Prepared by the Court

INFANT PLAINTIFFS A.B. O.B. AND J.B. By their f/n/g RICHARD BRACKEN, RICHARD BRACKEN, individually, ASHLEY BRACKEN, GEORGE ROSKO, FARRA ROSKO, et. al. Plaintiffs, Vs. PARAMOUNT HOMES AT GRANDVIEW AVENUE, LLC. THE MARKETING DIRECTORS, INC. et. al. Defendants	SUPERIOR COURT OF NEW JERSEY LAW DIVISION OCEAN COUNTY DOCKET No. OCN-L-3205-17 CIVIL ACTION OPINION OF THE COURT

This matter comes before the Court on motion of defendant Paramount Homes at Grandview Avenue, LLC for summary judgment and to bar the testimony of the plaintiff's proposed expert, Harpreet Pall, M.D. After considering the motions, the Court conducted a R. 104 hearing on March 31, 2022, to determine the issue of whether the opinions provided by Dr. Pall were net opinions under In Re Accutane, 191 A.3d 560 (N.J. 2018). After the court granted extensions of time for the parties to submit post hearing briefs all briefs were submitted on June 30, 2022. Having considered the testimony presented and the briefs submitted, the court now bars the testimony of Dr. Pall for violation of the required standards set forth in Evidence Rule 703 and In Re Accutane. Id.

In August 2014, the plaintiff, Farra Rosko, moved into her newly constructed home. The plaintiffs' homes were built by Paramount Homes, in the Whispering Meadows development in Jackson, New Jersey. Four months later, Mrs. Rosko became pregnant, and she gave birth to her daughter, T.R., in the fall of 2015. T.R. was diagnosed at two months of age with biliary atresia, a

serious condition affecting the bile ducts of the liver. Alleging that the Defendants negligently constructed their homes, which resulted in excessive exposure to toxins that caused the children to suffer injuries, the Roskos, along with the Brackens, filed suit on November 16, 2017. The Complaint included negligence for personal injuries Counts I, II, Breach of Contract III, Breach of Warranties IV, Common Law Fraud V, Consumer Fraud VI-VII, and Attorney's Fees VIII.

On August 26, 2020, Plaintiffs Bracken and Rosko submitted the Expert Report of GreenWorks Environmental, ("GreenWorks"). After conducting a Mold and Mycotoxin Investigation for the Plaintiffs' homes, GreenWorks noted that construction defects were causing intermittent and seasonal moisture intrusion into both the Bracken and Rosko homes. GreenWorks further noted indicators of elevated seasonal relative humidity and visible mold growth. Lab tests confirmed the presence of toxic mold, mV06, and volatile organic compounds (VOC). GreenWorks claims the presence of these toxins is a direct result of negligent construction practices in the construction of the plaintiffs' homes. The Defendants did not provide contrary evidence of the levels of mold, mycotoxins, or VOCs in either the Brackens' or Roskos' homes.

On September 8, 2021, Dr. Harpreet Pall, a Board-Certified Pediatric Gastroenterologist, provided the Rosko plaintiffs with his medical opinion concerning T.R.'s illness. Dr. Pall concluded that the harm experienced by the Rosko child's liver and the onset of biliary atresia was caused by the inability of the bile ducts to properly form during pregnancy. The supporting rationale for Dr. Pall's opinion is an article published in a peer-reviewed medical journal discussing the etiology of biliary atresia. Dr. Pall's methodology for his conclusion that the Rosko child's biliary atresia was substantially caused by her exposure to the environmental toxins present in the Rosko home while *in utero* lacks the necessary quantum of scientific certainty.

Plaintiffs filed their complaint in November 2017. Through the course of discovery numerous answers, counterclaims, and third-party complaints were filed. The number of parties to this suit has grown to approximately sixty. This Court ordered the matter to be managed as a track IV case in September 2018.

This action was assigned to an independent outside mediator after a Mediation notice was issued on November 13, 2019. Mediation was again ordered by the Court to occur in July 2021 with a report from the mediator by August 15th of that year. After the parties were permitted forty-seven months to conclude their discovery, the court terminated discovery on October 15, 2021.

Third-party defendant Woodhaven filed a motion for summary judgement on July 14, 2021, with opposition and cross-motions for summary judgement filed shortly thereafter. Between the date of filing for summary judgment and the court's consideration of the motion the plaintiffs' filed the September 8, 2021, report of Dr. Harpreet Pall, M.D. Oral argument on the issue of barring expert reports on the basis of net opinion were entertained on November 18, 2021. Following the oral argument, the parties returned to the Court appointed mediator and were provided ample time to settle the dispute. After the dispute persisted without any likelihood of resolution, this Court issued an opinion ordering a Rule 104 hearing on December 16, 2021, to consider the testimony of Dr. Harry A. Milman, Ph.D., Dr. Brain Gelbman, M.D., and Dr. Harpreet Pall, M.D. The Rule 104 hearing took place on March 31, 2022. This Court now issues its opinion on the Defendant's motion to bar the Rosko plaintiff's witness on causation, Dr. Harpreet Pall.

The immediate issue before this Court is whether to grant the defendant's motion to exclude the expert witness testimony of Dr. Harpreet Pall. The Supreme Court has offered guidance on the admissibility of expert scientific testimony, with the focus on the expert's methodology for arriving at their conclusion. See generally, In re Accutane Litigation, 191 A.3d

560 (N.J. 2018); Landrigan v. Celotex Corp., 605 A.2d 1079 (N.J. 1992); Rubanick v. Wiltco Chemical Corp., 593 A.2d 733 (N.J. 1991). The Court has determined that the expert report of Dr. Pall articulates a conclusion that is not drawn from a sound, scientific basis, but rather speculates about a causal link in which the available science offered by Dr. Pall is wholly uncertain. This Court therefore grants the defendant's motion to exclude Dr. Pall's testimony. Moreover, since Dr. Pall's opinions constitutes the only proposed evidence on the causal connection between the environmental toxins in the GreenWorks report and the Rosko child's biliary atresia, the plaintiff cannot, as a matter of law, satisfy their burden of proof in their toxic tort action against the defendants. However, because the defendant Woodhaven filed its summary judgment motion prior to the plaintiff's submission of Dr. Pall's report, the court will permit the defendants to submit dispositive motions in light of the courts determination in this opinion.

I

Rule 702 of the New Jersey Rules of Evidence states that

> [i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education may testify thereto in the form of an opinion or otherwise.

N.J.R.E. 702. Relating to the basis of the expert's opinion, Rule 703 goes on further:

> [t]he facts or data of the particular case upon which an expert bases an opinion or inference may be those perceived by or made known to the expert at or before the hearing. If of a type reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject, the facts or data need not be admissible in evidence.

N.J.R.E. 703. In interpreting the rules of evidence, the Supreme Court has acknowledged the role of trial courts as gatekeepers when reviewing the reliability of scientific expert testimony

in civil cases. See Accutane, 191 A.3d at 564. While that gatekeeping function does not require the expert to offer opinions that are generally accepted within the scientific community to be admissible, their opinions must be grounded in a sound methodology subjected to the standards of scientific norms, and not mere speculation and "unsubstantiated personal beliefs." Landrigan, 605 A.2d at 1084; see Accutane, 191 A.3d at 589 ("The trial court is the spigot that allows novel expert testimony in areas of evolving medical causation science, provided the proponent of the expert can demonstrate that the expert adheres to scientific norms in distinct ways that we have identified.").

The process by which a trial court examines the reliability of a witness must necessarily be rigorous, as the inquiry investigates the factual basis for the particular expert's opinion. However, while it is not the role of the trial court to "bless new 'inspired' scientific theory," the court may still permit expert testimony using a novel theory of causation if the expert can demonstrate that they conformed to reasoning accepted by those in their community. Id. at 593. To provide direction for trial courts' analysis of the reliability of an expert witness, the Supreme Court offered the guidelines considered in Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 593-94 (1993) as aids in the inquiry. Id. at 594-95. The Daubert factors are not an exhaustive list, nor is the list a checklist, nor is the full body of case law from Daubert incorporated into New Jersey law. See id. ("Like several other states, we find the factors useful, but hesitate to embrace the full body of Daubert case law as applied by state and federal courts."). After examining the methodology applied by the expert in reaching their opinion, it is up to the trial court to exercise its role as gatekeeper and decide whether the expert has a basis for the opinion that conforms to their community's standards. Accutane, 191 A.3d at 595. If not, the expert testimony' may be excluded on the grounds that it is unreliable.

In Daubert, the United States Supreme Court articulated factors to be considered when determining the reliability of scientific expert witness testimony. Among these considerations were whether the scientific knowledge or theory can be (or has been) tested, whether the theory has been subjected to peer review or publication, the potential rate of error for a particular scientific technique,[1] and whether the methodology has garnered general acceptance within its scientific community. Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 593-94 (1993). None, however, of these factors are determinative, and the failure to satisfy one of the factors is not fatal to the expert's proponent. See In re Accutane, 191 A.3d 560, 594 (N.J. 2018) (noting that the Daubert factors are "perhaps pertinent for consideration, but not dispositive or exhaustive…."). Further, on the last factor – whether a methodology has garnered general acceptance – the Court in Accutane suggested that cherry-picking evidence is a method that violates core scientific principles and may point to the testimony's classification as unreliable. See id. at 592-93.

The Appellate Division has already grappled with the Supreme Court's clarified guidelines post Accutane. In Lanzo v. Cyprus Amax Minerals Co., the Appellate Division reversed the trial court and barred the testimony of several experts because of the failure of the experts to satisfactorily ground their opinions that non-asbestiform fibers can cause mesothelioma in a sound scientific methodology. Lanzo v. Cyprus Amax Minerals Co., 254 A.3d 691, 711-12 (N.J. Super. Ct. App. Div. 2021). The studies relied on by the experts were each deficient in critical ways, such as the lack of discrimination between asbestiform and non-asbestiform fibers, or the presence of

[1] This factor is more suited for testimony in which the expert is testifying to the results of a test or analysis that are directly implicated in the case at hand. See Daubert, 509 U.S. at 594, (1993), (citing several prior cases where the scrutinized testimony was itself the results of contested scientific techniques). As such, a discussion of the "potential rate of error" factor is omitted from this opinion.

conclusory statements within the articles without further explanation for their basis. Id. at 710. Further, one of the experts admitted that he was not aware of any study that specifically looked at the non-asbestiform fibers at issue in Lanzo and a potential causal link to mesothelioma. Id. The appellate court reasoned that the lack of study data from peer-reviewed publications addressing the specific causal theory in the case and the absence of general acceptance of the experts' opinions within their scientific community required the trial court to exercise its gatekeeping power to prohibit such testimony from presentation before a jury. Id. at 711-12.

The GreenWorks report identified the presence of several VOCs, including 1, 2-dichloroethane, benzene, and ethylbenzene, along with low levels of formaldehyde within the Rosko home. The plaintiff's witness on the issue of causation, Dr. Pall, opined that T.R.'s exposure to those toxins *in utero* are a substantial cause of her biliary atresia. For Dr. Pall's testimony to be considered reliable, and therefore admissible, the plaintiff needs to demonstrate a sound methodology, rooted in the standards of Dr. Pall's profession, that served as the basis for his conclusion. More narrowly, the witness must have a legally adequate basis for connecting the exposure of the specifically identified toxins in the GreenWorks report, not environmental toxins in the abstract, as a cause of biliary atresia. Pure conjecture on behalf of the witness is insufficient, and no set of credentials – however impressive – may be substituted for the standard set forth by the law. To guide our analysis on the reliability of Dr. Pall's testimony, we will assess the expert's basis in light of the Daubert factors as far as they are applicable in this case.

As indicated by Astha Malik, in *Recent developments in etiology and disease modeling of biliary atresia: a narrative review.* (Cincinnati Children's Hospital Medical Center, 2020), Biliary Atresia (BA) is a severe hepatobiliary disease restricted to early infancy that results in fibroinflammatory destruction of the extra- and the intra-hepatic bile ducts, development of rapid

cholestasis, cirrhosis, and progression to hepatic failure. Although classified as a rare disease with a global incidence of 1:3,000 – 1:15,000, BA is the most common cause of obstructive cholestasis that accounts for approximately 50% of all pediatric liver transplantations in the United States. Astha Malik et al., *Recent Developments in Etiology and Disease Modeling of Biliary Atresia: A Narrative Review*, DIGESTIVE MED. RSCH., Dec. 30, 2020, at 1-2.

In reviewing expert opinions for scientific reliability, the first two Daubert factors; whether the scientific theory can be (or has been) tested, and whether the theory has been subjected to peer review or publication, run in conjunction in this case, so they will be analyzed together. In his letter, Dr. Pall hypothesizes that environmental toxins present in the Rosko home, specifically, 1,2-dichloroethane, ethylbenzene, benzene, and formaldehyde – while Mrs. Rosko was pregnant probably triggered T.R.'s biliary atresia. Dr. Pall Letter at 3. To a substantial degree, Dr. Pall relies on the Malik article published in a peer reviewed medical journal, Digestive Medicine Research (DMR). Id. at 4. While Dr. Pall concludes that, "it stands to reason that environmental exposure was a substantial factor in her developing biliary atresia," Dr. Pall never identifies what element of "environmental exposure" caused the biliary atresia. Dr. Pall indicates that he reviewed and agreed with the reports of Greenworks Environmental and Dr. Harry Milman, however, Dr. Pall never provides any analysis for how a particular exposure to any of the chemicals identified caused the plaintiff to develop biliary atresia.

The Malik article discusses various potential triggers for biliary atresia, such as genetic family history, viral infection, and immune disorders. Id. at 3-6. While the Malik article gives differing substantive weight to each of these theories, the authors are careful to avoid designating any of them as triggers for biliary atresia. See id. at 4-5.

> **While the exact cause of BA remains unknown**, the etiopathogenesis of syndromic or congenital BA is associated primarily with a variety of structural anomalies at birth… the underlying causality of the embryonic form was linked originally to developmental complications and potential contributions from genetic aberrations linked mostly to defects in early embryonic patterning.

Malik, *supra*, at 3. (Emphasis added).

> Despite these evidences, the direct relevance of these findings as "triggers" of Biliary Atresia Splenic Malformation (BASM) and syndromic BA still require validation analyses to firmly establish their functional roles… the triggers of non-syndromic or acquired BA, however, are hypothesized to involve perinatal exposures to noxious stimuli. The most widely accepted hypothesis of initial hepatobiliary injury in acquired BA involves potential viral infection in the early perinatal period, corroborated by extensive experimental evidence from rhesus rotavirus (RRV)-induced BA in newborn mice.

Id.

> However, none of these studies were able to conclusively link the hepatic viral signatures to development of BA and more importantly distinguish between primary and secondarily acquired infections … While [viral and other agents inducing biliary injury] remains speculative and largely unanswered due to paucity of disease-relevant models, identification of the causative factors of BA disease remains a topic of paramount importance … However, the hepatotoxic properties of MDI in mice and humans remain unknown. Id. at 4-5.

In fact, the Malik article goes to great lengths to state that the current level of data and analysis is insufficient to draw any firm conclusions on the disease's etiology. See id. Even during the discussion of environmental toxins as a cause of biliary atresia, the article speaks in generalities – newborns are highly sensitive to exposure to volatile organic compounds, a class of compounds. Id. at 5. The environmental toxins identified in the Malik research are biliatresone, a purified plant toxin, and two polyurethane derivatives, Diphenylmethane diisocyanate (MDI) and Methylenedianiline (MDA). While biliatresone, was first shown to cause spontaneous outbreaks of extrahepatic biliary disease resembling BA in Australian neonatal livestock, these incidences were associated with severe drought resulting in animals grazing on Dysphania glomulifera which were exposed on lands typically located under water. Malik indicates, "Although these findings lend credibility to the involvement of an exogenous toxin in BA and may explain the non-

mendelian inheritance, twin discordance and genetic abnormalities, human exposures to biliatresone are almost completely unlikely." Malik, *supra*, at 5.

Regarding exposure to polyurethane derivatives, the only study referred to by Malik was a case where rodents were injected with MDA which caused hepatic necroinflammation, fibrosis and portal inflammation. The article notes, "The hepatotoxic properties of MDI in mice and human remain unknown." Id. at 5. "Though animal models are an exceptional tool for investigating immunopathogenic underpinnings, differences in murine and human immune makeups may fail to recapitulate the phenotype of human diseases." Id. at 8. Significantly, the type of MDA or MDI exposure an infant may experience is not limited to construction materials found in residential homes. "Varying amounts of residual MDA and MDI have reportedly been found in diapers, transfer of azo dyes in colored prints and textiles due to wet diaper areas, plastic pacifiers, infant crib mattresses…" Id. at 5.

Dr. Pall claims in his September 8, 2021, report that Malik "describes the potential environmental triggers in the early prenatal period which are likely to play a substantial role in the pathogenesis of biliary atresia." Malik makes no such claim. No environmental triggers were identified by Malik as "likely" to play a substantial role in the development of biliary atresia.

Furthermore, Dr. Pall claims, "Collectively, these findings establish a probable role for toxins in the pathogenesis of biliary atresia." Dr. Pall Letter at 4. Dr. Pall stands alone in his opinion that human biliary atresia is caused by exposure to environmental toxins. While Malik identifies MDA and MDI as causing hepatic problems in laboratory rats and mice, no analysis was ever provided linking either MDA or MDI with biliary atresia in humans. In his final conclusion set forth in his September 8, 2021, report Dr. Pall indicates, "However, it is also clear from supporting

scientific evidence, such as that described in the Malik paper, that chemical toxins such as those identified in Talia's home are the probable environmental trigger for Talia's biliary atresia." Pall at 4.

Malik sets forth in his first sentence under the heading of "Triggers of BA: advances and mechanisms" the following declaration,

> While the exact cause of BA remains unknown, the etiopathogenesis of *syndromic* or *congenital* (also referred to as *"embryonic")* BA is associated primarily with a variety of structural anomalies at birth.

Malik, *supra*, at 3. (Emphasis provided).

> The most widely accepted hypothesis of initial hepatobiliary injury in acquired BA involves potential viral infection in the early perinatal period, corroborated by extensive experimental evidence from rhesus rotavirus (RRV) - induced BA in newborn mice.

Id. at 3.

> In rodents, administration of MDA causes hepatic necroinflammation and fibrosis and portal inflammation… However, the hepatotoxic properties of MDI in mice and human remain unknown.

Id. at 5. Malik reviews three separate insults as possible factors in the etiology of BA. The triggers of acquired BA are hypothesized to involve perinatal exposures to noxious stimuli. The first category of discussion is the impact of viral infection which has been corroborated by evidence from rhesus rotavirus (RRV) induced BA in newborn mice. Malik indicates that the most widely accepted hypothesis of acquired BA involves potential viral infection in the early perinatal period. A second study looked at a purified plant toxin, biliatresone, to induce BA-like cell injury and destruction of bile ducts in zebrafish and newborn mice. However, Malik notes that human exposures to biliatresone are almost completely unlikely. Finally, Malik reviews the impact of two chemicals, Diphenylmethane diisocyanate (MDI) and 4-4' Methylenedianiline (MDA). The article

indicates that the administration of MDA to rodents causes hepatic inflammation and fibrosis. The only reported case of MDA impacting the health of humans is a case study occurring fifty-seven years ago in the Epping district of Essex, England. In 1965 a jar of epoxy resin hardener spilled onto a sack of flour which was later used to bake bread. The active ingredient in the resin hardener was an MDA known as 4-4' Methylenedianiline. The eighty-four adults who contracted jaundice in the Epping District of Essex all ate a brown bread baked at the same bakery which used the contaminated flour. (The Epping Jaundice; H. Kopelman, *British Medical Journal*, 1966). The author explained that children typically did not eat this brown bread because it called for an acquired taste. While Dr. Pall points to the exposure of MDA as causing liver damage in humans, the exposure that Dr. Pall relies upon was an unwitting human consumption of MDA after a bakery produced loaves of bread using flour contaminated with 4-4' Methylenedianiline. The plaintiff has never ingested MDA, and the 1965 British study revealed that the eighty-four adults who consumed the contaminate bread contracted jaundice. No cases of biliary atresia in humans were ever reported in the study relied upon by Dr. Pall.

Malik notes, that the hepatotoxic properties of MDI on mice and human remain unknown. Nevertheless, Malik does indicate that, "Collectively these data establish a probable role for environmental and chemical toxins in the etiopathology of BA." Malik, *supra*, at 6. While little contest exists that BA like symptoms develop after direct injection of MDI into newborn mice, and that jaundice develops in humans after consumption of 4-4' Methylenedianiline, no data has been presented linking inhalation of the VOC's identified in the Rosko home, ethylbenzene, benzene, and 1,2-dichloroethane, to any development of acquired BA. Moreover, there is no mention in the Malik article of any of the chemicals (including formaldehyde) which Dr. Pall links to the plaintiff's BA. Despite this lacuna of evidence surrounding the issue of proximate cause

between the VOC's found in the Rosko home and the acquisition of biliary atresia, Dr. Pall declares that the toxins found in the plaintiff's home are the "probable environmental trigger for Talia's biliary atresia." No evidence has been presented that inhalation of any of the contaminants found in the Rosko home have ever been associated with biliary atresia. While the plaintiff claims to have avoided contracting any viruses during her pregnancy, similarly, there is no evidence that the plaintiff was ever exposed to any known contaminate identified by Malik, biliatresone, MDA or MDI, as being associated with biliary atresia.

Concluding the article on the etiology of biliary atresia, Malik looks to the collection of data then available and indicates:

> These **findings may be consequential** due to the (I) presence of residual chemicals in items that newborns are potentially exposed to and (II) plausible existence of a toxic compound in beets, chard and other consumable plaints that co-purifies with biliatresone. More importantly, the existence of multiple EHBD disease phenotypes, molecular representations of inflammatory and fibrosis subtypes at diagnosis, differential response to KHPE, short- and long-term survival with native liver, etc. all point to an involvement of more than a singular trigger of biliary pathogenesis **Further studies are warranted** to identify signatures of these chemicals and toxins or their derivatives or structurally similar entities using patient derived specimens.

Malik, *supra*, at 12-13. (Emphasis added). In his testimony at the Rule 104 hearing, Dr. Pall cites another article in a peer reviewed publication (Tatekawa article) that modeled biliary atresia in rats, induced post birth, through the injection of pure ethanol. See generally Yukihiro Tatekawa et al., *Intrahepatic Biliary Atresia with Pure Ethanol: An Experimental Model of Biliary Atresia*, 43 SURGERY TODAY 661 (2013). To test the hypothesis that ethanol injection into the bile ducts of rats could induce biliary injury, thirty-four rats were subjected to ethanol injection, while only thirteen were relegated to the control group, where they were injected with a saline solution.

Id. at 661-62. The experiment in Tatekawa was successful in inducing biliary injury in many of the rat specimens directly injected with ethanol, id. at 663, and perhaps in a case with a different set of facts the results of this experiment would be more potent. However, the lack of ethanol present as a potential environmental trigger in this case is a critical deficiency. No claim has been presented by the plaintiff that she suffered an exposure to ethanol which led to the development of BA.

The same is also true for another piece of literature cited by Dr. Pall, a study that exposed zebrafish larvae to the compound biliatresone (Lorent article). See generally Kristin Lorent et al., *Identification of a Plant Isoflavonoid that Causes Biliary Atresia*, SCI. TRANSLATIONAL MED., May 6, 2015. Once again, the article does not specifically examine the effects of fetal exposure on any of the toxins listed in the Greenworks report as a cause of baby Rosko's biliary disease, but instead establishes the effect of an unrelated compound, biliatresone, on the livers of zebrafish and Australian livestock.

> These data … lead us to conclude that biliatresone is highly likely to be the toxic cause of the BA syndrome in the Australian livestock. Whether exposure to biliatresone or a related environmental agent can be directly linked to human BA is unknown and understanding its relevance to this human disease will require further study.

Id. at 10-11. In sum, while Dr. Pall's causal theory is testable, none of the VOCs described in the Greenworks report appear to have been tested to date as potential causes of biliary atresia. In his Rule 104 hearing testimony, Dr. Pall attempts to explain the concept of biliary injury via environmental toxins through the methodology adopted by these latter two studies. However, the plaintiff has never alleged that she was exposed to the environmental toxins of biliatresone and ethanol, which have been demonstrated to cause the disease in animal studies. Dr. Pall's claim that

inhalation of structurally unrelated toxins which were not part of the medical studies, cause biliary atresia is unsupported by any study in the scientific or medical community. The Court is mindful of the distinction drawn by the Appellate Division in Lanzo between the different types of fibers as causes of mesothelioma. As set forth in *Accutane*, an expert's opinion on causation may be admitted when "based on a sound, adequately-founded scientific methodology involving data and information of the type reasonably relied on by experts in the scientific field." Accutane, 191 A.3d at 565 (quoting *Rubanick v. Witco Chem. Corp.*, 125 N.J. 421, 449, 593 A.2d 733 (1991)). In cases "involving novel theories of causation," a court must review the "data and studies relied on by experts proffering an opinion in order to 'determine whether the expert's opinion is derived from a sound and well-founded methodology that is supported by some expert consensus in the appropriate field.'" Id.

Here the Court has no difficulty accepting the methodology adopted by either Malik, Lorent, or Tatekawa. However, Dr. Pall conducted no studies of his own and proffers his opinion based upon the studies conducted others. Critically, none of the studies relied upon by Dr. Pall examine the effects of the toxins found with the plaintiff's home. Dr. Pall claims because the plaintiff alleges that she did not contract a virus during her pregnancy, the biliary atresia in her child must have developed from another source. Dr. Pall conjectures that because ingestion and injection of biliatresone, ethanol, and MDA have been found to cause liver damage in both rodents and humans, then 1,2-dichloroethane, ethylbenzene, benzene, and formaldehyde, must cause biliary atresia, because there is no other explanation for how the BA developed. Dr. Pall ignores the overwhelming consensus in the scientific community the declares, "The cause of biliary atresia in infants is unknown." There is no support in the scientific community that biliary atresia is caused

by inhalation of the substances found in the plaintiff's home. Dr. Pall does not cite to one authority that supports his claim.

In analyzing Dr. Pall's claim under the *Daubert* factors the Court finds:

1) That Dr. Pall's theory that inhalation of 1,2-dichloroethane, ethylbenzene, benzene, and/or formaldehyde, causes biliary atresia has never been tested by anyone in the scientific community.
2) Dr. Pall's theory that 1,2-dichloroethane, ethylbenzene, benzene, and/or formaldehyde, during pregnancy causes biliary atresia has never been peer reviewed through any publication.
3) Dr. Pall's theory is not subject to any potential rate of error because it has never been tested at any level by any scientific analysis.
4) Dr. Pall's theory has never been presented for analysis by any scientific committee for review of its methodology and analysis.

Here, the plaintiffs failed to establish that Dr. Pall's "methodology involved data and information of the type reasonably relied on by experts in the scientific field." Rubanick, 125 N.J. at 449. The methodology adopted by Dr. Pall is no more than a shell game. Dr. Pall claims that biliatresone, ethanol, and MDA are chemical compounds that have been shown to cause either liver damage or BA like symptoms. While 1,2-dichloroethane, ethylbenzene, benzene, and formaldehyde, are chemical compounds that have been identified as containing toxic properties, none of these chemicals have never been linked to biliary atresia. For some unexplained reason Dr. Pall claims that 1,2-dichloroethane, ethylbenzene, benzene, and formaldehyde were found in the plaintiffs' home, and therefore exposure to these chemicals must cause biliary atresia because the plaintiff claims that she did not contract a virus while she was pregnant. The conclusion of Dr.

Pall is without support in the scientific community. Dr. Pall's conclusions are no more that his "unsubstantiated personal beliefs" Kemp, 174 N.J. at 427.

To further examine whether Dr. Pall's theory has garnered general acceptance within the scientific community, Dr. Pall's theory must have gained general acceptance which includes the idea that the scope and strength of an expert's conclusions matches the same of its cited basis. Otherwise, an expert's conclusions drift into the realm of speculation. See Lanzo, 254 A.3d at 711-12 (noting the failure of the Roggli study to discriminate between asbestiform and non-asbestiform fibers was critical in determining whether the expert's causal theory could be admissible testimony).

The Malik article suggests multiple triggers of biliary atresia, including environmental toxins, but does not conclude with specificity which toxins may trigger the development of biliary atresia. See Malik, *supra*, at 13 (noting a likelihood of more than a singular trigger of biliary pathogenesis). However, Dr. Pall leaps in his own mind to conclude that the environmental toxins in the GreenWorks report were a substantial cause of T.R.'s biliary atresia. Dr. Pall presents as support for his theory the absence of the other accepted triggers – such as the lack of familial history of biliary atresia, and the lack of an identifiable viral infection claimed by Mrs. Rosko during the course of her pregnancy. Postulating on a causal connection merely based on a correlation of circumstances will not satisfy the methodological requirements for an expert witness's testimony to be admissible.

While Malik raises possible causes of BA, and notes that his findings may be consequential, he concludes that biliary atresia is caused by the involvement of more than a single trigger. The current state of science does not allow the medical community to identify the

"signatures of these and toxins or their derivatives or structurally similar entities." Malik calls for additional research using patient derived specimens rather than the animal studies using rodents or zebra fish which have been the basis of studies previously relied upon by others in his field. Malik is consistent with the entirety of the published medical literature on the etiology of biliary atresia. The plaintiff has supplied the peer reviewed articles relied upon by their experts which contain the following conclusions:

1) Tan and Moscoso: *Pathology International* 1994; 44: 600-610;

"The cause of inflammation and sclerosis in biliary atresia is as yet unknown."

2) Mysore: *Journal Pediatric Gastroenterol Nutr.* October 2019;

"A toxin –biliatresone– is responsible for causing BA-like disease in Australian sheep. One unresolved issue with maternally-ingested toxin such as biliatresone is that it would affect both fetuses in twin pregnancies, unlike BA which is discordant in twins. Finally, an in-utero onset adds to our understanding of **the elusive factors that cause bile duct injury before birth** and trigger what ultimately in the postnatal period is identified as BA." (Emphasis added).

3) *Cincinnati Children's Hospital:* February 2022;

"The causes of biliary atresia are not completely understood. For some children, biliary atresia may occur because the bile ducts did not form properly during pregnancy. For other children with biliary atresia, the bile ducts may be damaged by the body's immune system in response to a viral infection acquired after birth."

4) Shwarz and Haber et.al., *Hepatology,* November 2013;

"The etiology of biliary atresia (BA) is unknown."

5) Lorent and Gong, et. al., *Sci Transl Med.,* 2015 May 6;

"Whether exposure to biliatresone or a related environmental agent can be directly linked to human BA is unknown and understanding its relevance to this human disease will require further study."

6) Malik: *Dig. Me. Res.* 30 December 2020;

> "While the exact cause of BA remains unknown, the etiopathogenesis of syndromic or congenital (also referred to as "embryonic") BA is associated primarily with a variety of structural anomalies at birth."

Furthermore, the identified VOCs in the GreenWorks report – ethylbenzene, benzene, and 1,2-dichloroethane – are absent in both Dr. Pall's letter and the Malik article. There is a distinction between claiming that environmental toxins *as a class* may be *linked* to biliary atresia, and that the *specifically identified* VOCs may cause the disease. The Malik article does the former, and Dr. Pall's letter interprets Malik to do the latter. Compare Malik, *supra*, at 6 ("Collectively, these data establish a probable role for environmental toxins in the etiopathology of BA"); with Dr. Pall Letter at 3 ("However, it is also clear from supporting scientific evidence, such as that described in the Malik paper, that chemical toxins ***such as those identified in Talia's home*** are the probable environmental triggers.") (Emphasis added). While the Malik article raises additional possibilities for the causes of biliary atresia, Dr. Pall presents a degree of certainty in a singular cause that is not justified by his purported analysis.

Likewise, the same may be said of the Tatekawa and Lorent articles. Neither of the toxic compounds tested in those studies are alleged to have caused the Rosko child's biliary atresia, nor are there any claims that ethanol and biliatresone are structurally similar to the compounds at issue. In the Lorent article the authors repeatedly state that the etiology of biliary atresia is unknown. While Dr. Pall's statements about a proof of exposure to toxins cause liver damage, no study he identifies goes so far as to link the compounds found within the Rosko's home to a human contraction of biliary atresia. See Lorent, *supra*, at 9 *("***The etiology of BA is unknown**, although there is evidence implicating an environmental exposure – **either infectious or toxic** – in genetically susceptible individuals**.**") (Emphasis added); Dr. Pall Rule 104 Hr'g. 141:3-9 ("So, in

our particular baby here, baby [T.R.], what we have is a baby who **did not have any obvious genetic abnormalities, who did not have a family history of biliary atresia**, who did not have a known virus … and nor was there any evidence of immune dysregulation.") (Emphasis added); see also id. at 135:14-138:7 (responding to questions on the absence of studies that examine the causal nexus between compounds such as xylene, toluene, and ethylbenzene and biliary atresia). In short, the Dr. Pall concludes that since T.R. contracted biliary atresia, and the child did not have any obvious genetic abnormalities, and her mother did not contract a virus, and there is no history of biliary atresia, and no known immune dysregulation, the biliary atresia must have been caused by the mother's exposure to the chemicals in her home which were identified by the GreenWorks report.

This Court has determined that Dr. Pall's opinion which claims a causal relationship exists between the specific VOCs identified in the Rosko home and T.R.'s biliary atresia, is without the support of any conclusion on the relationship between such toxins and BA in the relevant scientific literature. While Dr. Pall refers to Dr. Milman's example that toluene has been linked to reproductive and environmental toxicity, toluene was not found to be present at elevated levels in the Rosko home. Three VOCs were identified in the Greenworks report as being present in the Rosko home at elevated levels; Benzene; 1,2 Dichloroethane, and Ethylbenzene. The plaintiff has provided to its experts and the Court the California Environmental Protection Agency Draft Hazard Identification of the Developmental and Reproductive Toxic Effects of Benzene. The study indicated,

> In some of these studies, but not all, maternal toxicity was reported to occur concurrent with exposures that produced adverse fetal effects. There is little indication that benzene causes structural malformations. There are no studies with postnatal endpoints. Dose dependance is seen and some benzene concentrations produce these effects in the absence of reported maternal toxicity. Human studies

> of pregnancy outcome from maternal exposure are characterized by limited exposure ascertainment, simultaneous exposure to multiple chemicals and low power. They neither support nor contradict the animal data.

Cal. EPA, *Draft Hazard Identification of the Developmental and Reproductive Toxic Effects of Benzene*, at 80 (1997). The plaintiff also provided the International Agency for Research on Cancer (IARC) Summaries and Evaluations for Benzene. The report indicated that Benzene was tested for carcinogenicity in mice and rats through oral ingestion, inhalation, and subcutaneous injection of benzene. The study concluded, "Chromosomal aberrations in human peripheral lymphocytes were associated with occupational exposure to benzene, although many of the studies are very difficult to interpret." IARC, *Summaries & Evaluations: Benzene*, at 2 (1987). No studies have been provided by the plaintiff or its experts, to suggest that exposure to benzene causes abnormal fetal development resulting in biliary atresia.

The plaintiff has produced for review the Environmental Protection Agency report on Ethylene Dichloride (1,2-Dichloroethane). The report indicates: "Reproductive / Development Effects: No information is available on the reproductive or developmental effects of ethylene dichloride in humans. Decreased fertility and increased embryo mortality have been observed in inhalation studies of rats." EPA, *Ethylene Dichloride (1,2-Dichloroethane)*, at 2 (2000). Neither the plaintiff nor its experts have provided any studies which suggest that human exposure in any form to Ethylene Dichloride causes abnormal fetal development resulting in biliary atresia.

Lastly, the plaintiff has supplied the Environment Protection Agency's report on Ethylbenzene. The report indicates: "Reproductive / Developmental Effects: No information is available on the developmental or reproductive effects of ethylbenzene in humans… EPA has classified ethylbenzene as a Group D, not classifiable as to human carcinogenicity." EPA,

Ethylbenzene, at 2 (2000). No reports or studies have been cited by the plaintiff or its experts indicating that human exposure in any form to ethylbenzene causes abnormal fetal development resulting in biliary atresia.

Dr. Pall's theory of causation falls into the bucket of personal speculation due to its lack of support from peer reviewed publications which the doctor claimed was available to give credence to his opinions. This is not to say the Court is making a scientific determination on the etiology of biliary atresia – it is not. However, even under the relaxed standards that govern the admissibility of expert testimony under Accutane, Dr. Pall's testimony simply falls short of the standard. It may be the case that some environmental toxins may induce biliary atresia, while perhaps others do not. But this is not a trial on the effects of all toxic compounds, only a select few. The science is just too immature at the present moment for the causal theory presented by Dr. Pall to be admissible at trial. The court has undertaken a substantial and thorough review of the medical literature and the support provided by Dr. Pall. After providing all favorable inferences to the plaintiff, this court is constrained and compelled to prohibit the introduction of any testimony from Dr. Pall. The Defendant's motion to bar the testimony of Dr. Pall is granted.

In conclusion, as a matter of law, the plaintiff cannot satisfy their burden of proof to claim that the plaintiff's biliary atresia was caused by inhalation of hazardous toxins found within her home. The plaintiffs are without the ability to proffer any evidence on the required element of causation. The failure to produce any evidence that may convince a reasonable jury that the plaintiff's exposure to toxic chemicals caused T.R. to develop biliary atresia compels the court to grant the defendant's motion to bar plaintiff's expert on causation, Dr. Harpreet Pall, M.D.

At this juncture, the parties are free to file or renew any motion which is now ripe for the court's determination.