stated that she used her own experiences as a neonatologist and statistical support from peer-reviewed articles to support her opinion "ruling out" prematurity. Based

on her experience as a neonatologist, Dr. Bearer stated that there is "no associational relationship" between infants born at Tyler Junk's gestational age (32 weeks) with the same transient respiratory distress and neurologic impairment and delay. [Dr. Bearer Decl. ¶ 19(dd)]. She stated that prematurity is a substantial risk factor for neurological disorders when infants are born at 24 to 26 weeks of gestation. [Dr. Bearer Decl. ¶ 19(ee)]. She quoted a statistic from a peer-reviewed article that states, "The prevalence of cerebral palsy was 20% at 24 to 26 weeks of gestation, compared with 4% at 32 weeks." [Dr. Bearer Decl., ¶1 9(ee)].[5] She quoted another study that stated, "[t]he changes in the rate of cerebral palsy by gestational age fall over the period for children born at 28-31 weeks' gestation." [Dr. Bearer Decl. ¶ 19(ee)].[6] Dr. Bearer also considered Tyler Junk's birth weight of five pounds in "ruling out" prematurity as a cause of his condition. [Dr. Bearer Decl., ¶ 19(ee)]. Citing a peer-reviewed article, Dr. Bearer stated that the incidence for major handicaps occur in six-percent to eight-percent of infants in Tyler Junk's birth weight range. [Dr. Bearer Decl., ¶ 19(ee)].[7]

c. Hypoxia

Dr. Bearer ruled out hypoxia, or low fetal oxygen circulation, because her review of Tyler Junk's medical records allegedly showed that no evidence of hypoxia existed at the time of delivery. [Dr. Bearer Decl., ¶ 18]. While there is an issue of fact as to whether Tyler Junk suffered from hypoxia, Dr. Bearer sufficiently considered this evidence in

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[5] Pierre-Yves Ancel et al., *Cerebral palsy among very preterm children in relation to gestational age and neonatal ultrasound abnormalities: The EPIPAGE cohort study*, 117 Pediatrics 828-835 (2006).

[6] MJ Platt et al., *Trends in cerebral palsy among infants of very low birthweight (<1500 g) or born prematurely (<32 weeks) in 16 European centres: a database study*, Lancet. 2007; 369:43-50.

[7] GP Aylward, *Neurodevelopmental outcomes of infants born prematurely*, J. Dev. Behav. Pediatr. 2005; 26:427-40.

giving her opinion. Dr. Bearer stated that Tyler Junk's level of nucleated red blood cells do not suggest that he was hypoxic at delivery, or that hypoxia was the cause of his condition. [Dr. Bearer Decl., ¶ 18(bb)]. Dr. Bearer stated that Tyler Junk's umbilical cord pH at delivery was normal, thereby demonstrating that he was not hypoxic prior to delivery. [Dr. Bearer Decl., ¶ 18(bb)].

In her preliminary and supplemental reports, Dr. Bearer maintained that Tyler Junk's first blood gas was taken at two hours of life and showed no acidosis or base deficit indicating hypoxia. [Dr. Bearer Prelim. Rep., p.1; Dr. Bearer Sup. Rep., pp. 1-2]. In her deposition and declaration, however, Dr. Bearer acknowledged that an earlier test, Tyler Junk's foot stick blood gas test from 15 minutes of life, showed that he suffered from respiratory acidosis. [Dr. Bearer Depo., p. 86; Dr. Bearer Declaration, ¶ 18(bb)]. According to Dr. Bearer, respiratory acidosis is an excess of carbon dioxide in the lungs, not a lack of oxygen in the bloodstream. [Dr. Bearer Decl., ¶ 18(bb)]. Tyler Junk was treated for that condition by receiving supplemental oxygen and being placed in the neonatal intensive care unit. [Dr. Bearer Depo., pp.86-87]. Dr. Bearer opined that Tyler Junk's respiratory acidosis was "transient" not unusual for a newborn, and not empirically associated with neurological harm. [Dr. Bearer Decl., ¶ 18(bb)]. She further stated that respiratory acidosis of that degree and nature is not connected to neurologic harm. [Dr. Bearer Decl., ¶ 18(bb)]. Dr. Bearer stated that Tyler Junk's Apgar scores at the time of birth did not warrant "acute concern." [Dr. Bearer Decl., ¶ 18(cc)]. Tyler Junk's umbilical cord was noted to be of normal length. [Dr. Bearer Decl., ¶ 18(cc)]. Finally, Dr. Bearer stated that if hypoxia was the cause of Tyler Junk's neurodevelopmental delay, then damage to other organs, such as the kidneys or liver, would have been observed because it is the natural process of the fetus to sacrifice functions of other organs prior to the brain. [Dr. Bearer Decl., ¶ 18(cc)]. Dr. Bearer states that no such damage to the

organs existed at Tyler Junk's birth, therefore indicating that his neurodevelopmental delay was not caused by hypoxia. [Dr. Bearer Decl., ¶ 18(cc)].

d. Umbilical Chorioangioma

Dr. Bearer acknowledged that the chorioangioma in Tyler Junk's umbilical cord was unusual, and she testified that it may have had some effect on him in utero. [Dr. Bearer Depo., pp. 135-36]. However, Dr. Bearer ruled it out as the cause of the Tyler Junk's condition because of her belief the medical evidence did not show that the chorioangioma caused Tyler Junk to suffer decreased blood supply in utero or hypoxia at time of birth. Dr. Bearer referenced medical records from Tyler Junk's ultrasounds, stating that physicians noted that "placental blood flow appeared normal" on June 30, 1992. [Dr. Bearer Declaration, ¶ 18(cc)]. A discussion of Dr. Bearer's "ruling out" of hypoxia appears above and is incorporated herein. Furthermore, Dr. Bearer opined that chorioangiomas are not associated with neurodevelopmental delay. [Dr. Bearer Prelim. Rep., p. 1; Dr. Bearer Supp. Rep., p. 2; Dr. Bearer Depo., pp. 132, 136; Dr. Bearer Decl., ¶ 20]. Dr. Bearer stated that chorioangiomas are associated with severe hydrops fetalis, from which Tyler Junk did not suffer. [Dr. Bearer Prelim. Rep., p. 1; Dr. Bearer Supp. Rep., p. 2; Dr. Bearer Depo., pp. 132, 136; Dr. Bearer Decl., ¶ 20].

After excluding the other possible causes for Tyler Junk's neurodevelopmental delay, Dr. Bearer concluded that the only remaining plausible cause of his condition was in utero and postnatal exposure to chlorpyrfios.

The Court finds that Dr. Bearer sufficiently went through the process to rule out other possible causes of Tyler Junk's injury. When completing a differential diagnosis, an expert must "systematically rule[] out" all other possible causes. Turner, 229 F.3d at 1208.

> In making its initial determination of whether proffered testimony is sufficiently reliable, the court has broad latitude to consider whatever factors bearing on validity that the court finds to be useful; the particular factors will depend on the unique circumstances of the expert testimony involved.

Westberry, 178 F.3d at 261 (citation omitted). Dr. Bearer considered genetic abnormality, prematurity, hypoxia, and the umbilical cord chorioangioma as other possible causes of Tyler Junk's injury. Dr. Bearer stated that she directly addressed the medical evidence that Dow AgroSciences and Terminix contends supports other plausible causes of Tyler Junk's injury. Dr. Bearer's exclusion of other plausible causes was not conclusory in nature; she stated that she analyzed Tyler Junk's medical records and provided evidence from those records to support her determinations. Dr. Bearer provided scientific research to support her exclusion of other plausible causes of Tyler Junk's injury. Furthermore, Dr. Bearer is a neonatologist with more than twenty years of experience. [Junk App., p. 1]. She is board-certified in pediatrics and neonatal/perinatal medicine. [Junk App., p. 6]. She is the director of a neonatalogy training program and an associate professor in pediatrics and neurosciences. [Junk App., p. 1]. Dr. Bearer is well-qualified to interpret the medical evidence regarding Tyler Junk's prenatal health and his premature birth. In sum, Dr. Bearer's "ruling out" of other possible causes using the medical evidence, her more than twenty years of experience as a neonatologist, and scientific research meets the requirements of Daubert.

"[N]othing in Rule 702, *Daubert*, or its progeny requires 'that an expert resolve an ultimate issue of fact to a scientific absolute in order to be admissible." Kudabeck, 338 F.3d at 861(quoting Bonner, 259 F.3d at 929). "[The] requirement [of "ruling out" other plausible causes] cannot be carried to the quixotic extreme." Lauzon v. Senco Products, Inc., 270 F.3d 681, 693 (8th Cir 2001). A differential diagnosis is "patient-specific" and is evaluated under Daubert for admissibility on a "case-specific" basis. Ruggiero, 424 F.3d at 254 ("A differential diagnosis is a 'a patient-specific process of elimination . . . ."); Hollander v. Sandoz Pharmaceuticals Corp., 289 F.3d 1193, 1210 (10th Cir. 2002) ("Because the *Daubert* reliability inquiry is case-specific . . . ."). "If a properly qualified medical expert performs a reliable differential diagnosis through which, to a reasonable

degree of medical certainty, all other possible causes of the victims' condition can be eliminated, leaving only the toxic substance as the cause, a causation opinion based on that differential diagnosis should be admitted." Turner, 229 F.3d at 1209. "We agree that a medical opinion about causation, based upon a proper differential diagnosis, is sufficiently reliable to satisfy *Daubert*." Id. at 1208. (citations omitted). For the foregoing reasons, the Court finds that Dr. Bearer's differential diagnosis is scientifically reliable and admissible pursuant to Rule 702.

V. CONCLUSION

The Court finds that Dr. Bearer's opinions regarding general and specific causation are admissible. Accordingly, Dow AgroSciences' and Terminix's motions to exclude the expert causation testimony of Dr. Bearer [Dkt. Nos. 140, 143] are denied.

DATED this 11th day of September, 2008.

JOHN A. JARVEY
UNITED STATES DISTRICT JUDGE
SOUTHERN DISTRICT OF IOWA