IN THE SUPERIOR COURT OF THE STATE OF DELAWARE

IN AND FOR NEW CASTLE COUNTY

ANAJAI CALCAÑO PALLANO, et al.,	)	
	)	
Plaintiffs,	)	
	)	
v.	)	C.A. No. N09C-11-021 JRJ
	)	
THE AES CORPORTATION, et al.,	)	
	)	
Defendants.	)	

ORDER

AND NOW TO WIT, this 11th day of December, 2015, the Court having heard and duly considered Plaintiffs' Motion to Exclude the Testimony of Defendants' Expert Samuel Moore, M.D.;[1] AES's Opposition;[2] and Plaintiffs' Reply;[3] **IT APPEARS THAT:**

1. It is undisputed that Minor Plaintiff Isael Altagracia Andujar suffers from Hirschsprungs disease, a congenitally acquired condition arising from the

[1] Plaintiffs' Motion to Exclude the Testimony of Defendants' Expert Samuel Moore, M.D. ("Pls.' Mot. Exclude Dr. Moore") (Trans. ID. 57346390). This Motion is one of nineteen *Daubert* Motions filed by the parties. Defendants have challenged seven of Plaintiffs' causation experts and four of Plaintiffs' exposure experts. Plaintiffs have challenged six of Defendants' causation experts and two of Defendants' exposure experts. The parties submitted twenty-six Joint *Daubert* Exhibits, which include each expert's report, deposition, and curriculum vitae ("J. Ex.") (Trans. ID. 57342400). *See* J.Ex. 20.A Dr. Samuel Moore Expert Report on Isael Altagracia Andujar ("Dr. Moore Expert Report").

[2] AES's Opposition to Plaintiffs' Motion to Exclude the Testimony of Dr. Samuel Moore at 12 ("Defs.' Opp'n.") (Trans. ID. 57536580).

[3] Plaintiffs' Reply in Further Support of its Motion to Preclude the Testimony of Defendants' Expert Samuel Moore, M.D. ("Pls.' Reply") (Trans. ID. 57627760).

abnormal development of the nerves of the gastrointestinal system.[4] For over thirty years, Defendants' expert Samuel Moore, M.D. ("Dr. Moore") has focused his research and clinical practice on Hirschsprungs disease. In addition to treating over 500 Hirschsprungs patients, Dr. Moore has researched, published, and presented extensively on the genetic causes of Hirschsprungs disease, including a peer-reviewed publication identifying the RET gene as a primary contributor to Hirschsprungs disease.[5]

2. According to Dr. Moore, "the widely accepted [ ] opinion on the etiology of Hirschsprungs disease [is] that it results chiefly from genetic changes resulting in the enteric nervous system maldevelopment and not environmental factors,"[6] and, "[a]lthough there is no specific single genetic site attributed to Hirschsprungs disease etiology, Hirschsprungs disease is widely recognised as resulting primarily from variations in the RET proto-oncogene (with some autosomal dominant

[4] *See Pallano, et al. v. AES Corp., et al.*, 2015 WL 7776612 (Del. Super. Nov. 24, 2015) (Opinion Denying Defendants' *Daubert* Motion to Exclude the Testimony of William P. Konicki). The Court incorporates by reference the facts, background, and the discussion of Delaware Rule of Evidence 702 and *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993), set forth in that opinion.

[5] Dr. Moore received his medical degree in 1971 from the University of Cape Town. Dr. Moore Expert Report at 1. He has published 134 publications, thirty-one abstracts, twenty textbook chapters, and authored two student books about paediatric surgery. *Id.* Fifty of Dr. Moore's publications directly address aspects of Hirschsprungs disease, and twenty-eight publications address the development of the enteric nervous system and related dysfunctional syndromes. *Id.* at 2. Dr. Moore has presented at 195 scientific meetings, fifty-one relating to Hirschsprungs disease and allied conditions. *Id.* In 2014, Dr. Moore received the Denis Browne Medal for an outstanding contribution to paediatric surgery from the British Association of Paediatric Surgeons. *Id.* at 1.

[6] *Id.* at 5.

inheritance)."[7] Dr. Moore also explains that there are also reports of susceptibility mutations in at least eleven other genes.[8] In describing the complex genetic etiology of Hirschsprungs disease, Dr. Moore explains that Hirschsprungs disease follows a "multigenetic" model—*i.e.* that Hirschsprungs disease usually results from the interaction of variants in several genes, rather than from a variant in a single gene.[9]

3. Dr. Moore opines that Plaintiff Isael's Hirschsprungs disease is most likely caused by a genetic variation, and not environmental factors.[10] Plaintiffs seek to exclude Dr. Moore's opinion under D.R.E 702 and *Daubert* arguing that: (1) he lacks the requisite qualifications to offer a causation opinion about genetics because he is a surgeon not a geneticist; (2) his opinion is not peer-reviewed, it is untestable, and was developed only for litigation; and (3) he did not rule out alternative causes such as the individual constituents of Coal Ash waste.

4. Dr. Moore's opinion is based on the causal links between Plaintiff Isael's individual genetic variations and Hirschsprungs disease, and the absence of any known study showing an association between Hirschsprungs disease and teratogenic exposure. Dr. Moore relies on genetic testing showing that Plaintiff Isael has at least three genetic variations: (1) a variation on the RET gene (exon

[7] *Id.*
[8] *Id.*
[9] *Id.* at 3.
[10] *Id.* at 3–5. ("[T]he evidence for a predominant genetic etiology (cause) for Isael's Hirschsprungs disease is overwhelming . . .").

20); (2) copy loss at chromosome 21; and (3) copy loss at chromosome 22.[11] According to Dr. Moore, each of these variations is located in a highly sensitive area close to critical genes involved in the development of the enteric nervous system.[12] Consistent with the multigenetic nature of Hirschsprungs disease, the identification of multiple genetic variations in Isael's genetic testing (each associated with chromosomal and/or congenital abnormalities) also strengthens the strong likelihood of a genetic cause.[13]

5. Plaintiffs argue that Dr. Moore's theory with respect to Plaintiff Isael's RET variation is not peer-reviewed and is untestable because a RET gene variation at the identical sub-genetic location (exon 20) has never been described in literature linking the variant with Hirschsprungs disease.[14] Isael's exon 20 variation is located in the last region ("tail") of the RET gene, the particular area of the RET gene where Dr. Moore has extensively focused his research.[15] Dr. Moore explains that this critical intracellular tyrosine kinase tail of the RET gene plays a critical role in the development of the enteric nervous system and it is common for Hirschsprungs patients with variations in the RET gene to have them in this

[11] Dr. Moore Expert Report at 2–3.
[12] *Id.* at 3.
[13] *Id.*
[14] Pls.' Mot. Exclude Dr. Moore at 14–15.
[15] Dr. Moore Expert Report at 2. Dr. Moore has focused his research on the "under researched" intracellular receptor tyrosine tail of the intracellular tyrosine kinase portion of the gene, which includes exon 20. *Id.*

specific area.[16] Accordingly, based on the position of the variation at the very tail of the RET gene, Dr. Moore concludes that Plaintiff Isael's "alteration in exon 20 must be considered as highly likely to play a part in the etiology of Isael's Hirschsprungs disease in conjunction with other contributing genetic defects" such as the multigenetic nature of Hirschsprungs disease.[17] Dr. Moore also cites three pieces of literature finding exon 20 variations in Hirschsprungs patients.[18] Finally, Dr. Moore notes that Isael's genome scan showed that he inherited the variation in exon 20 from his unaffected mother.[19] Relying on literature relating to familial transmission of Hirschsprungs disease, Dr. Moore states that, "[o]ne of the strongest arguments for the genetic origin of Hirschsprungs disease is the consistent recurrence in families,"[20] and "[t]he consistency of familial transmission of Hirschsprungs disease strongly supports the current genetic etiology for Isael's condition."[21]

6. With respect to Isael's copy number variations, Dr. Moore explains that the clinical association between Hirschsprungs disease and chromosome 21 and chromosome 22 has long been established.[22] For example, Down syndrome, which

[16] *Id.* at 3–4.
[17] *Id.* at 4.
[18] Defs.' Opp'n at 12–13; Dr. Moore Export Report at 9, 24–32.
[19] Dr. Moore Export Report at 9, 15.
[20] *Id.* at 15. Dr. Moore cited an article finding "180 times higher familial risk than the estimated 0.26% expected in the general population." *Id.*
[21] *Id.*
[22] *Id.* 9–12.

is caused by having an extra copy of chromosome 21, is the most common chromosomal anomaly associated with Hirschsprungs disease.[23] In support of this opinion, Dr. Moore cites several studies explaining the clinical association between these missing chromosomes and Hirschsprungs disease, including the importance of the location of chromosome 21 and chromosome 22 in connection with Hirschsprungs disease.[24]

8. Dr. Moore's opinion passes muster under D.R.E. 702 and *Daubert*. Based on Dr. Moore's research, experience, and extensive expertise related to Hirschsprungs disease, the Court finds Dr. Moore is qualified to opine about the genetic causes of Hirschsprungs disease. Dr. Moore's report thoroughly discusses the complex genetic etiology of Hirschsprungs disease, the functional significance of Isael's specific gene variations, and notes the absence of any known study showing an association between Hirschsprungs disease and teratogenic exposure (specifically any of the substances found in Coal Ash Waste). Dr. Moore's proffered testimony is based upon sufficient facts and data, is the product of reliable principles and methods, and he has applied the principles and methods reliably to the facts of this case. Dr. Moore articulates his thought process, evaluation methods, and conclusions.

WHEREFORE, because the Court finds that the opinions set forth in Dr.

[23] *Id.* at 9–11.
[24] *Id.* at 9–12.

Moore's Expert Report are both relevant and reliable, the Plaintiffs' Motion to Exclude is **DENIED.**

IT IS SO ORDERED.

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Jan R. Jurden, President Judge