

Def.'s Interrogs. No. 18. Traecina's blood was tested for the presence of lead ten times between August of 1997 and February of 2008. The results of such testing are as follows:

Table 1. Summary of Traecina's Blood Lead Levels²

Date	Blood Lead Level (BLL) (mcg/dl)³
8/18/97	25
12/24/97	19
7/13/98	51
7/17/98	31
7/22/98	36
9/2/98	20
10/27/98	15
12/15/98	20
2/24/99	15
2/27/08	4

Following the detection of elevated levels of lead in Traecina's system, "the RI Department of Health inspected the [Defendant's property] on July 22, 1998. [Wendy] was told that there were lead paint hazards on the property at 71 Magill Street, Pawtucket, RI." Id. No. 27. On August 14, 1998, the Rhode Island Department of Health issued a Notice of Violation to Defendant. Pl.'s Compl. ¶ 9.

II

Parties' Arguments

Plaintiff alleges that during her tenancy at 71 Magill Street, she was exposed to "dangerous, hazardous and illegal levels of lead-based paint, plaster, and materials inside the dwelling and generally within and about the [Defendant's] dwelling." Id. at ¶ 5. As such, Plaintiff alleges that the direct and proximate cause of her injuries was Defendant's negligence through, *inter alia*, allowing the Plaintiff to occupy a dwelling that contained potentially

² The table was taken from the report of Plaintiff's expert, Dr. James Besunder, D.O. See Pl.'s Obj. to Def.'s Mot. for Summ. J., Ex. D (Dr. Besunder's Report).

³ Blood lead levels (BLL) are measured in micrograms per deciliter (mcg/dl).

hazardous materials on the interior surfaces; allowing the Plaintiff to occupy a dwelling that contained lead levels in excess of the acceptable environmental lead levels; and failing to inform Plaintiff's parents of violations in the dwelling of the health and safety codes. See id. at ¶ 15.⁴ In support of Plaintiff's allegations, she proposes to use the testimony of Theodore L. Lidsky, Ph.D. (Dr. Lidsky) and James Besunder, D.O. (Dr. Besunder) for the proposition that exposure to lead caused her cognitive deficiencies.

Defendant has filed a motion for summary judgment arguing that (1) Plaintiff's expert, Dr. Lidsky, is a psychologist, not a physician, and thus cannot give a medical opinion as to causation; and (2) Plaintiff's experts have not established that exposure to lead, at 71 Magill Street, was the proximate cause of Traecina's injuries. With respect to said exposure, Defendant argues that neither Dr. Lidsky nor Dr. Besunder can say, with any probability or degree of certainty, that exposure to lead at 71 Magill Street was the proximate cause of the Plaintiff's injuries. See Def.'s Mot. for Summ. J. 19. In response, Plaintiff contends that (1) Dr. Lidsky's opinion on causation is admissible; and (2) the reports of Dr. Besunder and Dr. Lidsky, taken together, establish that Traecina's exposure to lead, at 71 Magill Street, proximately caused her injuries. See Pl.'s Mem. in Opp'n to Def.'s Mot. for Summ. J. 3, 18.

Following the Defendant's summary judgment motion, Plaintiff filed a series of motions *in limine*. Specifically, Plaintiff's motions *in limine* ask the Court to (1) adopt the "indivisible injury" rule; (2) exclude or limit the testimony of the Defendant's expert, Arlene Weiss; (3) exclude any mention of alleged domestic violence or abuse; (4) exclude any mention of Plaintiff's father's criminal record; (5) exclude any mention or evidence regarding the mental or

⁴ The Plaintiff has also brought a negligent misrepresentation claim; however, as the Defendant's summary judgment motion only raises the proximate causation issue as to the negligence claim, this Court shall cabin its analysis accordingly.

physical condition of the Plaintiff's siblings or parents; and (6) exclude or limit the testimony of Defendant's expert, Paul Chervin, M.D. The Court shall address the summary judgment motion and the motions *in limine* in turn.

III

Standard of Review

A

Summary Judgment

“Summary judgment is proper if no genuine issues of material fact are evident from ‘the pleadings, depositions, answers to interrogatories, and admissions on file, together with the affidavits, if any’ and, in addition, the motion justice finds that the moving party is entitled to prevail as a matter of law.” Lavoie v. N.E. Knitting, Inc., 918 A.2d 225, 227-28 (R.I. 2007) (citing Super. R. Civ. P. 56(c)). It is well-settled that a genuine issue of material fact is one about which reasonable minds could differ. See, e.g., Brough v. Foley, 572 A.2d 63, 67 (R.I. 1990).

The moving party bears the initial burden of establishing that no such issues exist. Heflin v. Koszela, 774 A.2d 25, 29 (R.I. 2001). If the moving party is able to sustain its burden, then the “litigant opposing a motion for summary judgment has the burden of proving by competent evidence the existence of a disputed issue of material fact and cannot rest upon mere allegations or denials in the pleadings, mere conclusions or mere legal opinions.” Am. Express Bank, FSB v. Johnson, 945 A.2d 297, 299 (R.I. 2008) (citations omitted). Although the opposing party must demonstrate evidence beyond mere allegations, it need not disclose all of its evidence. See, e.g., Ludwig v. Kowal, 419 A.2d 297, 301 (R.I. 1980); Nichols v. R.R. Beaufort & Assocs., Inc., 727 A.2d 174, 177 (R.I. 1999); see also Bourg v. Bristol Boat Co., 705 A.2d 969, 971 (R.I. 1998) (citations omitted).

The trial judge reviews the evidence without passing upon its weight and credibility, and will deny a motion for summary judgment if the party opposing the motion has demonstrated the existence of a triable issue of fact. See Mitchell v. Mitchell, 756 A.2d 179, 181 (R.I. 2000); Palmisciano v. Burrillville Racing Ass'n, 603 A.2d 317, 320 (R.I. 1992). However, the Court will enter summary judgment “against a party who fails to make a showing sufficient to establish the existence of an element essential to that party’s case.” Lavoie, 918 A.2d at 227-28.

B

Motions in Limine

A motion *in limine* is “widely recognized as a salutary device to avoid the impact of unfairly prejudicial evidence upon the jury and to save a significant amount of time at the trial.” BHG, Inc. v. F.A.F., Inc., 784 A.2d 884, 886 (R.I. 2001) (quoting Gendron v. Pawtucket Mut. Ins. Co., 409 A.2d 656, 659 (Me. 1979)). “As [the motion *in limine*] has developed, it has become a tool for narrowing the issues at trial and enhancing the parties’ preparation for trial. Despite this development, it seems clear that a motion *in limine* is not intended to be a dispositive motion.” Ferguson v. Marshall Contractors, Inc., 745 A.2d 147, 150 (R.I. 2000) (citing Gendron, 409 A.2d at 660). “Rather, it has been used in this state primarily to ‘prevent the proponent of potentially prejudicial matter from displaying it to the jury . . . in any manner until the trial court has ruled upon its admissibility in the context of the trial itself.’” Ferguson, 745 A.2d at 150 (quoting Lagenour v. State, 268 Ind. 441, 376 N.E.2d 475, 481 (1978)).

Furthermore, “[i]t is well settled that ‘[d]ecisions about the admissibility of evidence on relevancy grounds are left to the sound discretion of the trial justice.’” State v. Cook, 782 A.2d 653, 654 (R.I. 2001) (quoting State v. Botelho, 753 A.2d 343, 350 (R.I. 2000)). Accordingly, “[a]bsent a showing of abuse of discretion [our Supreme Court] will not overturn [a] trial

justice's ruling on the admissibility of evidence.” State v. Oliveira, 774 A.2d 893, 917 (R.I. 2001).

IV

Analysis

A

Summary Judgment

“In this lead paint exposure personal injury action [the] plaintiff[] ha[s] the burden of proving: 1) the plaintiff[’s] exposure to lead; 2) general causation, which is proof that the toxin in question (lead) can in fact cause the illness . . . ; and 3) specific causation—meaning the likelihood that plaintiff[’s] illness was caused by lead, including eliminating other potential causes of the disease.” Adams v. Rizzo, 831 N.Y.S.2d 351 (Sup. Ct. 2006). Moreover, “[t]o prove causation in a toxic tort case, a plaintiff 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 v. ISP Technologies, Inc., 259 F.3d 924, 928 (8th Cir. 2001); see Miranda v. Dacruz, 2009 WL 3515196, at *3 (R.I. Super. Oct. 26, 2009) (Gibney, P.J.) (quoting 3 Faigman, Kaye, Saks & Sanders, Modem Scientific Evidence § 23:2, at 5 (2005-2006 ed.) (“In a toxic tort case, it is necessary for the plaintiff to prove by a preponderance of the evidence both general and specific cause for the medical condition suffered by the plaintiff. ‘General causation asks whether exposure to a substance causes harm to anyone. Specific causation asks whether exposure to a substance caused a particular plaintiff’s injury.’”). Here, the genesis of the present summary judgment motion stems from the proposed testimony of the Plaintiff’s expert witnesses regarding proximate causation.

Admissibility of Dr. Lidsky's Testimony

The first issue before this Court is whether Dr. Lidsky—a licensed psychologist, with a specialty in neuropsychology⁵—is qualified under Rule 702 to testify as to the general causation of lead poisoning. Dr. Lidsky's professional focus includes the effects of drugs and metals on the brain and behavior; for more than two decades, he has concentrated his work on the effects of lead on the developing nervous system. See Pl.'s Obj. to Def.'s Mot. for Summ. J., Ex. A (Dr. Lidsky's Curriculum Vitae). Furthermore, he teaches and has done research on the relationship between cognitive deficits and lead toxicity. Id.

a

Dr. Lidsky's Report and Deposition Testimony

Dr. Lidsky performed a standard battery of tests in order to determine if Traecina suffered from any cognitive deficiencies.⁶ Dr. Lidsky's tests indicated both areas of cognitive

⁵ “A clinical neuropsychologist is a professional within the field of psychology with special expertise in the applied science of brain-behavior relationships. Clinical neuropsychologists use this knowledge in the assessment, diagnosis, treatment, and/or rehabilitation of patients across the lifespan with neurological, medical, neurodevelopmental and psychiatric conditions, as well as other cognitive and learning disorders. The clinical neuropsychologist uses psychological, neurological, cognitive, behavioral, and physiological principles, techniques and tests to evaluate patients' neurocognitive, behavioral, and emotional strengths and weaknesses and their relationship to normal and abnormal central nervous system functioning. The clinical neuropsychologist uses this information and information provided by other medical/healthcare providers to identify and diagnose neurobehavioral disorders, and plan and implement intervention strategies.” Pl.'s Mem. at 15 (quoting Official Statement of the National Academy of Neuropsychology (NAN), Definition of a Clinical Neuropsychologist, The Clinical Neuropsychologist 1989, Vol. 3, No.1, p. 22, modified in 2001).

⁶ Dr. Lidsky administered (1) the Wechsler Adult Intelligence Scale, 3 Edition (WAIS-III) to establish IQ; (2) the Controlled Word Association Test and the Visual Naming Test to assess language skills; (3) the Diamonds subtest of the WRIT to test sensory-motor functioning; (4) the Conners' Continuous Performance Test (CPT II V.5) to assess attention; (5) the Story Learning, Shape Learning, and List Learning subtests from the Neuropsychological Assessment Battery (NAB), and Digit Span subtest from WMS-III to measure learning and memory; (6) the

deficiency as well as other functions within the normal range. On one hand, the “[n]europsychological testing identified focal impairments of naming, impulse control, scanning, sustained attention, verbal learning and memory as well as visual memory.” Lidsky Report 5. On the other hand, Traecina’s “overall level of intellectual functioning, as reflected by IQ, [was] in the Average range” and “other aspects of [Traecina’s] neurocognitive functioning (e.g., verbal fluency, construction, concept formation and abstract reasoning) were within normal limits.” Id.

The neuropsychological test that Dr. Lidsky administered to Traecina was a standard test which was designed only to “test for brain injury . . . no matter what the cause of th[at] brain injury[.]” Lidsky Dep. 66:14-25, Apr. 15, 2015. He admitted that this battery of tests “would [be] give[n] to any[one] . . . who [was] suspected of brain damage.” Id. at 66:9-13. Accordingly, “the results of this [test] would only tell [him] if a person ha[d] a brain injury[.]” not whether the injury was a result of lead poisoning. Id. at 66:22-24.

Dr. Lidsky testified that because lead does not have a “signature injury[.]” id. at 26:6-11, there is not a “particular pattern of neuropsychological testing results that would indicate an injury caused by lead[.]” Id. at 26:13-19. As such, he could not simply “look at the neuropsychological test results and say ‘Lead did this.’” Id. at 26:9-11. However, his analysis included reviewing all of a patient’s medical records in order to perform a “differential diagnosis[.]”⁷ Id. at 14:9-18; 15:7-21. In this case, Dr. Lidsky’s review of Traecina’s medical

Similarities, Picture Arrangement, and Matrix Reasoning subtests of the WAIS-III to test executive functions; (7) and the Test of Memory Malingering to detect lack of effort, exaggeration, or cheating. See Pl.’s Obj. to Def.’s Mot. for Summ. J., Ex. F (Dr. Lidsky’s Report).

⁷A “[d]ifferential diagnosis is ‘a standard scientific technique of identifying the cause of a medical problem by eliminating the likely causes until the most probable one is isolated.’” Pluck v. BP Oil Pipeline Co., 640 F.3d 671, 678 (6th Cir. 2011) (quoting Hardyman v. Norfolk & W. Ry. Co., 243 F.3d 255, 260 (6th Cir. 2001)); see Baker v. Dalkon Shield Claimants Trust, 156 F.3d 248, 253 (1st Cir. 1998) (“[D]ifferential diagnosis is a standard medical technique.”).

records revealed, in his opinion, two significant pieces of information. First, Traecina’s medical history indicated that she “had a significant level of lead at a peak time of vulnerability.” *Id.* at 49: 12-14. Second, “[r]eview of her medical history identifie[d] no other influence, event or illness, with the potential to derail normal brain development.” Lidsky Report 5. In light of the fact that “cognitive impairments similar to those seen in [Traecina] [were] observed following childhood exposure to lead, and the absence of other factors that could explain her neuropsychological deficits[,]” Dr. Lidsky opined that “brain damage from lead underlies [Traecina’s] neuropsychological impairments[.]”⁸ *Id.*

b

General Causation under Rule 702

“In a toxic tort case, general causation⁹ requires that the plaintiff ‘show that the toxicant in question is capable of causing the injury complained of[.]’” *Arias v. DynCorp*, 928 F. Supp.

⁸ Dr. Lidsky’s deposition testimony reads, in pertinent part:

“Q. So you are in fact making a causal connection between Tra[ecina]’s elevated blood-lead levels and her cognitive d[e]ficiencies; would that be correct?

“A. Yes.” Lidsky Dep. 159:10-14.

⁹ ““In a toxic tort case, general causation addresses whether a substance is capable of causing a particular injury or condition in a population, while specific causation addresses whether a substance caused a particular individual’s injury.”” *Blanchard v. Goodyear Tire & Rubber*, 30 A.3d 1271, 1274-75 (2011) (quoting *King v. Burlington Northern Santa Fe Ry.*, 762 N.W.2d 24, 34 (2009)). Here however, neither party addresses whether Dr. Lidsky’s opinion is directed at establishing general or specific causation. Nevertheless, Dr. Lidsky’s deposition testimony clearly indicates that he does not have an opinion as to specific causation. His testimony reads, in relevant part:

“Q. So you couldn’t say that, in your conclusion of your report, . . . when you opine that brain damage from lead underlies the neuropsychological impairments, you could not causally connect that to my client?

2d 10, 20-21 (D.D.C. 2013) (quoting Young v. Burton, 567 F. Supp. 2d 121, 128 (D.D.C. 2008) aff'd, 354 F. App'x 432 (D.C. Cir. 2009)). “General causation conclusions are relevant when they form a link in a causal chain that helps a jury reach a conclusion on the ultimate causation question.” Perry v. Novartis Pharm. Corp., 564 F. Supp. 2d 452, 463 (E.D. Pa. 2008). “As other courts have recognized, while ‘the incidence of adverse effects in the general population[,] when exposed, cannot indicate the actual cause of a given individual’s disease or condition,’ the admission of general causation evidence is an attempt to ‘balance the need to compensate those who have been injured by the wrongful actions of another with the concept deeply imbedded in our jurisprudence that a defendant cannot be found liable for an injury unless the preponderance of the evidence supports cause in fact.’” Id. (quoting Merrell Dow Pharm. Inc. v. Havner, 953 S.W.2d 706, 718 (Tex. 1997)).

“A. No. I don’t know who your client is.” Lidsky Dep. 54:6-13.

“ . . .

“Q. So, you don’t have or you don’t get, you’re not concerned with necessarily the source of the lead?

“A. Correct.” Id. at 50:18-21.

“ . . .

“Q. Now, in this case, do you know where Traecina was exposed to lead?

“A. No. That’s really outside of my area of expertise.

“Q. You weren’t retained for that purpose?

“A. Correct.” Id. at 53:23-54:5.

As such, it is apparent that Dr. Lidsky intends to speak to general causation, i.e., that exposure to lead can cause the type of ailments from which the Plaintiff claims to suffer. Properly framed, the issue this Court shall now address is whether Dr. Lidsky is qualified to give an expert opinion as to *general* causation.

However, “[b]efore an expert is permitted to testify [as to general causation], the trial court applies Rhode Island Rule of Evidence 702¹⁰, commonly referred to as the Daubert¹¹ test, to determine whether the witness is able to clear two evidentiary hurdles: (1) he or she has applicable expert qualifications, and (2) he or she will impart reliable and relevant expert testimony that will assist the trier of fact. A party must prove by a preponderance of the evidence that its expert can meet these two requirements.” Miranda, 2009 WL 3515196, at *2. “The [Daubert] Court described the first requirement as one of ‘evidentiary reliability,’ and the second as one of ‘fit’ or relevance. Amorgianos v. Nat’l R.R. Passenger Corp., 137 F. Supp. 2d 147, 162 (E.D.N.Y. 2001) aff’d sub nom. Amorgianos v. Nat’l R.R. Passenger Corp., 303 F.3d 256 (2d Cir. 2002) (quoting Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 590 (1993)); see Knight v. Kirby Inland Marine Inc., 482 F.3d 347, 352 (5th Cir. 2007) (“The Daubert standard ensures that the proffered evidence is both ‘reliable’ and ‘relevant.’”). “Reliability is determined by assessing ‘whether the reasoning or methodology underlying the testimony is scientifically valid.’ Relevance [or ‘fit’] depends upon ‘whether [that] reasoning or methodology properly can be applied to the facts in issue.’” Knight, 482 F.3d at 352 (quoting Daubert, 509 U.S. at 592-93).

¹⁰ Rule 702 of the Rhode Island Rules of Evidence (Rule 702), provides as follows:

“If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of fact or opinion.” R.I. R. Evid. 702.

¹¹ Our Supreme “Court has recognized the applicability of Daubert to situations in which scientific testimony is proposed in Rhode Island state courts (that is, where Rule 702 of the Rhode Island Rules of Evidence comes into play).” See Raimbeault v. Takeuchi Mfg. (U.S.) Ltd., 772 A.2d 1056, 1061 (R.I. 2001).

Reliability

In order “[t]o meet the threshold for reliability, a neuropsychologist . . . must demonstrate that the individual tests he or she administered as part of the battery, not the battery as a whole, have been tested, have been subject to peer review and publication, and have known or potential error rates.” Baxter v. Temple, 949 A.2d 167, 184 (N.H. 2008). Here, Dr. Lidsky administered a comprehensive battery of tests in order to gauge the Plaintiff’s language skills, sensory-motor functions, attention, learning and memory abilities, executive functions, and the Plaintiff’s level of effort. See Lidsky Report 2-6. The Defendant has articulated no argument that the specific tests performed by Dr. Lidsky do not constitute reliable principles and methods by which a neuropsychologist can determine whether a cognitive deficit exists. Moreover, Dr. Lidsky testified that “[i]n 2015, there were 20 peer-reviewed papers using the WAIS-III” test and although the battery of tests can produce a false negative, such is “true of any battery.” See Lidsky Dep. 77:7-8; 65:22-25. Accordingly, this Court finds that the flexible battery approach, as administered by Dr. Lidsky, is generally a reliable approach to neuropsychological assessment, and is thus a reliable methodology for determining a person’s cognitive status. See Bado-Santana v. Ford Motor Co., 482 F. Supp. 2d 192, 196 (D.P.R. 2007) (finding a neuropsychologist to be “qualified to render expert testimony” on mild traumatic brain injury allegedly resulting from car accident); c.f. Baxter, 949 A.2d at 185 (finding that, despite “the somewhat subjective nature of plaintiffs’ neurocognitive injuries,” neuropsychologist had a reliable basis to testify to test results of plaintiffs with lead exposure, and that plaintiffs suffered from certain neurocognitive deficits, where neuropsychologist administered a battery of tests to plaintiffs, including the WISC III “and selected subsets of the . . . Children’s Memory Scale”).

Nonetheless, Defendant does not argue that Dr. Lidsky is unqualified to testify regarding the Plaintiff's cognitive abilities; rather, Defendant contends Dr. Lidsky is not qualified to opine as to the cause of such brain injuries. Specifically, Defendant argues that as a psychologist, Dr. Lidsky is not qualified, under Rule 702, to testify that exposure to lead can cause the injuries Traecina is alleged to have suffered.

“[T]he fact that [Dr. Lidsky] is not a neurologist or physician[,] [however,] does not resolve whether [he] is qualified to render expert testimony on” the causal relationship between brain damage and lead poisoning. Bado-Santana, 482 F. Supp. 2d at 195; see U.S. v. Hoffman, 832 F.2d 1299, 1310 (1st Cir. 1987) (explaining that “[e]xpertise is not necessarily synonymous with a string of academic degrees or multiple memberships in learned societies” and emphasizing the value of “extensive practical experience”). In fact, “the American Psychological Association has stated that neurological examinations are very limited in their capacity to detect brain damage, and that neuropsychological testing is the only means of diagnosing some forms of brain damage.” Id. at 195; see Lidsky Dep. 65:16-19 (“Neuropsychological testing is very -- you have to have a real deficit to show up with a problem in neuropsychological testing.”). “Although expert testimony is generally used to establish each of the links of causation in a lead-based paint case, certainly, there is no requirement that causation be proved by direct proof or with absolute certainty. Circumstantial evidence may support an inference of causation as long as it ‘amounts to a reasonable likelihood, rather than a mere possibility.’” Roy v. Dackman, 219 Md. App. 452, 477, 101 A.3d 448, 463 (2014) cert. granted, 441 Md. 217, 107 A.3d 1141 (2015); see Ranes v. Adams Labs., Inc., 778 N.W.2d 677, 688 (Iowa 2010) (“A lack of absolute certainty goes to the weight of the expert’s testimony, not to its admissibility.”). In fact, it is well established that “[w]hen no scientific basis exists for

conclusively identifying causation between the plaintiff's medical condition and the alleged wrong, medical experts recognize certain protocols to permit an opinion on causation to be expressed in terms of a reasonable medical certainty." Ranes, 778 N.W.2d at 691.

Here, the nature of the injury, i.e., exposure to lead, prevents Dr. Lidsky from stating with "absolute certainty" that lead caused Traecina's neurocognitive deficiencies.¹² Lidsky Dep. 26:6-11 ("If you ask me does lead have a signature injury . . . the answer is no, it does not. In other words, you cannot look at the neuropsychological test results and say, 'Lead did this.'"); see Ranes, 778 N.W.2d at 691 (quoting 4 Jack B. Weinstein & Margaret A. Berger, Weinstein's Federal Evidence § 702.06(c)(i), at 702–127 (Joseph M. McLaughlin ed., 2d ed. 2009) (noting "[p]roblems can often arise in showing reliability of causation testimony in toxic-tort cases because of the 'uncertainties concerning the mechanisms by which medical conditions develop from [exposure to a toxic substance] and the difficulties of ruling out other potential causes of those conditions'"); Jones v. NL Indus., 2006 WL 5157750, at *5 (N.D. Miss. May 24, 2006) (recognizing "the fact that lead toxicology is itself very much an inexact science").

Nevertheless, the record indicates that Dr. Lidsky is sufficiently experienced, trained, and educated to render expert testimony on the effects lead has upon the brain and whether exposure to lead, based upon a patient's medical history, is capable of causing brain damage. Dr. Lidsky has authored at least six published, peer-reviewed articles regarding the effects lead has upon the

¹² "Rarely is any particular toxic agent the exclusive source of a given disease. Insidious diseases generally have several sources, each of which may by itself be sufficient to bring about the condition . . . [G]iven current limits on our knowledge of the etiology of insidious diseases and given the generality of statistical data, it is impossible to pinpoint the actual source of the disease afflicting any specific member of the exposed population." Rosenberg, The Causal Connection in Mass Exposure Cases: "A Public Law" Vision of the Tort System, 97 Harv. L. Rev. 856–857.

brain.¹³ Furthermore, his deposition testimony evidences his scientific understanding of how lead affects brain function.¹⁴ See In re Ephedra Products Liab. Litig., 393 F. Supp. 2d 181, 189

¹³ See Schneider, J.S. et al., Enriched environment during development is protective against lead-induced neurotoxicity, 896 Brain Res., 48, 49-55 (2001) (using an animal model of lead poisoning to examine the extent to which different environmental milieus may modify the effects of lead on the developing brain); Lidsky, T.I., Schneider, J.S., Lead Neurotoxicity in Children: Basic Mechanisms and Clinical Correlates, 127 Brain., 5-19 (2003) (discussing the current state of knowledge concerning the effects of lead on the cognitive development of children and specifically addressing the reasons for the child's exquisite sensitivity, the behavioral effects of lead, how these effects are best measured, and the long-term outlook for the poisoned child); Lidsky, TI, Schneider, J.S., Lead and Public Health: Review of Recent Findings, Reevaluation of Clinical Risks, Journal of Environmental Monitoring, 6 J. of Environ. Monit. 36-42 (2004); Schneider J.S., Anderson D.W., Wade T.V., Smith M.G., Leibrandt P., Zuck L., Lidsky, T.I. Inhibition of progenitor cell proliferation in the dentate gyrus of rats following postweaning lead exposure, 26 Neurotoxicology, 141-145 (2005) (examining the effects of postnatal lead exposure on progenitor cell proliferation in the hippocampus); Lidsky T.I., Schneider J.S., Autism and Autistic Symptoms Associated with Childhood Lead Poisoning, 5 J. of Applied Research In Clinical and Experimental Therapeutics, 80-87 (2005) (presenting two case histories of children who, during periods of severe lead poisoning, developed autism or autistic symptoms); Lidsky T.I., Schneider J.S., Adverse Effects of Childhood Lead Poisoning: The Clinical Neuropsychological Perspective, 100(2) Environ. Res., 284-93 (examining individual lead-poisoned children from the perspective of the clinical neuropsychologist).

¹⁴ His testimony provides, in relevant part:

“Q. By what mechanism does lead affect the cognitive development of performance?”

“A. Lead affects the brain in two ways: It binds to sulfhydryl groups and carboxyl groups and structural functions of proteins. It changes the function. It also mimics calcium, and probably zinc, and in all the processes that calcium is involved, lead will substitute It comes in very low levels. For example, there are a class of chemicals within neurons called [sic] second messengers. Once the synaptic transmission between one neuron and another one, it activates a process in the brain that's mediated by . . . the second messenger. And that does all the processes of a brain cell; its viability, its development, its ability to signal, its ability to store information. . . . The effects of lead on those chemical systems occurs at levels of a thousand to a million times lower than 10 macrometers per deciliter, and they disrupt those systems. So it affects synaptic transmission; it affects the viability of the brain cells; it affects the ability to store information; it affects the development; it also has effects on mitochondria, which in mild

(S.D.N.Y. 2005) (holding “analogy, inference and extrapolation can be sufficiently reliable steps to warrant admissibility so long as the gaps between the steps are not too great”). “While the literature may be equivocal about the prevalence of specific types of problems, [Dr. Lidsky] has a reliable basis to discuss the known association between lead and certain neurocognitive impairments.” Palmer v. Asarco Inc., 510 F. Supp. 2d 519, 530 (N.D. Okla. 2007); see generally State v. Lead Indus., Ass’n, Inc., 951 A.2d 428, 437 (R.I. 2008) (“There seems to be little public debate that exposure to lead can have a wide range of effects on a child’s development and behavior.”). Furthermore, although Dr. Lidsky’s analysis involved a “differential diagnosis[.]” such a methodology does not undermine his testimony. See McCulloch v. H. B. Fuller Co., 61 F.3d 1038, 1044 (2d Cir. 1995) (“Disputes as to [a medical expert’s] use of differential etiology as a methodology . . . go to the weight, not the admissibility, of his testimony”); Ranes, 778 N.W.2d at 693 (holding “[m]any courts . . . have found expert opinions based in part on case

effect lowers the metabolism of brain cells. In more extreme effects, it causes something . . . called cell suicide, where the cell, basically . . . dies, it kills itself. And also at high levels it will have effects on the blood-brain barrier, breaking it down. So that’s synopsis. It does more, but just to give you a picture.” Id. at 120:9-122:4.

...

“Q. I know we covered this, but again, because it gets so technical, when the lead enters the brain, it does so through the blood, and then it effects [sic] different areas differently?

“A. Yes, it does. The areas that are developing are much more vulnerable to the effects of lead. And different parts of the brain develop at different times. One of the reasons why executive functioning in aspects of memory are particularly vulnerable to the effects of lead is those systems develop for a very long time. . . . It’s during periods of development . . . when these systems of development are vulnerable. So that’s why executive functioning systems and fine memory systems are particularly vulnerable to the effects of lead.” Id. at 130:3-131:5.

studies and reports and in part by other data and individual research to be reliable”); Bonner, 259 F.3d at 929 (observing that “[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”) (internal citations omitted); see also Steve Gold, Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence, 96 Yale L.J. 376, 380 (1986) (observing that “[t]oxic tort litigation . . . involves inferences on causation derived from group-based information, rather than specific conclusions regarding causation in the individual case”). Accordingly, this Court finds that the reasoning or methodology underlying Dr. Lidsky’s testimony is scientifically valid. See Canada By & Through Landy v. McCarthy, 567 N.W.2d 496, 502 (Minn. 1997) (permitting a neuropsychologist to testify at trial that the Plaintiff “was likely exposed to lead during” a “critical time” of development).

ii

Relevance or Fit

This Court’s “Daubert responsibilities, however, do not end with reliability, because the trial court’s gatekeeping function also requires it to judge whether an expert’s testimony is ‘relevant to the task at hand’ in that it logically advances a material aspect of the proposing party’s case.” Terry v. Caputo, 875 N.E.2d 72, 78 (2007). “Thus, even if an expert’s proposed testimony constitutes scientific knowledge, his or her testimony will be excluded if it is not scientific knowledge for purposes of the case.” In re Paoli R.R. Yard PCB Litig., 35 F.3d 717, 743 (3d Cir. 1994); see Terry, 875 N.E.2d at 78 (noting “[r]eliability and relevance are not mutually exclusive findings, and they may overlap in some instances”). “[I]n a toxic tort case, expert testimony on the issue of *general causation* meets Daubert’s ‘fit’ requirement only if the testimony includes an opinion that (1) exposure to the particular substance at issue, (2) in the

dose to which the plaintiff was exposed, (3) for the duration in which plaintiff was exposed, (4) can cause the particular condition(s) of which the plaintiff complains.” Amorgianos, 137 F. Supp. 2d at 163 (emphasis added).

Turning to the first factor, Dr. Lidsky—after reviewing Traecina’s medical history—was able to say that “she had a significant level of lead at a peak time of vulnerability.” Id. at 49:11-

14. Second, as to a dose that would be harmful, his testimony provides:

“Q. Would you expect a 15-month old child with a blood-lead level of 25 to have significant impairments as a result of the exposure to lead at that age?

“A. In my experience, it’s more likely than not.” Id. at 137:18-25.

However, Dr. Lidsky could not testify to an exact dose effect.¹⁵ Nonetheless, “it is not always necessary for a plaintiff to quantify exposure levels precisely or use the dose-response relationship, provided that whatever methods an expert uses to establish causation are generally accepted in the scientific community.” Parker v. Mobil Oil Corp., 857 N.E.2d 1114, 1121 (2006); see Westberry v. Gislaved Gummi AB, 178 F.3d 257, 264 (4th Cir. 1999) (holding that “while precise information concerning the exposure necessary to cause specific harm to humans and exact details pertaining to the plaintiff’s exposure are beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial

¹⁵ Regarding a dose effect, Dr. Lidsky’s testimony provides:

“Q. Is there a dose effect?

“A. I’m not sure what that means.

“Q. Would you expect a dose effect if environmental and genetic factors were controlled for?

“A. I’m not sure what they mean. Unless they mean dose response, and that’s been shown with the IQ literature.” Lidsky Dep. 129:8-130:15.

exposure and need not invariably provide the basis for an expert's opinion on causation"). As such, this Court "do[es] not require a mathematically precise table equating levels of exposure with levels of harm, but there must be evidence from which a reasonable person could conclude that a defendant's emission has probably caused a particular plaintiff the kind of harm of which he or she complains." Wright v. Willamette Indus., Inc., 91 F.3d 1105, 1107 (8th Cir. 1996). Here, Dr. Lidsky's testimony, which is based upon his peer reviewed publications, as well as his medical knowledge, demonstrates that a dose of lead, above 25 mcg/dl, can cause the kind of harm of which the Plaintiff complains.

Third, regarding the duration of the Plaintiff's exposure, Dr. Lidsky testified that lead stays in the blood stream for "30 to 35 days." Lidsky Dep. 124:22-23. Moreover, he stated that "what you see in the blood is basically a snapshot of exposures that are relatively recent." Id. at 152:18-20. Accordingly, such testimony provides a link between Traecina's alleged exposure to lead at the Defendant's property and her elevated BLLs on July 13th, 17th, and 22nd in 1998. See Carlson v. Okerstrom, 675 N.W.2d 89, 106-07 (2004) (holding that "[w]hen a patient develops symptoms after encountering an agent which is known to be capable of causing those symptoms, courts have been more willing to admit expert testimony relying on the temporal connection between exposure and the onset of symptoms."); Arias, 928 F. Supp. 2d at 21 (finding that "[i]n compelling circumstances, a temporal relationship between exposure to a toxin and a plaintiff's injury alone is sufficient to establish general causation"). However, he acknowledged that there is "no scientifically supported way to parcel out what percentage of her injuries were caused by blood-lead levels in '97 versus the blood-lead levels in '98[.]" Id. at 161:19-24. As such, he could only state, to "a reasonable degree of certainty" that "Traecina ha[d] cognitive deficits as a result of *all* of her elevated blood-levels[.]" Id. at 161:6-7 (emphasis added).

Finally, as to whether such exposure can cause the particular condition of which the Plaintiff complains, Dr. Lidsky testified that lead does not have a “signature injury.” Lidsky Dep. 26:6-11. Put another way, he could not simply “look at the neuropsychological test results and say ‘Lead did this.’” Id. at 26:9-11. Rather, his analysis involved performing a “differential diagnosis” in which he looked to the patient’s medical history in order to determine if there were any other factors which could cause such neurocognitive injuries. In this instance, “[he] found no biologically-neuropsychologically factors, other than the fact that she had been poisoned with a substantial level of lead at a time of peak vulnerability.” Id. at 37:24-38:6. This absence of other factors, coupled with the fact that Traecina’s cognitive impairments were similar to those seen in other patients who had been exposed to lead during their childhood, led Dr. Lidsky to conclude that “brain damage from lead underlies the [Plaintiff’s] neuropsychological impairments.”¹⁶ Lidsky Report 5. Here, Dr. Lidsky uses a “scientific analysis known as differential etiology [or differential diagnosis] (which requires listing possible causes, then eliminating all causes but one); and reference to various scientific and medical treatises.” McCulloch, 61 F.3d at 1044. As such, “[d]isputes as to the strength of his credentials, faults in his use of differential etiology as a methodology, or lack of textual authority for his opinion, go to the weight, not the admissibility, of his testimony.” Id.; see Campbell v. Metropolitan Prop. and Cas. Ins. Co., 239 F.3d 179, 186 (2d Cir. 2001) (concluding that “gaps or inconsistencies in the reasoning leading to [the] opinion . . . go to the weight of the evidence, not to its admissibility”). As to the Defendant’s concerns regarding Dr. Lidsky’s methodology,

¹⁶ Dr. Lidsky testified:

“Q. But you haven’t seen anybody with a blood-lead level of that amount and not have a brain injury?”

“A. I don’t believe so.” Lidsky Dep. 99:7-10.

“[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence.” Daubert, 509 U.S. at 596. In sum, Dr. Lidsky is qualified, under Rule 702, to testify as to general causation, *i.e.*, that exposure to lead—in the levels observed in Traecina’s bloodstream—can cause the injuries alleged by the Plaintiff.

2

Proximate Causation

More generally, Defendant argues that he is entitled to summary judgment because neither Dr. Lidsky nor Dr. Besunder can state that lead poisoning, from the Defendant’s property, was the proximate cause of Traecina’s alleged cognitive deficiencies. Here, Plaintiff cannot survive the Defendant’s motion “for summary judgment on h[er] toxic tort claim unless [she] is able to point to evidence suggesting a probability, rather than a mere possibility, that (1) [s]he was exposed to the specified chemical at a level that could have caused h[er] physical condition (general causation); and (2) the exposure to that chemical did in fact result in the condition (specific causation).” Blanchard, 30 A.3d at 1274 (citing Golden v. CH2M Hill Hanford Group, Inc., 528 F.3d 681, 683 (9th Cir. 2008)). Indeed, “proof of causation must be such as to suggest ‘probability’ rather than mere ‘possibility,’ precisely to guard against raw speculation by the fact-finder.” Sakaria v. Trans World Airlines, 8 F.3d 164, 172–73 (4th Cir. 1993); see Vassallo v. Am. Coding & Marking Ink. Co., 784 A.2d 734, 740 (App. Div. 2001) (“On a motion for summary judgment in a toxic-tort case, the narrow issue is whether reasonable jurors could infer, based on the expert testimony, a nexus between plaintiff’s exposure to the offending product and her condition.”).

All cognizable negligence claims in Rhode Island must set forth four essential elements: duty, breach, causation, and damages. Santana v. Rainbow Cleaners, 969 A.2d 653, 658 (R.I. 2009). “With regard to causation, ‘[a] plaintiff must not only prove that a defendant is the cause-in-fact of an injury, but also must prove that a defendant proximately caused the injury.’” Sweredoski v. Alfa Laval, Inc., 2013 WL 3010419 (R.I. Super. June 13, 2013) (Gibney, P.J.) (quoting Almonte v. Kurl, 46 A.3d 1, 18 (R.I. 2012) and Lead Indus. Ass’n, Inc., 951 A.2d at 451). Indeed, “[t]he word ‘proximate,’ in the legal context of ‘proximate cause,’ requires a factual finding that the harm would not have occurred *but for* the [act] and that the harm [was a] natural and probable consequence of the [act].” Pierce v. Prov. Retirement Bd., 15 A.3d 957, 964 (R.I. 2011) (third alteration in original) (internal quotation marks omitted) (emphasis added).

“In the toxic-tort field[, however,] the modern trend has been to relax or broaden the standard of determining medical causation. This is because, in the toxic-tort context, ‘proof that a defendant’s conduct caused a [plaintiff’s] injuries is more subtle and sophisticated than proof in cases concerned with more traditional torts.’” Vassallo, 784 A.2d at 739 (quoting Landrigan v. Celotex Corp., 605 A.2d 1079, 1084 (1992)); see Lawrence G. Cetrulo, 1 Toxic Torts Litigation Guide § 5:3 (2014) (“While use of the ‘but for’ test has become the standard test of causation in non-toxic tort cases, *where there are potentially multiple, concurrent causes of a plaintiff’s injury, application of the ‘but for’ formula may allow each actor responsible for the plaintiff’s injuries to escape liability.*”) (Emphasis added). “Because the application of traditional causation principles, such as the ‘but for’ test, create a substantial barrier to recovery in toxic tort cases, a minority of jurisdictions have adopted the ‘frequency, regularity, and proximity’ test used in Lohrmann v. Pittsburgh Corning Corp.[, 782 F.2d 1156 (4th Cir. 1986).]” 1 Toxic Torts Litigation Guide § 5:3.

Here, this Court has previously recognized and applied the Lohrmann “frequency, regularity, and proximity” test within the context of an asbestos exposure case. See Sweredoski, 2013 WL 3010419, at *5 (“This Court . . . will apply the ‘frequency, regularity, proximity’ test as the proper causation standard for asbestos cases in Rhode Island.”). In Lohrmann, the Fourth Circuit upheld the trial court’s use of what has come to be known as the “frequency, regularity, and proximity” test. As set forth by the Fourth Circuit, in order “[t]o support a reasonable inference of substantial causation from circumstantial evidence, there must be evidence of exposure to a specific product on a regular basis over some extended period of time in proximity to where the plaintiff actually worked.” Lohrmann, 782 F.2d at 1162-63. The Fourth Circuit found that the trial judge’s “use of the ‘frequency, regularity and proximity test’ was appropriate in determining whether the inferences raised by the testimony were within the range of reasonable probability so as to connect a defendant’s product to the plaintiff’s disease process.” Id.

This Court finds that the “frequency, regularity, and proximity” test “comports with our state’s proximate causation jurisprudence because a plaintiff may satisfy the test by presenting expert testimony ‘show[ing] that the result most probably came from the cause alleged.’” Sweredoski, 2013 WL 3010419, at *5 (quoting Almonte, 46 A.3d at 18); see Cartier v. State, 420 A.2d 843, 848 (R.I. 1980) (“Causation is proved by inference.”). As our Supreme Court noted in Seide v. State:

“[a]lthough proximate cause may not be established by conjecture or speculation, proximate cause can be established by circumstantial evidence, and specific direct evidence of proximate cause is not always necessary. When inference is employed to establish causation, proof by inference need not exclude every other possible cause[;] rather[,] it must be based on reasonable inferences drawn from the facts in evidence.” 875 A.2d 1259, 1268-69 (R.I. 2005) (internal citations and quotations omitted).

Thus, “under the test, a plaintiff ‘*need not exclude every other possible cause*’ of his or her injury and need only present evidence sufficient to base a finding of causation ‘on reasonable inferences drawn from the facts’” Id. (Quoting Gianquitti v. Atwood Med. Associates, Ltd., 973 A.2d 580, 592-93 (R.I. 2009) (emphasis added)). Therefore, this Court shall apply the “frequency, regularity, and proximity” test as the proper causation standard for lead poisoning cases in Rhode Island. Accord Green v. Alparma, Inc., 373 Ark. 378, 284 S.W.3d 29 (2008) (concluding that a plaintiff in a toxic-tort case must prove the following elements: (1) plaintiff was exposed to toxic product spread by defendants; (2) with sufficient frequency and regularity; (3) in proximity to where plaintiff actually worked, lived, or went to school; (4) such that it is probable that exposure to the product caused plaintiff’s injuries”); James v. Bessemer Processing Co., 155 N.J. 279, 304, 714 A.2d 898, 911 (1998) (holding “that a plaintiff in [a] . . . , toxic-tort case may demonstrate medical causation by establishing: (1) factual proof of the plaintiff’s frequent, regular and proximate exposure to a defendant’s products; and (2) medical and/or scientific proof of a nexus between the exposure and the plaintiff’s condition”).

As discussed above, in order “[t]o prove causation in a toxic tort case, the plaintiff must show general and specific causation.” Arias, 928 F. Supp. 2d at 5 aff’d, 752 F.3d 1011 (D.C. Cir. 2014). “[G]eneral causation addresses whether a substance is capable of causing a particular injury or condition in a population, while specific causation addresses whether a substance caused a particular individual’s injury.” Blanchard, 30 A.3d at 1275 (quoting King, 762 N.W.2d at 34). “Both causation inquiries involve scientific assessments that must be established through the testimony of a medical expert. Without this testimony, ‘a plaintiff’s toxic tort claim will fail.’” Pluck, 640 F.3d at 677 (quoting Baker v. Chevron USA, Inc., 680 F.

Supp. 2d 865, 874 (S.D. Ohio 2010) aff'd sub nom. Baker v. Chevron U.S.A. Inc., 533 F. App'x 509 (6th Cir. 2013)).

Here, Plaintiff relies upon three sources of evidence to avoid summary judgment. First, she offers her own testimony, and that of her mother, Wendy, regarding their various places of residence. Second, Plaintiff relies upon her medical history, including her recorded BLLs over time as well as the Rhode Island Department of Health Environmental Lead Inspection Report. Third, Plaintiff relies upon the testimony of her two experts, Dr. Lidsky and Dr. Besunder. As detailed above, Dr. Lidsky is qualified to testify as to general causation, i.e., that exposure to lead is capable of causing the injuries of which the Plaintiff complains. Dr. Besunder, a board certified pediatrician with specialties in “pediatric critical care and pediatric pharmacology and toxicology[.]” seeks to testify that Traecina’s exposure to lead caused her cognitive deficiencies. Besunder Dep. 7:14-16.¹⁷

Defendant argues that neither of the Plaintiff’s experts can say, with any probability or degree of certainty, that exposure to lead at 71 Magill Street, if such exposure even occurred, was the proximate cause of the Plaintiff’s injuries. “In the context of proximate cause, the Rhode Island Supreme Court has stated that ‘[e]xpert testimony, if it is to have any evidentiary value, must state with some degree of certainty that a given state of affairs is the result of a given cause.’” Wallace v. United States, 335 F. Supp. 2d 252, 262 (D.R.I. 2004) (quoting Gray v. Stillman White Co., 522 A.2d 737, 741 (R.I. 1987)). Furthermore, “[i]t is well settled in this state that when expert medical testimony is offered to establish a causal relationship between a

¹⁷ This Court finds, and the Defendant does not argue to the contrary, that Dr. Besunder is qualified under Rule 702 to opine as to the cause of Traecina’s injuries. Dr. Besunder is employed by Akron Children’s Hospital, where he is the “director of the division of pediatric critical care medicine, the medical director of the pediatric care unit, the medical director of respiratory therapy, [the] attending physician in the division of pharmacology and toxicology, [and] a senior consultant to the lead clinic[.]” Besunder Dep. 6:21-7:4.

defendant's act or omission and the plaintiff's injury, such testimony must speak in terms of 'probabilities' rather than 'possibilities.'" Gray, 522 A.2d at 741 (quoting Parrillo v. F.W. Woolworth Co., 518 A.2d 354, 355-56 (R.I. 1986)).

Here, Dr. Besunder could not "say that the injuries complained of [by the Plaintiff] were more likely than not caused by the alleged ingestion of lead dust while [she] liv[ed] at 71 Magill Street[,]" Besunder Dep. 108:9-13, because "[he] [could not] divide her neuropsychological deficits by exposure." Id. at 104:8-11. Rather, his "opinion [was] that lead poisoning ha[d] been a significant contributor to her deficits." Id. at 63:9-18. Moreover, he was able to state that "within a reasonable degree of medical certainty that [Traecina's] lead level increased . . . due to exposure at 71 Magill Street[,]" id. at 106:22 and "lead was a significant contributing factor in her behavioral problems." Id. at 94:19-23. Dr. Besunder's testimony, that exposure to lead was "a significant contributor to [Traecina's] deficit[,]" was not equivocal in nature. Wallace, 335 F. Supp. 2d at 262. "While lead exposure may not have been the sole cause of [Traecina's] [neurocognitive deficiencies], [Dr. Besunder's] testimony states with a 'degree of positiveness,' that lead exposure was a contributing cause of [her] problems. The weight of this testimony must be left to the fact-finder." Wallace, 335 F. Supp. 2d at 262 (quoting Sweet v. Hemingway Transp., Inc., 114 R.I. 348, 354, 333 A.2d 411, 415 (1975)); see Wright, 91 F.3d at 1107 (concluding that a "mathematically precise table equating levels of exposure with levels of harm" is not necessary; rather there "must be evidence from which a reasonable person could conclude that" the defendant caused the particular harm that the plaintiff complained of); Parker, 857 N.E.2d at 1121 (holding that it is not always necessary for a plaintiff to quantify exposure levels precisely . . . , provided that whatever methods an expert uses to establish causation are generally accepted in the scientific community"). As such, Dr. Besunder's testimony, for the

purposes of summary judgment, establishes both specific and general causation. See Pluck, 640 F.3d at 676-77 (“In a toxic-tort case the plaintiff must establish both general and specific causation through proof that the toxic substance is capable of causing, and did cause, the plaintiff’s alleged injury.”).

More generally, looking at the evidence submitted in the light most favorable to the nonmoving party, this Court is satisfied that the Plaintiff has met the “frequency, regularity, and proximity” causation standard. Sweredoski, 2013 WL 3010419, at *5; see Vassallo, 784 A.2d at 739 (“At least for summary judgment purposes, where there has been exposure to multiple products over an extended period of time, . . . [p]laintiff must “prove ‘an exposure of sufficient frequency, with a regularity of contact, and with the product in close proximity’ to the plaintiff.”). Under the first prong, the Plaintiff, via her medical records, has shown that she was exposed to lead throughout her life, and, more particularly, while she resided at the Defendant’s property. See Dr. Besunder’s Report 5 (BLL Table). As to the second prong, there is sufficient evidence that Traecina was exposed to lead with sufficient “frequency and regularity.” Here, her BLL readings suggest that she was exposed to a significant dose of lead sometime in either late June or early July of 1998. See Lidsky Dep. 124:22-23 (“[W]hat you see in the blood is basically a snapshot of exposures that are relatively recent.”). Looking to the third prong, this Court finds there is evidence that Traecina was exposed to lead in the proximity of the Defendant’s property. Specifically, Dr. Besunder indicated that it would be unlikely that she would be exposed to an outside source, but rather that the timing of her symptoms demonstrated that “her lead level increased . . . due to exposure at 71 Magill Street.” Besunder Dep. 104:19-22; 106:22-107:1. In addition, the Department of Health, which inspected the property only days after Traecina was diagnosed with lead poisoning, determined that lead hazards were present at

the property. See Rhode Island Dep't of Health Environ. Lead Inspection Report. Lastly, regarding the fourth prong, Dr. Besunder unequivocally testified that “exposure to lead [was] a significant contributing factor to her deficits.” Besunder Dep. 109:14-22.

In sum, the Plaintiff has put forth sufficient evidence to deflect the scythe of summary judgment with regard to the “frequency, regularity, and proximity” causation standard. For the purposes of summary judgment, she has made a sufficient showing that (1) she was exposed to lead; (2) with sufficient frequency and regularity; (3) in proximity to 71 Magill Street; (4) such that it is probable that the exposure to the lead at the Defendant’s property caused her injury. As such, this Court denies the Defendant’s summary judgment motion.

B

Motions In Limine

1

Motion in Limine Regarding the Indivisibility of Plaintiff’s Injuries

Plaintiff argues that her brain injuries, which she claims result from exposure to multiple sources of lead, are indivisible. Moreover, Plaintiff contends that it is scientifically impossible to attribute any portion of her injury to any particular exposure. As such, the Plaintiff—relying upon the First Circuit decision of McInnis v. A.M.F., Inc., 765 F.2d 240 (1st Cir. 1985)—asks this Court to adopt the so-called “indivisible injury” rule. Plaintiff posits that the “indivisible injury” rule shifts the burden of proving that a harm is capable of being separated to each defendant. As such, Plaintiff contends that if a defendant cannot prove that the injury is divisible, joint and several liability should be imposed and the plaintiff should be entitled to recover all of his or her damages from any or all defendants.

In response, Defendant argues that the “indivisible injury” rule, under which an alleged tortfeasor may be held one hundred percent liable for an injury caused by many sources, is not

good law in Rhode Island. Rather, Defendant posits, that under Rhode Island law, a tortfeasor is only liable for the injury caused by his or her negligence. Furthermore, citing our Supreme Court's decision of Wilson v. Krasnoff, 560 A.2d 335 (1989), Defendant argues that he is not a "joint tortfeasor" under G.L. 1956 § 10-6-2.

This Court begins its analysis with a review of the relevant law in Rhode Island regarding joint tortfeasors. Pursuant to § 10-6-2, "the term 'joint tortfeasors' means two (2) or more persons jointly or severally liable in tort for the same injury to person or property, whether or not judgment has been recovered against all or some of them[.]" G.L. 1956 § 10-6-2. In Wilson, our Supreme Court discerned that there are two requirements in order for parties to be joint tortfeasors. Wilson, 560 A.2d at 339.

"First, the parties must be 'liable in tort.' The phrase 'liable in tort' has been construed to mean to have negligently contributed to another's injury. Zarella v. Miller, 100 R.I. at 548, 217 A.2d at 675. Second, the statute refers to the same injury. The same injury is caused by parties who engage in common wrongs. To constitute joint tortfeasors under the act, both parties must have engaged in common wrongs." Id.

Furthermore, "[i]n determining whether an occurrence between two or more parties is a common wrong," the Court found that "two important factors will be the time at which each party acted or failed to act and *whether a party had the ability to guard against the negligence of the other.*" Id. at 340 (emphasis added).

Here, the Plaintiff cannot show that the Defendant and the unnamed third parties engaged in common wrongs because none of the parties "had the ability to guard against the negligence of the other." Id. at 340. Quite simply, the separate landlords, who may have allowed lead paint to remain in the apartments they rented, acted separately and had no way of knowing of or controlling the actions of the other landlords. Accordingly, because the Defendant is not a "joint

tortfeasor” under our Supreme Court’s current interpretation of § 10-6-2, this Court declines to adopt the “indivisible injury” rule.

However, this Court’s analysis does not end in holding that the Defendant is not a joint tortfeasor. Even though the Defendant is not a “joint tortfeasor[,]” the Plaintiff may still establish a *prima facie* case of negligence. Specifically, regarding proximate causation, this Court notes that a “proximate cause ‘need not be the sole and only cause. It need not be the last or latter cause. It’s a proximate cause if it concurs and unites with some other cause which, acting at the same time, produces the injury of which complaint is made.’” Pierce, 15 A.3d at 966 (quoting Hueston v. Narragansett Tennis Club, Inc., 502 A.2d 827, 830 (R.I. 1986)). Moreover, in making out a claim, a plaintiff “‘need not exclude every other possible cause’ of . . . her injury and need only present evidence sufficient to base a finding of causation ‘on reasonable inferences drawn from the facts’” Gianquitti, 973 A.2d at 593 (quoting Seide, 875 A.2d at 1268-69) (quotation marks omitted).

As described supra—in light of the challenges presented in proving proximate causation in such a toxic tort case—this Court has departed from the “but for” standard and adopted the “frequency, regularity, and proximity” test. Under this test, a Plaintiff can prove proximate causation if she can present evidence that the “frequency, regularity, and proximity” of her exposure to lead at the Defendant’s property caused her injuries. Sweredoski, 2013 WL 3010419, at *8. She need not exclude every other possible cause. Gianquitti, 973 A.2d at 593. Accordingly, although the Defendant is not a joint tortfeasor, the Plaintiff may still establish a *prima facie* case of negligence.

Motion *in Limine* to Exclude or in the Alternative to Limit the Testimony of Arlene Weiss

Plaintiff seeks to exclude the testimony of the Defendant's expert witness, Arlene Weiss (Ms. Weiss). Ms. Weiss is a certified toxicologist with a master's degree in environmental health from New York University. See Weiss Dep. 15:3-9, Feb. 25, 2014. Ms. Weiss wrote a report, on behalf of the Defendant, setting forth her opinion regarding pertinent toxicological issues in the case. As such, Ms. Weiss's report concludes, to a high degree of toxicological, hygienic, and epidemiological certainty, that:

"A. The residence located [at] 71 Magill Street, Pawtucket, RI was not a contributory source of lead exposure for Traecina Claiborne.

"B. Traecina exhibited elevated blood lead levels prior to her tenancy at 71 Magill Street[,] Pawtucket, RI.

"C. Traecina exhibited a normal FEP value on 7/21/96 which suggests that exposure was recent and not over the past three months (April-May-June 1996) during her tenancy at 71 Magill Street.

"D. With respect to Traecina, her elevated BLLs were most likely the result of a summer outdoor exposure. Her history of fluctuations mimicked the seasonal variations normally found in the peers-reviewed published scientific literature." Weiss Report 9.

Here, the issue is whether this Court should exclude Ms. Weiss's expert testimony. This question turns on whether Ms. Weiss's opinion was expressed with sufficient credibility or validity to assist the fact-finder. See Raimbeault, 772 A.2d at 1061 ("The critical inquiry for deciding whether to admit expert testimony is whether the expert testimony reflects scientific knowledge that can be tested by scientific experimentation and whether the expert testimony logically advances a material aspect of the plaintiff's [or defendant's] case."). Although Ms. Weiss does not use the term "causation," her conclusions go directly to whether lead at the

Defendant's property caused Traecina's injuries. Accordingly, her testimony "must speak in terms of 'probabilities' rather than 'possibilities.'" Sweet, 114 R.I. at 355, 333 A.2d at 415.

Here, Ms. Weiss's testimony, as to what may or may not have caused Traecina's elevated BLLs, is so equivocal that it fails to provide the essential evidentiary basis on which proximate causation can be properly assessed. In essence, Ms. Weiss is expected to testify that lead is "ubiquitous" and thus Traecina's lead exposure can be explained by exposure to lead in the environment around her house. See Weiss Dep. 76:11-14 ("It just goes to my argument that lead is ubiquitous, it's everywhere and is found in a lot of different places."). However, as is illustrated below, this Court's careful review of Ms. Weiss's deposition testimony indicates that her conclusions are speculative, equivocal, and/or entirely contradicted by her own testimony.

First, Dr. Weiss concludes that "71 Magill Street, Pawtucket, RI was not a contributory source of lead exposure for Traecina Claiborne." Weiss Report 9. However, Dr. Weiss admits that "there was an enormous amount of lead paint hazards in [the] apartment[.]" Weiss Dep. 73:2-6, and that the Rhode Island Health Department lead inspectors found a "number of lead paint hazards . . . in every room[.]" Id. at 93:8-13. As such, she concedes that she cannot state to any degree of scientific certainty that Traecina was not exposed to lead at 71 Magill Street. Her testimony reads, in pertinent part:

"Q. Okay. And you're not saying to a reasonable degree of scientific certainty that the plaintiff was not exposed to the lead hazards set forth in the July 22nd inspection report, are you?"

"A. No, I'm not saying that. I am saying, I don't know the specific source of her elevated blood lead level and I do know she came to the residence with prior exposure to lead."

"Q. Okay. But you're excluding the lead paint hazards in each of the rooms?"

“A. *No, I’m not. I’m not excluding any hazards anywhere.*” Id. at 74:8-20 (emphasis added).

Here, Ms. Weiss directly contradicts her stated conclusion—that the Defendant’s property was not a contributory source of lead exposure—and can only speculate that “any of the sources are possible.” Id. at 77:1-2.

Second, in her report, Dr. Weiss states, “Traecina exhibited a normal [free erythrocyte protoporphyrin] (FEP) value on 7/21/96 which suggests that exposure was recent and not over the past three months (April-May-June 1996) during her tenancy at 71 Magill Street.”¹⁸ Weiss Report 9. Essentially, Dr. Weiss’s testimony is that if Traecina were exposed to lead in April, May, or June, her FEP level should have been elevated. Since her FEP level was not elevated, she “guess[es] she didn’t have [the exposure in] March, April, or May. [Rather,] something in the summer caused [the exposure].” Id. at 78:6-12. However, she agreed that “a high lead level and a normal FEP level is evidence of acute poisoning[,]” id. at 79:2-5, and that “it’s normal for an FEP result to lag behind elevated blood lead” levels. Id. at 79:21-24.

Finally, Ms. Weiss concludes that “[w]ith respect to Traecina, her elevated BLLs were most likely the result of a summer outdoor exposure.” Weiss Report 9. However, Ms. Weiss’s deposition testimony indicates that she is only capable of generally stating that other sources of lead exist. As such, Ms. Weiss broadly lists a number of “external environmental sources of lead” such as “smelters, auto repair shops, and specific industrial practices” as “potential sources” of exposure. Weiss Dep. 69:3-12. However, she admits that “there’s no evidence of

¹⁸ This Court notes that the Center for Disease Control (CDC) has stated that “erythrocyte protoporphyrin (EP) is not a sensitive test to identify children with blood lead levels below about 25 µg/dL, and therefore it is no longer the screening test of choice.” William L. Roper et al., Preventing Lead Poisoning in Young Children, Ctrs. Disease Control, Oct. 1, 1991, available at <http://www.cdc.gov/nceh/lead/publications/books/plpyc/contents.htm>. As such, the CDC recommends that “measurement of blood lead levels should replace the EP test as the primary screening method.” Id.

any of these sources in this case[.]” Id. at 14-16. Moreover, when asked if there “[was] any evidence in this case that Tra[ecina] was exposed to . . . [alternative sources,]” she replied, “[s]pecifically in this case, no. It just goes to my argument that lead is ubiquitous, it’s everywhere and is found in a lot of different places.” Id. at 76:8-15. As to her theory that Traecina’s elevated BLLs were caused by a “summer exposure[.]” she states, “[t]hat would be my guess, that it was a summer exposure in July.” Id. at 102:8-9. She further speculates that “[m]aybe it was a park she went to[.]” Id. at 102:14-15. However, when asked whether she recalled the “testimony about . . . Tra[ecina] visiting a park that summer[.]” she states that “[she] do[esn’t] remember that.” Id. at 103:5. Furthermore, while it is her opinion that Traecina’s elevated BLLs “may be explained by a summertime outdoor exposure[.]” she admits that “[she] doesn’t know the exact source” and that “any of the sources are possible.” Id. at 76:19-77:2.

In sum, this Court finds Ms. Weiss’s testimony to be equivocal at best. She “guess[es]” that Traecina’s BLLs were caused by a “summer time exposure” and states that “any sources [of exposure] are possible.” Id. at 78:10-12; 77:1-2. Morra v. Harrop, 791 A.2d 472, 477 (R.I. 2002) (quoting Montuori v. Narragansett Elec. Co., 418 A.2d 5, 10 (R.I. 1980)) (“The expert’s opinion . . . must have ‘substantial probative value’ and *not be speculation, mere conjecture or surmise.*”) (Emphasis added). Ms. Weiss’s testimony, that it is “possible” that Traecina was exposed to lead, in some dose, somewhere, does not rise to the level of “probability.” See Gray, 522 A.2d at 741 (excluding expert’s testimony because the court found that “[t]he words ‘likely’ and ‘consistent with’ do not connote ‘probability’”); Skene v. Beland, 824 A.2d 489, 492 (R.I. 2003) (disallowing expert testimony that was too speculative and lacked evidentiary support); Evans v. Liguori, 118 R.I. 389, 397-98, 374 A.2d 774, 778 (1977) (finding testimony that decedent’s symptoms are “consistent with” suicidal intent “merely begs” question of causal

relationship). As such, her proffered testimony about whether 71 Magill Street was not a contributory source and that Traecina may have been exposed to other sources of lead is mere speculation. See Rodriguez v. Kennedy, 706 A.2d 922, 924 (R.I. 1998) (precluding expert from testifying because he had not conducted the necessary tests and thus his testimony was mere speculation); see generally DiPetrillo v. Dow Chem. Co., 729 A.2d 677, 688 (R.I. 1999) (recognizing that “[e]xpert evidence can be both powerful and quite misleading . . .”). Accordingly, this Court excludes all of Ms. Weiss’s testimony, finding it to be too speculative to provide the essential evidentiary basis on which proximate causation can be properly assessed ¹⁹

3

**Motion *in Limine* to Exclude any Mention
or Evidence of any Alleged Domestic Violence or Abuse**

Based upon the Defendant’s line of questioning at the depositions of Wendy Claiborne, Dr. Besunder, and Dr. Lidsky, Plaintiff anticipates that the Defendant will attempt to mention or introduce evidence of alleged domestic altercations between Wendy Claiborne and Traecina’s father, Lenneth O’Neill (Mr. O’Neill). At the hearing on May 27, 2015, counsel for the Defendant indicated that “domestic violence . . . is just another one of those environmental factors in her upbringing, and it . . . goes to the credibility.” Hr’g Tr. 72:17-19, May 27, 2015. Plaintiff admits that discovery revealed some police involvement during disputes between Wendy Claiborne and Mr. O’Neill. However, Plaintiff points out, there is no record of Traecina

¹⁹ Ms. Weiss also concludes that “Traecina exhibited elevated blood lead levels prior to her tenancy at 71 Magill Street[,] Pawtucket, RI.” Weiss Report 9. While true, such a statement is not contested by either the Plaintiff or her experts. As such, her testimony is not helpful to the trier of fact. See Daubert, 509 U.S. at 591 (“Expert testimony which does not relate to any issue in the case is not relevant and, ergo, non-helpful.”); Raimbeault, 772 A.2d at 1061 (quoting DiPetrillo, 729 A.2d at 686) (“scientific expert evidence is admissible only if it is ‘relevant, appropriate, and of assistance to the jury.’”).

being abused or neglected. Furthermore, Plaintiff posits that there is no indication that Traecina witnessed the alleged altercations between her mother and father.

Rule 401 of the Rhode Island Rules of Evidence defines “relevant evidence” as “evidence having any tendency to make the existence of any fact that is of consequence to the determination of the action more probable or less probable than it would be without the evidence.” R.I. R. Evid. 401 (Rule 401). As such, R.I. R. Evid. 402 (Rule 402) provides that:

“[a]ll relevant evidence is admissible, except as otherwise provided by the Constitution of the United States, by the constitution of Rhode Island, by act of congress, by the general laws of Rhode Island, by these rules, or by other rules applicable in the courts of this state. Evidence which is not relevant is not admissible.” Rule 402 (emphasis added).

However, R.I. R. Evid. 403 (Rule 403) limits this broad standard and states that “[a]lthough relevant, evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence.” Rule 403.

While this Court is cognizant that “[t]he intertwined proof of breach of duty, causation, biological consequence and damage a toxic tort case requires, moreover, justifies a commensurately wide scope of argument, explanation and comment by counsel[.]” the Court has not been presented with any evidence that Traecina was abused or that such abuse could cause the brain damage she alleges to have suffered. Elam v. Alcolac, Inc., 765 S.W.2d 42, 212 (Mo. Ct. App. 1988). In fact, Dr. Lidsky testified that neuropsychological deficits are generally not “due to poor parenting skills; lack of emotional stimulation; poverty; neglect; [and] inconsistent or absent parents . . . except for extraordinarily extreme cases[.]” i.e., “where the parents are locked up for child abuse” or “worse than that.” Id. at 95:8-96:5. Dr. Lidsky testified that “certainly nothing in this case” indicates such abuse. Id. at 96:11-13. Furthermore, he

acknowledged that “some of the [social] factors . . . could affect test performance on some tests[,]” but overall would “not cause a pattern of results on neuropsychological testing consistent with brain injury[.]” Id. at 96:19-97:3.

At this time, prior to trial, this Court finds that any evidence regarding alleged abuse or domestic disputes between Traecina’s parents is not relevant to the case at bar and hereby precludes all testimony thereof. This Court reserves the right to reconsider such a ruling if additional evidence comes to light showing that Traecina was abused *and* such abuse is shown, through expert testimony, to cause the injuries complained of by the Plaintiff. See Cook, 782 A.2d at 654 (“The trial justice can reconsider the motion *in limine* during the trial or in rebuttal.”).

4

Motion *in Limine* to Exclude Evidence of Lenneth O’Neill’s Criminal Record

During Mr. O’Neill’s deposition, Defendant asked questions regarding his criminal record and background. As such, Plaintiff anticipates that Defendant will attempt to introduce evidence or otherwise mention Mr. O’Neill’s criminal history. Plaintiff seeks to preclude the introduction of such evidence. During the hearing, counsel for the Defendant indicated that he “fully expect[s] to subpoena [Mr. O’Neill][,]” but acknowledged that “if [Mr. O’Neill] doesn’t testify, then I don’t know how we get his criminal record in So that would be moot if he’s not available.” Hr’g Tr. 72:13-16.

Our Supreme Court has stated that “a trial justice’s discretion to exclude evidence under Rule 403 must be used sparingly. . . . It is only when evidence is marginally relevant and enormously prejudicial that a trial justice must exclude it.” State v. DeJesus, 947 A.2d 873, 883 (R.I. 2008). Here, the Court finds that whether Traecina’s father, Mr. O’Neill, has a criminal

history does not bear on the issue of whether Traecina was exposed to lead or that her injuries were caused by such exposure. See Miranda, 2009 WL 3515196, at *4 (holding “it is beyond this Court’s imagination why the [plaintiff’s] father’s traffic citation . . . h[as] the slightest relevance to [plaintiff’s] cognitive abilities or a neuropsychological evaluation[.]”); Bridges v. Enter. Products Co., 2007 WL 571074, at *4 (S.D. Miss., Feb. 20, 2007) (finding that “whether the subject individuals have criminal histories does not bear on the issue of whether they have suffered a loss of companionship”). As such, this Court shall preclude the Defendant from making reference or introducing evidence of Mr. O’Neill’s criminal history.²⁰

5

Motion *in Limine* to Exclude any Mention of Evidence of the Mental or Physical Condition of Traecina’s Siblings and Parents

Plaintiff seeks to preclude the Defendant from introducing testimony regarding the mental or physical condition of Traecina’s siblings and parents. Ostensibly, Defendant seeks to discover and present information about Traecina’s parents and siblings in order to show that some genetic disorder or environmental factor caused the injuries she is alleged to have suffered. However, this Court finds that any claim by the Defendant that there is a “correlation” between the Plaintiff’s impairments and her siblings’ or parents’ mental or physical condition, is “speculative at best.” Nieves ex rel. Cleare v. 1845 7th Ave. Realty Assocs., L.P., 710 N.Y.S.2d 782, 786 (Sup. Ct. 2000) (finding “any claim . . . that there is a correlation between the infant plaintiff’s impairments and his siblings’ academic performance, is speculative, at best”). The record before this Court contains no indication that Traecina’s siblings and parents share her cognitive impairments. As such, this Court shall preclude all mention of the Traecina’s siblings’

²⁰ This Court notes that if Mr. O’Neill is called to testify, the Defendant may be permitted to impeach his testimony pursuant to R.I. R. Evid. 609.

and parents' physical or mental condition. See Miranda, 2009 WL 3515196, at *4 (finding that "details about [plaintiff's] parents' own education, . . . and their relationship with their own children [were] not sufficiently relevant to the instant case to outweigh the strongly prejudicial effect on the jury").²¹

6

Motion *in Limine* to Exclude the Testimony of Paul Chervin, M.D.

Plaintiff argues that Dr. Chervin's opinions should be excluded because they do not "fit" the facts of the case as they do not assist the trier of fact in determining whether or not Traecina suffered injuries as a result of her exposure to lead as a child. More specifically, Plaintiff contends that while Traecina suffered lead levels that were sufficient to cause neuropsychological damage, such damage was not so severe as to be capable of being detected by Dr. Chervin's neurological assessment.

Dr. Chervin, the Defendant's expert witness, is a neurologist with a clinical practice in Woburn, Massachusetts. Chervin Dep. 5:12-13, Feb. 26, 2014. Dr. Chervin reviewed the Plaintiff's medical history as well as the reports and deposition testimony of the other expert witnesses in the case. Ultimately, Dr. Chervin, despite acknowledging that "Traecina had elevated blood lead levels during her second [and] third years of life[.]" opined that "he d[id] not find objective evidence that she manifested any neurological dysfunction from her elevated lead

²¹ This Court notes that "the granting of a motion *in limine* need not be taken as a final determination of the admissibility of the evidence." State v. Fernandes, 526 A.2d 495, 500 (R.I. 1987). The Defendant is currently entitled to present evidence that the Plaintiff's condition is the result of a genetic disorder or some environmental factor other than lead. However, the Court notes that at this time, Defendant has not submitted any credible evidence that would suggest that the mental or physical condition of the Plaintiff's parents or siblings would support such a theory. Nevertheless, this Court may "reconsider the motion *in limine* during the trial or in rebuttal." Cook, 782 A.2d at 654.

levels of childhood.” Pl.’s Mot. *in Limine* to Exclude the Testimony of Dr. Paul Chervin, Ex. A (Chervin Report).

In support of this conclusion, Dr. Chervin makes two principal arguments. First, he argues that lead did not cause Traecina’s neurological dysfunction because, as he states, “all of us who existed in the 1940’s, 50’s, 60’s and earlier lived with a lot more lead and are not all mentally retarded and unable to compete in the real world.” Chervin Dep. 21:22-22:2. Essentially, Dr. Chervin’s opinion is that lead could not have caused Traecina’s injuries because he was likely exposed to lead growing up and he has not suffered any brain injuries.²² However, when asked if he could “point to . . . any peer-reviewed medical literature that supports that proposition” he stated that he could not, but rather such a conclusion was “common sense.” *Id.* at 25:3-6. Furthermore, he could not state whether his blood lead levels were ever checked. *Id.* at 25:7-9.

Dr. Chervin’s second argument is that he arrived at his conclusion “in the same way that one would construct . . . a differential diagnosis[.]” *Id.* at 25:20-22. However, Dr. Chervin provides no alternative explanation for Traecina’s neuropsychological deficiencies, nor does he refute the scientific literature that indicates that lead, in the levels that Traecina was exposed to,

²² The colloquy between Plaintiff’s counsel and Dr. Chervin, regarding his opinion, reads in pertinent part:

“Q. Okay, I . . . asked you the basis of your opinion, and basically it sounds to me like your opinion is based on your personal experience, your personal life, correct? *You listed a number of ways you likely were lead poisoned and you’re doing pretty good ‘cause you’re a neurologist. I mean, that seems to be the gist of your opinion and the basis of your opinion; is that correct?*

“A. Well that’s the personal response, the subjective response to your question, extrapolating that out to the individuals who were alive at the same time I was a child.” *Id.* at 24:14-25:2 (emphasis added).

is known to cause neuropsychological deficiencies.²³ Instead, he lists a number of “negative factors” including family stress and a chaotic home life that may have impacted Traecina’s upbringing, id. at 28:5-29:8, and a series of symptoms—encephalopathy, seizures, and peripheral neuropathy—which he admits only occur as a result of blood lead levels in excess of what was measured in Traecina’s bloodstream. Id. 34:2-35:6.

In fact, Dr. Chervin’s deposition testimony directly contradicts his own conclusion. He acknowledges Traecina’s impairments “include complex visual motor integration, word reading, math computation, and nonverbal extraction” and “that those neuropsychological impairments have been related to elevated blood lead levels in the medical literature.” Id. at 29:10-23. Furthermore, he admits that Traecina’s lead poisoning “may have” contributed to her neuropsychological deficits and her learning disabilities. Id. at 36:18-24; 39:21-40:2; 41:11-14.

As this Court previously explained, Rule 702 requires that expert testimony be both reliable and relevant. See Knight, 482 F.3d at 352 (quoting Daubert, 509 U.S. at 592-93) (“Reliability is determined by assessing ‘whether the reasoning or methodology underlying the testimony is scientifically valid.’”). Furthermore, in order to testify as to causation, an expert must speak in terms of “probabilities” rather than “possibilities.” Gray, 522 A.2d at 741 (quoting

²³ Dr. Chervin’s deposition testimony reads, in pertinent part:

“Q. Okay. Now, you’ll agree with me that children with lead levels that went up as high as Tra[ecina’s] and documented 51 venous that lead can cause, based on the medical literature, neuropsychological problems, Correct?”

“A. That’s correct.

“Q. And learning disabilities?”

“A. That is correct, yes. Again, based on the literature, yes.” Id. at 38:17-39:1

Parrillo, 518 A.2d at 355-56). Here, Dr. Chervin does not specialize in treating lead poisoned children, he has not done any research on lead poisoning, nor has he written any articles on lead poisoning.²⁴ This lack of experience was reflected in his testimony. See DiPetrillo, 729 A.2d at 688 (asserting that the trial court must scrutinize the reliability of an expert witness’s underlying principles and methodology due to the danger that the expert will confuse or mislead the jury). The fact that Dr. Chervin obtained his Ph.D. after allegedly being exposed to some level of lead as a child is in no way relevant to whether Traecina was injured as a result of exposure to lead. See Marks v. Zoning Bd. of Review, 102 R.I. 545, 551, 232 A.2d 382, 385 (1967) (finding an expert witness opinion to be a “mere assertion completely lacking in probative force” where it was “unsupported by factual data to which such opinion could be related”). Furthermore, he essentially concedes that (1) the scientific literature has shown that exposure to lead, in the levels Traecina was exposed to, has been shown to cause neuropsychological impairments; and (2) that encephalopathy, seizures, and peripheral neuropathy would only manifest themselves if the patient was exposed to lead levels in excess of those to which Traecina was exposed. Id. at 35:7-

²⁴ Dr. Chervin’s testimony provides, in relevant part:

Q. And under your “Research” heading on the last page [of your CV], and I know you have testified in the past . . . , but you’ve done no research on lead poisoning, correct?

A. That’s correct.

Q. Or written any medical articles on lead poisoning?

A. That’s correct.

“ . . .

Q. In your practice, you don’t specialize in treating lead poisoned children, do you?

A. No.

36:3; see Miranda, 2009 WL 3515196, at *4 (finding expert's "theories on lead poisoning general causation to be so significantly outside the mainstream of medical acceptance and completely lacking factual basis, that allowing her to present her opinion would serve only to confuse the jury"); see N.B.S., Inc. v. Harvey, 709 A.2d 162, 166 (Md. App. 1998) (questioning and ultimately excluding the testimony of the expert witness because the expert was "unable to point to a single medical doctor currently practicing medicine or involved in such research who would agree with her view of the effects of lead poisoning"). Accordingly, this Court shall preclude Dr. Chervin from testifying because the Court finds that his methodology is neither reliable nor scientific, does not fit the facts of this case, and he cannot say with any certainty or probability that Traecina's neurological dysfunction was not caused by her elevated lead levels as a child.

V

Conclusion

For the aforementioned reasons, this Court finds that Dr. Lidsky is qualified, pursuant to Rule 702, to testify as to general causation. Furthermore, for the reasons described above, this Court denies the Defendant's summary judgment motion. Finally, this Court (1) denies the Plaintiff's motion *in limine* to adopt the "indivisible injury" rule; (2) grants the Plaintiff's motion *in limine* to exclude the testimony of Arlene Weiss; (3) grants the Plaintiff's motion *in limine* to exclude any mention or evidence of domestic violence or abuse; (4) grants the Plaintiff's motion *in limine* to exclude any mention or evidence of Lenneth O'Neill's criminal record; (5) grants the Plaintiff's motion *in limine* to exclude any mention or evidence of the mental or physical condition of Traecina's siblings or parents; and (6) grants the Plaintiff's motion *in limine* to exclude the testimony of Dr. Chervin. Counsel shall submit an appropriate Order for entry.



RHODE ISLAND SUPERIOR COURT

Decision Addendum Sheet

TITLE OF CASE: Wendy Claiborne, Individually and as Parent, Natural Guardian and Next-of-friend to her minor child, Traecina Claiborne v. Duncan Duff

CASE NO: PC 10-6330

COURT: Providence County Superior Court

DATE DECISION FILED: June 23, 2015

JUSTICE/MAGISTRATE: Presiding Justice Alice B. Gibney

ATTORNEYS:

For Plaintiff: Vincent L. Greene IV, Esq.; Jonathan D. Orent, Esq.; Robert J. McConnell, Esq.; Ashley J. Hornstein, Esq.

For Defendant: Anthony J. Gianfrancesco, Esq.; Albin S. Moser, Esq.