IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA

ARMY JOE LEAKE, II,	:	
Plaintiff,	:	**CIVIL ACTION**
v.	:	
UNITED STATES OF AMERICA,	:	**No. 09-4564**
Defendant.	:	

MEMORANDUM OPINION

Goldberg, J. **December 29, 2011**

In March of 2008, Plaintiff, Army Joe Leake, II, suffered acute liver failure which necessitated an immediate liver transplant. Plaintiff alleges that this illness was the result of his work as a painter on a naval cargo ship and has sued Defendant, the United States of America, under the Jones Act, 46 U.S.C. § 30104, and maritime law.

Presently before the Court are Defendant's Motions to Exclude the Reports and Testimony of Plaintiff's causation experts, Dr. Jeffery A. Handler, Dr. Kay Washington and Dr. Rudy Rai. Because we conclude that these experts have failed to offer reliable opinions that will assist the trier of fact regarding the cause of Plaintiff's injury, Defendant's motions will be granted. Further, as the exclusion of this evidence precludes Plaintiff from establishing causation, we also grant Defendant's motions for summary judgment as to each of Plaintiff's claims.

I. Factual Background

Unless otherwise indicated, the following facts are undisputed.

Plaintiff worked aboard the United States Naval Ship GILLILAND as an Able Bodied Seaman, intermittently, from 2005 until March 1, 2008. From at least November 19, 2007 to December 16, 2007, and thereafter from January 8, 2008 to February 29, 2008, Plaintiff worked overtime chipping and painting the cargo hold of the ship. During the last week of February 2008, Plaintiff also spent three hours painting the laundry room and four days painting a stairwell on the ship. (Def.'s State. Facts, Doc. No. 29, Ex. 1, ¶¶ 1-4; Pl.'s Resp. State. Facts, Doc. No. 32, Ex. 4, ¶¶ 1-4.)

On March 3, 2008, Plaintiff began feeling ill, and on March 5, 2008, he was admitted to the hospital and diagnosed with acute liver failure. Plaintiff underwent a liver transplant on March 10, 2008. Although the transplant was successful, Plaintiff alleges that he has suffered several physical and mental complications. (Def.'s State. Facts, Doc. No. 29, Ex. 1, ¶¶ 8-13; Pl.'s Resp. State. Facts, Doc. No. 34, Ex. 4, ¶¶ 8-13; Compl. ¶ 13.)

Plaintiff contends that his exposure to chemicals in the paints and thinners he used aboard the ship caused his liver failure.[1] Plaintiff attempts to establish causation for his injuries through experts, Drs. Jeffrey A. Handler, Kay Washington and Rudy Rai. In summary, Drs. Handler and

[1] Plaintiff's claims, which arise under the Jones Act and general maritime law, are asserted against his former "employer" and the "owner" of the GILLILAND, the United States of America, not the manufacturer of the paints and thinners at issue. See Sloan v. United States, 603 F.Supp.2d 798, 804-12 (E.D.Pa. 2009) (Jones Act provides a remedy for a seaman against "his employer" and a maritime claim of unseaworthiness provides him a remedy against "the owner of a ship"); Irons v. Matthews, 2010 WL 2540347 at **5-6 (D.N.J. Jun. 15, 2010) (acknowledging that a general maritime law claim for maintenance and cure may be asserted against a "shipowner").

Washington identified three chemical compounds found in the paints and thinners that could cause liver damage: methyl n-amyl ketone ("MAK"), n-butanol and psuedocumene. Dr. Handler opined that Plaintiff's exposure to MAK resulted in an immune-based reaction that resulted in liver failure. Handler further concluded that Plaintiff's exposure to n-butanol and psuedocumene resulted in liver damage in the "the remaining liver cells." Dr. Washington also opined that MAK was capable of causing the immune-based reaction responsible for Plaintiff's liver failure, but concluded, more generally, that "toxic exposure to organic compounds present in the paint fumes" caused his injury. Similarly, Dr. Rai opined that Plaintiff "developed liver failure from his exposure to known hepatotoxins that were inhaled in high concentrations in enclosed spaces over a period of time." (Def.'s State. Facts, Doc. No. 29, Ex. 1, ¶ 16; Pl.'s Resp. State. Facts, Doc. No. 34, Ex. 4, ¶ 16; Handler Rpt. at 3-5; Washington Rpt. at 2-4; Rai Rpt. at 3.)

II. Legal Analysis

A. Federal Rule of Evidence 702

Federal Rule of Evidence 702 governs the admissibility of expert testimony, and states:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993). The current version of Rule 702 "embodies a trilogy of restrictions on expert testimony: qualification, reliability and fit." Schneider v. Fried, 320 F.3d 396, 404 (3d Cir. 2003). In evaluating whether an expert opinion is admissible, the district court acts as a gatekeeper, excluding opinion testimony that does not meet

these requirements. Id.

An expert's opinion is reliable if it is based upon "'methods and procedures of science' rather than subjective belief or unsupported speculation." In re Paoli R.R. Yard PBC Litig., 35 F.3d 717, 742-43 & n.7 (3d Cir. 1994) (quotation omitted.) In considering whether an expert's method is reliable, courts should consider: (1) whether it is based upon testable hypotheses; (2) subject to peer review; (3) the known or potential error rate; (4) the existence and maintenance of standards controlling the technique's operation; (5) whether it is generally accepted; (6) the relationship of the method to other methods that have been deemed reliable; (7) the expert's experience or qualification with the technique or method; (8) non-judicial uses the method has been put to; and (9) all other relevant factors. Id. The reliability requirement is not to be applied "too strictly" and is satisfied as long as the expert has "good grounds" for his or her opinion. Holbrook v. Lykes Bros. S.S. Co., 80 F.3d 777, 784 (3d Cir. 1996).

There also must be a "valid scientific connection" or fit, between the facts of the case and the expert's opinion. Daubert, 509 U.S. at 591; Holbrook, 80 F.3d at 777. This requirement ensures that the opinion is relevant and will "aid the jury in resolving a factual dispute." Id.

Finally, we recognize that Rule 702 is to be interpreted liberally in favor of admissibility, since "[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."[2] Schneider, 320 F.3d at 469. The burden is on the party offering the evidence to establish admissibility by a preponderance of the evidence. See Padillas v. Stork-Gamco, Inc., 186 F.3d 412,

[2] An expert is qualified if he or she has specialized knowledge "greater than the average layman." Waldorf v. Shuta, 142 F.3d 601 (3d Cir. 1998). Defendant does not challenge the qualifications of Plaintiff's experts.

418 (3d Cir. 1999).

B. Causation Analysis

In toxic tort cases, a plaintiff must demonstrate that the substance at issue is capable of causing the observed harm (general causation), and that the substance actually caused the harm suffered by the plaintiff (specific causation).[3] See In re Paoli R.R. Yard PBC Litig., 35 F.3d 717, 752 (3d Cir. 1994) ("[T]he personal injury plaintiffs must show that they were exposed to the chemicals released by the defendants, that these chemicals can cause the types of harm they suffered, and that the chemicals in fact did cause them harm"); Perry v. Novartis Pharmaceuticals Corp., 564 F.Supp.2d 452, 463-64 (E.D.Pa. 2008) (analyzing expert's opinion in light of general and specific causation standards in a case where the plaintiff allegedly developed lymphoma as a result of using a topical cream).

In light of these causation standards, Defendants contend that Plaintiff's experts have failed to offer "good grounds" to support a reliable opinion as to general or specific causation. Plaintiffs

[3] Although the causation standard for Plaintiff's Jones Act negligence claim is less stringent than the causation standards applicable to his maritime law claims, Plaintiff must establish general and specific causation to sustain each of his claims. Compare Sloan v. U.S., 603 F.Supp.2d 798, 804-12 (E.D.Pa. 2009) (reflecting that Jones Act causation standard is satisfied if the "proofs justify with reason the conclusion that employer negligence played any part, even the slightest, in producing the injury[,]" while the causation standard claims for an unseaworthiness claim is "more demanding," requiring proof that the alleged unseaworthy condition was a proximate and substantial cause of his injury) (quotation omitted); Wills v. Amerada Hess Corp., 379 F.3d 32, 52 (2d Cir. 2002) (acknowledging that where a plaintiff's illness manifests after service aboard a ship, he or she may not generally recover for maintenance and cure, "absent convincing proof of causal connection" between the injury and the plaintiff's service) (quotation omitted) with Clark v. Kellogg Brown & Root L.L.C., 414 Fed.Appx. 623, 626-27 (5th Cir. 2011) (analyzing toxic tort action under Jones Act and maritime law claims for unseaworthiness and maintenance and cure in light of general and specific causation standards); Green v. McAllister Bros., Inc., 2005 WL 742624 at **11-12 (S.D.N.Y. Mar. 25, 2005) (analyzing toxic tort claim under the Jones Act in light of general and specific causation standards).

disagree and assert that the causation opinions of Drs. Handler, Washington and Rai are sufficiently reliable.[4] Before resolving this dispute, we set forth our understanding of Plaintiff's experts' opinions.

Dr. Handler, Plaintiff's expert toxicologist, concluded that "[g]iven Plaintiff's employment and the activities he was involved with just before he became symptomatic, . . . exposure to chemicals in the paints and thinners that he was exposed to during his assignment on the [Ship] caused fulminate hepatic failure due to chemical-induced hepatic damage and a subsequent immune reaction." (Handler Rpt. at 3.) This portion of Handler's opinion seems to be premised upon a "temporal proximity" theory.[5] Dr. Handler noted that MAK, an organic compound in the paint products Plaintiff used, is capable of binding to liver proteins and promoting the development of "protein adducts" as well as "antibodies against these adducts." Dr. Handler explained that, due to the presence of the adducts and antibodies, a subsequent exposure to MAK could lead to the "formation of epitopes that the body regards as foreign objects, initiating an immune response[,]" which can severely damage the liver and ultimately cause liver failure as it did with Plaintiff. (Handler Rpt. at 3-5.) However, and as discussed in greater detail infra, Dr. Handler's theory regarding the "formation of epitopes" is not based upon exposure to MAK, the chemical Plaintiff

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[4] We note that Plaintiff has requested a Daubert hearing. The decision as to whether to hold a Daubert hearing rests with the sound discretion of the district court. See Kumho Tire Co., Ltd. v. Carmichael, 526 U.S. 137, 152 (1999); Oddi v. Ford Motor Co., 234 F.3d 136, 151-55 (3d. Cir. 2000). We conclude that a hearing is not necessary, given the parties' extensive briefing on the issues raised, including detailed analyses of each expert's conclusion and methodology. Further, the record includes the reports and deposition testimony of each expert at issue.

[5] Temporal proximity refers to proximity in time between Plaintiff's exposure to a toxin and the onset of symptoms. Cf. 92 AM. JUR. TRIALS 113, Litigating Toxic Mold Cases § 93 (2011).

was exposed to, but rather, a different chemical, halothane. Dr. Washington, Plaintiff's hepatic pathology expert, agreed with Dr. Handler's analysis, although she did not limit her causation opinion to MAK.[6]

As we understand Drs. Handler and Washington's opinions in lay-person's terms, they concluded that Plaintiff's exposure to the chemical compounds in the paints and thinners he used aboard the GILLILAND caused his liver failure to occur in two stages. They explain that initially, one of these chemicals, most specifically MAK, was incorporated into Plaintiff's liver over the course of his first several weeks working with the paints and thinners, resulting in the formation of protein adducts and antibodies. The formation of these adducts and antibodies prepared Plaintiff's liver for an autoimmune response in defense of a subsequent exposure. Thereafter, during his final week aboard the GILLILAND, Plaintiff was again exposed to MAK in a manner sufficient to trigger a severe autoimmune response. Handler explains that this type of second exposure could cause "severe hepatitis" in a "small subset of individuals" due to the presence of the antibodies, generated from the first exposure. (Handler Rpt. at 3-5; Washington Rpt. at 3-4.)

In reaching these conclusions, Drs. Handler and Washington primarily relied upon: (1) the temporal relationship between Plaintiff's exposure and his injury; (2) the "pattern of injury" to Plaintiff's liver, which, in their view, showed signs of an "immune-mediated" response; (3) a 1984 study which reported that the exposure of rats to radioactive labeled MAK caused the incorporation of the radioactivity into three unidentified liver proteins; (4) "precedent" describing the immune-

[6] (Washington Rpt. at 3-4) (reflecting opinion that Plaintiff's liver failure was the result of MAK's "incorporation . . . into the liver proteins, which can lead to the formations of neoantigens that the body regards as foreign, thus provoking an immune-mediated response[,]" but concluding that "organic compounds," including pseudocumene, n-butonol and [MAK,] caused Plaintiff's injury).

mediated reaction that occurs following "halothane" exposure; and (5) a differential diagnosis, which is "a standard scientific technique which identifies the cause of a medical problem by eliminating the likely causes until the most probable one is isolated."[7] (Handler Rpt. at 3-5; Washington Rpt. at 3.)

Defendants assert that these opinions should be excluded as unreliable, primarily because there is no scientific study that suggests MAK is capable of causing acute liver failure.[8] Plaintiff concedes that "there are no published studies addressing an immune-mediated [liver failure] resulting from exposure to the organic solvents found in the paints and thinners that were used by plaintiff." (Pl.'s Resp., Doc. No. 33, at 23.) Plaintiff contends, however, that the lack of a published

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[7] Dr. Rai's report does not describe the mechanism that may have caused Plaintiff's injury, but he testified that he was of the opinion that an immune-mediated response, consistent with that seen in certain cases of halothane exposure, caused Plaintiff's liver failure. Compare (Rai. Rpt. at 3) ("Based on the temporal relationship of his exposure to paints and thinners and ruling out of all other known causes of liver failure, it is my opinion that the patient developed liver failure from his exposure to known hepotoxins that were inhaled in high concentrations in enclosed spaces over time") with (Rai Depo. pp. 24, 38-44) (Plaintiff's initial exposures to paints and thinners "sensitize[d] him to an immune-mediated injury . . . . [and] created the foundation for that massive, final liver necrosis or injury" and noting that this kind of injury has been observed following "halothane" exposure). Dr. Rai, however, did not specify which "toxin" in the paints and thinners is capable of causing this reaction nor did he offer any additional support for his conclusion, beyond that contained in the opinions of Drs. Handler and Washington. Therefore, if the opinion of Drs. Handler and Washington are excluded, Dr. Rai's opinion will also be excluded.

[8] In toxic tort cases, general causation is often established through the use of "epidemiological studies," which examine the "pattern of disease in human populations." Prichard v. Dow Agro Sciences, 705 F.Supp.2d 471, 484-85 (W.D.Pa. 2010) (quoting General Elec. Co. v. Joiner, 522 U.S. 136, 144 n.2 (1997)). Animal studies may also provide sufficient scientific basis for an expert's general causation opinion, provided there are "good grounds to extrapolate [the findings] from animals to humans[.]" Fibrous v. Rexall Sundown, Inc., 2004 WL 1202984 at *7 (W.D.Pa. Jun. 2, 2004) (quotation omitted.)

study directly addressing his situation does not preclude his experts from offering a reliable opinion on general causation, particularly considering the temporal analysis and differential diagnosis performed by his experts.

In support of this position, Plaintiff primarily relies upon Heller v. Shaw Industries, Inc., 167 F.3d 146 (3d Cir. 1999). See (Pl.'s Resp., Doc. No. 33, at 17, 23-26.) In Heller, the United States Court of Appeals for the Third Circuit held that it is not necessary for a medical expert to "cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness." 167 F.3d at 154-55. The Court concluded that a strong "temporal relationship between an injury and a causal event" could obviate the need for a published study demonstrating a link between a substance and an injury.[9] Id. Specifically, the Court determined that a "temporal relationship" could form part of a properly performed "differential diagnosis," which in many instances will satisfy the requirements of Daubert and Rule 702. Id. at 155-56. The Court was careful to stress, however, that even in light of these "reliable methods," sometimes there "is simply too great an analytical gap between the data [in scientific studies] and the opinion proffered." Id. (quoting General Elec. Co. v. Joiner, 522 U.S. 135, 146 (1997). Indeed, we note that Daubert instructs that a reviewing court must always be satisfied that a proffered opinion is derived from scientific knowledge, not subjective belief or unsupported speculation. Daubert, 509 U.S. at 590.

[9] Specifically, the Heller Court determined that the plaintiff's expert was not precluded from testifying that the emission of volatile organic compounds (VOCs) from plaintiff's carpet caused her injury, simply because there were "no studies indicating that the level of VOCs detected in [plaintiff's home] could cause symptoms such as those experienced by [plaintiff]." Id. at 154-56. Ultimately, however, the Court determined that the temporal relationship between plaintiff's exposure to VOCs and the onset of her injury was "questionable" at best and affirmed the district court's exclusion of the expert's opinion. Id. at 157-58.

Plaintiff contends that the temporal analysis and differential diagnosis performed by his experts satisfies Heller and that any scientific studies offered in support of their opinions was "merely icing on the cake[.]" (Pl.'s Resp., Doc. No. 33, at 12.) Plaintiff urges that there is a strong temporal relationship between exposure and injury in this case. He points out that it is undisputed that he was exposed to paints and thinners from, at least, November 19, 2007 to December 16, 2007 and January 8, 2008 to February 29, 2008, and that he began feeling ill on March 3, 2008. (Def.'s State. Facts, Doc. No. 29, Ex. 1, ¶¶ 2-5, 13, 15; Pl.'s Resp. State. Facts, Doc. No. 32, Ex. 4, ¶¶ 2-5, 13, 15.) In Plaintiff's view, this sequence of events supports the opinions of Drs. Handler, Washington and Rai, which provide that Plaintiff's exposure to "toxins" prior to February 25, 2008 "primed" him for an immune-mediated response and that his subsequent exposure during the last week of February 2008 brought about the immune-mediated response that caused his liver failure in early March.[10]

Plaintiff also points to the testimony of his liver transplant surgeon, Dr. Robert A. Fisher, M.D., as supportive of this temporal analysis. Dr. Fisher testified that the period of time between

[10] (Pl.'s Resp., Doc. No. 33, at 18-23); see (Handler Rpt. at 4) ("In the case of [Plaintiff], his repeated exposure to [MAK in the paint products] . . . during his work in the No. 3 lower hold, ever at low levels below regulated exposure levels, would have produced sufficient time and exposures to generate protein adducts in his liver. His exposure to [MAK] during the last week on the vessel then caused a severe immune reaction that led to the significant liver damage and ultimately to the fulminate liver failure"); (Washington Rpt. at 3-4) ("The exposure to toxic substances in paint fumes is . . . temporally related to the development of liver injury in this case[,] in light of "the clear chronological link between exposure to the toxin or drug and the onset of liver injury" and the immune-mediated "mechanism" that caused the injury); (Rai Depo. pp. 39-40) (reflecting that past exposure to toxins "actually can sensitize him to an immune mediated injury. So it actually creates some adducts back then. And then when you have the final very high exposure, then that can cause the massive liver necrosis, because now you have these preformed antibodies that come in and cause very acute massive cell death").

Plaintiff's exposure to "some form of a toxin" in the paints and thinners and the onset of his symptoms was consistent with the impact of immune-mediated responses in rabbits.[11] Dr. Fisher, however, did not know what paints or thinners Plaintiff had worked with, the chemicals that were contained in those products or his level of exposure. Although Dr. Fisher was of the opinion that a "toxin" caused Plaintiff's injury, "which toxin [he] had no idea." (Fisher Depo. pp. 37-39, 52-53.)

We note several weaknesses in Plaintiff's arguments. First, although Plaintiff's experts contend that there is a temporal relationship between Plaintiff's exposure and injury, they do not point to any scientific evidence to suggest that the pattern or timing of his exposure was "sufficient" to "prime" him for an immune-mediated reaction and then facilitate such a reaction. Borrowing the example used in Heller, this is certainly not a case where "a person [is] doused with chemical X and immediately thereafter developed symptom Y[.]" Heller, 167 F.3d at 154 (quotation omitted). Plaintiff was exposed to the paints and thinners at issue for approximately three months prior to the alleged immune-mediated response that purportedly caused his liver failure. This extended time period weakens Plaintiff's experts' "temporal proximity" theory, which is based upon a close in time sequence between exposure and symptoms.

Most importantly, however, we do not read Heller to hold that even a strong temporal relationship between exposure and injury, in and of itself, is sufficient to establish a reliable general causation opinion. Heller instructs that a district court must consider the "analytical gap" between the scientific data and the expert's opinion in determining whether a temporal connection and

[11] (Fisher Depo. pp. 36-38) ("I know that biologically when you have a mitogen in animals . . . rabbits are very similar to humans in the way they have an immune response – if you give them a mitogen and then you take the mitogen away, the exposure, generally you see these horrible immune responses when we're looking at different disease process within about three to four days").

differential diagnosis provide "good grounds" for the opinion. Id. at 155-56, 158 ("We repeat that all of these reliable methods for making a diagnosis cannot sanitize an otherwise untrustworthy conclusion") (quotation omitted.) Thus, while a definitive study on general causation is not a prerequisite to the admissibility of an expert opinion, the scientific data an expert relies upon is not, as Plaintiff characterizes it, "merely icing on the cake." Rather, the district court must determine whether the "gap" between the scientific data and the opinions offered is simply too great to assist the trier of fact. For the following reasons, we conclude that the scientific evidence relied upon by Plaintiff's experts in support of their general causation opinion fails to pass muster under Heller and Daubert, even in light of their reliance upon temporal proximity and a differential diagnosis.

In determining that MAK is capable of causing Plaintiff's injury, Drs. Handler and Washington initially drew upon a 1984 study, performed by Phillip W. Albro, et al. ("Albro Study"), which demonstrated that radioactively labeled MAK "caused incorporation of the radioactivity into 3 unidentified liver proteins as well as into DNA, urea and cholesterol."[12] This study addressed the "binding of MAK and/or metabolites to proteins and DNA" in rat livers. The study did not, however, address whether a subsequent exposure to MAK could facilitate an immune-mediated response in rats or result in liver failure. Indeed, Dr. Handler testified that the animal studies in the literature "do not show hepatotoxicity upon exposure to MAK and aren't reflective of the conditions under which you would get the immune response[.]" Therefore, even if there are "good grounds" to extrapolate the findings of the Albro Study from rats to humans, the study does not demonstrate

[12] P. Albro et al., Metabolism of Methyl n-Amyl Ketone (2- heptanone) and its Binding to DNA of Rat Liver in vivo and in vitro, 51 Chemico-Biological Interactions 295-308 (1984).

that MAK is capable of causing what Drs. Handler and Washington claim occurred to Plaintiff – the development of "protein adducts" and an "immune-mediated" reaction resulting in liver failure.[13] See (Handler Resp. Rpt. at 3-4; Handler Depo. pp. 24-26, 38, 40); (Rai. Depo. pp. 25-27) (reflecting that the Albro study "did find binding, and it found novel protein-type chemicals formed," but "[t]hey say that they cannot conclusively prove or disprove the presence or absence" of "protein adducts").

To bridge to gap between the Albro study and liver failure in humans, Drs. Handler and Washington state that the pattern of injury seen in Plaintiff's liver is consistent with that seen following exposure to the compound halothane, which is used as a general anesthetic. We recognize that there is precedent in scientific literature that exposure to halothane can result in the development of protein adducts and antibodies in the liver, which upon subsequent exposure, can facilitate an immune-mediated response that causes liver failure. (Handler Rpt. at 4; Washington Rpt. at 3-4; Rai Depo. pp. 39-40.) Importantly, however, MAK, the substance at issue here, is chemically unrelated to halothane. In our view, reliance by Plaintiff's experts on science involving an entirely unrelated substance does not meet Daubert's requirements of a "valid scientific connection" between the facts and the opinion. Daubert, 509 U.S. at 591.

It is worth repeating that Plaintiff's experts have not pointed to any scientific evidence or an

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[13] Dr. Handler testified that no studies have been performed to demonstrate that MAK can bind to human liver proteins and form protein adducts, as was observed in the Albro study. (Handler Depo. p. 38.) It appears that the only indication in the record that MAK can impact the human liver is in the "Material Safety Data Sheet" for "Intergard 264 Dark Grey" paint, which notes that the National Institute for Occupational Health and Safety (NIOSH) associated MAK with "irritation, kidney liver" and the "New Jersey Department of Health and Senior Services Hazardous Substance Fact Sheet for MAK," which "notes that the compound may damage the liver." (Pl.'s Resp., Doc. No. 33, at 25-26 & Ex. 13; Handler Resp. Rpt. at 3.)

example from their clinical experience to suggest MAK, like haltohane, is capable of giving rise to an immune-mediated response or liver failure. Although Dr. Handler noted that "immune-based hepatotoxicity linked to protein adduct formation has been observed with other chemicals such as tienilic acid . . . and minocycline[,]" (Handler Resp. Rpt. at 3), there is no basis set forth in any of Plaintiff's expert opinions that suggests MAK may fall within the same category as these "other chemicals" or impact the liver in a similar manner. Indeed, it is telling that in response to Defendant's argument that there is no scientific evidence to suggest that MAK may give rise to an "immune response" or acute liver failure, Plaintiff is only able point to the expert opinions offered in connection with this case. (Def.'s State. Facts, Doc. No. 29, Ex. 1, ¶¶ 18-19; Pl.'s Resp. State. Facts, Doc. No. 32, Ex. 4, ¶¶ 18-19); see Heller 167 F.3d at 158-59 ("[T]he District Court could, however, properly consider the fact (rather than requiring it as a prerequisite to admissibility) that [the expert] relied on few, if any, studies linking exposure to the [chemical at issue] . . . to the illness"). Although an expert may extrapolate from existing scientific data, "nothing in either Daubert or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert."[14] General Electric v. Joiner, 522 U.S. 136, 146 (1997).

Plaintiff's experts attempt to downplay the lack of scientific support for their opinions by pointing out the "rare," "unpredictable" and "idiosyncratic" nature of immune-based hepatotoxicity.[15]

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[14] *Ipse dixit* is a Latin phrase meaning "something asserted but not proved." BLACK'S LAW DICTIONARY 833 (7th ed. 1999).

[15] Dr. Washington noted that the lack of studies was not surprising, as "[s]uch an immune mediated response will . . . occur in an unpredictable manner in a small subset of exposed individuals and will likely not be detected upon testing the general population when exposed in a similar manner." (Washington Resp. Rpt. at 3.) She further acknowledged that it was difficult to

While these descriptions may be accurate, we still may "not allow expert witnesses to speculate or base their conclusions on inadequate supporting science." Perry v. Novartis Pharmaceuticals Corp., 564 F.Supp.2d 452, 468 (E.D.Pa. 2008). '"[T]he courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it.'"[16] Id. (quoting Rosen v. Ciba-Giegy Corp., 78 F.3d 316, 319 (7th Cir. 1996); see also Wolfe v. Sec. of Health & Human Servs., 2006 WL 3419835 at *7 (Fed. Cl. Nov. 9, 2006) (excluding expert's opinion because it was based on subjective belief, rather than medical theory, where expert opined that plaintiff's injury was alleged "idiosyncratic reaction" to a vaccine, based upon a temporal connection between administration and injury and an absence of alternative causes).

Lastly, we reject Plaintiff's contention that his experts have provided reliable general causation opinions based upon their "properly performed differential diagnoses." (Pl.'s Resp., Doc. No. 33, at 11-13) (citing Heller v. Shaw Industries, Inc., 167 F.3d 146, 155 (3d Cir. 1999). In performing a differential diagnosis, "a physician begins by 'ruling in' all scientifically plausible causes of the plaintiff's injury." Glastetter v. Novartis Pharmaceuticals Corp., 252 F.3d 986, 989

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prove injuries "caused by specific drugs and toxins, in particular to those causing 'hypersensitivity' and 'idiosyncratic' reactions, as in this case." (Id. at 3) (citations omitted); see (Handler Resp. Rpt. at 3.) In addition, Dr. Rai testified that halothane toxicity only occurs in certain individuals, and may be due to "nutritional," "genetic" and "chronological" factors. (Rai Depo. p. 42.)

[16] In addition, Plaintiff claims that Drs. Handler and Washington do not limit their causation opinions to MAK, noting that both determined that other "toxins" from "the paints and thinners," either alone or in combination, may have caused the immune-mediated response that resulted in his acute liver failure. (Pl.'s Resp. State. Facts, Doc. No. 32, Ex. 4, ¶ 17.) Neither Dr. Handler nor Dr. Washington, however, offered any support for this conclusion, beyond that offered in support of their opinions regarding MAK. We, therefore, also conclude that their general causation opinions as to these "other toxins" do not rest on "good grounds" and must be excluded under Daubert.

(8th Cir. 2001). Thus, it is "assume[d] that general causation has been proven for a list of possible causes[,]" such that the physician can identify the probable cause by process of elimination. In re Rezulin Products, 2004 WL 2884327 **3-4 (S.D.N.Y. Dec. 10, 2004) (collecting cases); Soldo v. Sandoz Pharmaceuticals Corp., 244 F.Supp.2d 434, 524-25 (W.D.Pa. 2003).

A properly performed differential diagnosis, therefore, is built upon a reliable general causation finding – it does not establish general causation. Because the temporal analysis and scientific evidence offered by Plaintiff's experts are insufficient to suggest that MAK, or any other toxin in the paints and thinners, is capable of causing acute liver failure, we are unable to conclude that their differential diagnoses provide "good grounds" in support of a general causation opinion.[17] See Soldo, 244 F.Supp.2d at 516-17.

III. Motions for Summary Judgment

Defendants also filed two motions for partial summary judgment, seeking dismissal of Plaintiff's negligence and seaworthiness claims (Doc. No. 29) and his maintenance and cure claim (Doc. No. 30). Plaintiff concedes that all of his claims are contingent upon the causation opinions of Drs. Handler, Washington and Rai. (Doc. Nos. 31, 32.) Because we have concluded that Plaintiff has failed to offer any admissible evidence as to general causation, Defendant is entitled to summary judgment.[18] See Heller v. Shaw Industries Inc., 167 F.3d 146, 165-66 (3d Cir. 1999) (affirming grant

[17] We note that 14 to 15 % of cases of acute liver failure do not have an identified cause. (Washington Rpt. at 2.) Furthermore, "non-A to E" hepatitis or a "viral trigger" have not been "totally excluded" as potential causes of Plaintiff's liver failure. See (Washington Depo. pp. 86-87.)

[18] Defendant also argues that Plaintiff's Jones Act negligence claim is subject to dismissal because he failed to adduce sufficient evidence to suggest that his injury was not the result of a foreseeable risk of harm, noting that MAK is approved for consumption by the FDA and is used as a food additive. See (Def.'s Mot. Summ. J., Doc. No. 29, Ex. 2, at 15-18.) Because we

of summary judgment where district court excluded expert testimony on causation, because there was no genuine issue of material fact on causation); Fabrizi v. Rexall Sundown, Inc., 2004 WL 1202984 at *9 (W.D.Pa. Jun. 2, 2004) (holding that where a plaintiff has not provided sufficient evidence in support of general causation, his or her claim fails and there is no need to consider specific causation).

IV. Conclusion

For the reasons set forth above, Defendant's motions to exclude the expert testimony of Drs. Handler, Washington and Rai are granted. Defendant's motions for summary judgment as to each of Plaintiff's claims are also granted.

An appropriate order follows.

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conclude that Plaintiff has failed to adduce evidence to support a causation finding, however, we need not reach this issue.