IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF TEXAS
EL PASO DIVISION

LEANNE SPARLING and MICHAEL J. SPARLING, on behalf of and as representatives for MICHAEL L. SPARLING, deceased,	§	
Plaintiffs,	§	
v.	§	**No. EP-13-CV-323-DCG**
JONATHAN VINCENT DOYLE, an individual, JACOB GEISSLER, an individual, USPLABS JACK3D, LLC, USPLABS, LLC, USPLABS HOLDING, LLC, GNC CORPORATION, NATURAL ALTERNATIVES INTERNATIONAL, INC., and DOES 1-500, inclusive,	§	
Defendants.	§	

ORDER

On this day, the Court considered "Defendants' Motion[s] to Strike the Testimony of [Dr. Louis Cantilena, Dr. Mahmoud ElSohly, Dr. Edward Mills, and Dr. Daniel Rusyniak]" ("Defendants' Motions to Strike") (ECF Nos. 184, 185, 186, 187) filed by Defendants Jonathan Vincent Doyle, an individual, Jacob Geissler, an individual, USPLABS JACK3D, LLC, USPLABS, LLC, USPLABS HOLDING, LLC, and GNC Corporation (collectively, "Defendants") on January 22, 2015. On January 26, 2015, the matter was referred to this Court under 28 U.S.C. § 636(b) and Appendix C of the Local Court Rules. (ECF No. 188). Plaintiffs Leanne Sparling and Michael J. Sparling filed a Response ("Plaintiffs' Omnibus Response") (ECF No. 200) on February 10, 2015. Defendants subsequently filed four Individual Replies[1] (ECF Nos. 213, 219, 220, 221) and an Omnibus Reply (ECF No. 218) on March 6, 2015, and

[1] The Court will refer to Defendants' Individual Motions and Replies by the expert's name followed by Mot. or Reply, i.e., (Dr. Cantilena Mot. 1, ECF No. 184).

March 10, 2015, respectively. On April 27, 2015, and April 28, 2015, the Court held a hearing pursuant to *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993). (*See* Order 1-3, ECF No. 235; *see also* ECF No. 321). On July 24, 2015, the Court received a Joint Notice of Second Supplemental Reports from the parties containing further Supplemental Rule 26 Expert Reports.[2]

After considering the testimony of the experts, the pleadings,[3] and the applicable law, the Court orders that Defendants' Motion to Strike Dr. Cantilena, Dr. Mills, and Dr. Rusyniak should be **GRANTED** and Defendants' Motion to Strike Dr. ElSohly should be **DENIED**.

I. BACKGROUND

Plaintiffs allege that Michael L. Sparling ("Decedent") died as a result of his use of Jack3d, a dietary supplement containing DMAA.[4] Plaintiffs' First Amended Complaint alleges causes of action for negligence, strict products liability-defective design, strict products liability-failure to warn, breach of express warranty, breach of implied warranty, wrongful death, and survival action. (*See* Am. Compl. ¶¶ 34, 90-148).

In support of their allegations, Plaintiffs have designated six experts to testify at trial. (*See* ECF No. 106). Defendants now seek to strike the Rule 26 Expert Reports and testimony of four of those experts: 1) Louis Cantilena, M.D. ("Dr. Cantilena"); 2) Mahmoud ElSohly, Ph.D. ("Dr. ElSohly"); 3) Edward Mills, Ph.D. ("Dr. Mills"); and 4) Daniel Rusyniak, M.D. ("Dr. Rusyniak"). (ECF Nos. 184, 185, 186, 187).

[2] Per the Court's request, the parties submitted the Second Supplemental Rule 26 Expert Report and Declaration of Dr. Cantilena and the Supplemental Rule 26 Expert Report and Declaration of Dr. Mills. (ECF Nos. 327-1, 327-22).

[3] The Court fully considered the parties' arguments but declines to rehash the extensive briefing, except when necessary.

[4] DMAA is shorthand for the compound 1,3-Dimethylamylamine. (*See, e.g.*, Dr. Cantilena Mot. 9 n.36, ECF No. 184; Pls.' Omnibus Resp. 12, ECF No. 200).

II. LEGAL STANDARD

Federal Rule of Evidence 702, which governs the admissibility of expert testimony, provides:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if: (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue; (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reliably applied the principles and methods to the facts of the case.

In oft-cited cases, the Supreme Court has articulated the district court's gate-keeping function under Rule 702. *See Daubert*, 509 U.S. at 592-98; *see also Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999). In performing this gate-keeping function, the court has broad discretion in deciding whether or not to admit expert testimony. *See Johnson v. Arkema, Inc.*, 685 F.3d 452, 460 (5th Cir. 2012); *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 276 (5th Cir. 1998) (en banc). The burden of proof on a *Daubert* issue lies with the proponent of the expert's testimony. "The proponent need not prove that the expert's testimony is correct, but [he or] she must prove by a preponderance of the evidence that the testimony is reliable." *Moore*, 151 F.3d at 276.

In *Daubert*, the Supreme Court held that Rule 702 imposes an obligation upon the trial judge to "ensure that any and all scientific testimony admitted is not only relevant, but reliable." *Daubert*, 509 U.S. at 589. Generally, "an expert is permitted wide latitude to offer opinions, including those that are not based on firsthand knowledge or observation." *Id.* at 592. *Daubert*, however, sets forth four specific factors that the district court should ordinarily apply when considering the reliability of scientific evidence: 1) whether the technique can or has been tested; 2) whether it has been subjected to peer review or publication; 3) whether there is a known or potential rate of error; and 4) whether the relevant scientific community generally accepts the

technique. *Id.* at 592-94. *Daubert* also instructs courts that the issue at hand is based "solely on principles and methodology, not on the conclusions that they generate." *Id.* at 595.

In *Kumho Tire*, the Supreme Court concluded that a district court may consider one or more of the specific *Daubert* factors when doing so will help determine the reliability of the expert's testimony. *Kumho Tire Co.*, 526 U.S. at 151.

> But, as the Court stated in *Daubert*, the test of reliability is "flexible," and *Daubert*'s list of specific factors neither necessarily nor exclusively apply to all experts or in every case. Rather, the law grants a district court the same broad latitude when it decides *how* to determine reliability as it enjoys in respect to its ultimate reliability determination.

Id. at 142 (citing *General Elec. Co. v. Joiner*, 522 U.S. 136, 143 (1997)). Thus, whether *Daubert*'s suggested indicia of reliability apply to any given testimony depends on the nature of the issue at hand, the witness's particular expertise, and the subject of the testimony. *Id.* at 151-52; *see also Black v. Food Lion, Inc.*, 171 F.3d 308, 311-12 (5th Cir. 1999) ("In the vast majority of cases, the district court first should decide whether the factors mentioned in *Daubert* are appropriate."). Therefore, under *Kumho Tire*, district courts have broad latitude to determine whether or not the proffered testimony requires an application of the *Daubert* factors and the Supreme Court has acknowledged that "[t]oo much depends upon the particular circumstances of the particular case at issue" to set out rigid requirements to test reliability." *Id.* at 150.

In making the reliability determination, the district court should not require certainty but the testimony must demonstrate that the opinions offered are more than speculation. In considering this issue, the district court must consider the validity of the principles applied by the expert, the accuracy of the data relied upon by the expert, and the precision of the application of the principles to the relevant data. *See Newton v. Roche Labs., Inc.*, 243 F. Supp. 2d 672, 676 (W.D. Tex. 2002) (citing *Marcel v. Placid Oil*, 11 F.3d 563, 567 (5th Cir. 1994)). Thus, the Court's task is to "determine whether the evidence is genuinely scientific, as distinct from being

unscientific speculation offered by a genuine scientist." *Moore*, 151 F.3d at 278 (quoting *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 318 (7th Cir. 1996)).

The Fifth Circuit has been particularly careful to distinguish evidence that establishes a causal relationship between the product and the alleged injury, from evidence that merely "suggests" an association between the product at issue and the injury. *See Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 197 (5th Cir. 1996). Although evidence of an association may indicate the need for further research and be important in the scientific and regulatory contexts, the Fifth Circuit has cautioned that tort law requires a "higher standard" of causation than that used in the regulatory context. *Id.* at 198. Moreover, as Judge Posner has noted, "[l]aw lags science; it does not lead it." *Rosen*, 78 F.3d at 319. Courts "must resolve cases . . . on the basis of scientific knowledge that is currently available," and only evidence that demonstrates a causal relationship between a product and an alleged injury can be admitted as relevant and reliable. *See Moore*, 151 F.3d at 274, 276; *see also Brumley v. Pfizer, Inc.*, 200 F.R.D. 596, 602 (S.D. Tex. 2001) (requiring evidence of causation and noting that "the lack of proof of a drug's safety does not prove that it is dangerous"). However, "[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.

"The relevancy requirement ensures that the expert testimony will actually 'assist the trier of fact to understand the evidence or to determine a fact in issue.'" *Lofton v. McNeil Consumer & Specialty Pharm.* (*Lofton I*), Civ. A. No. 3:05-CV-1531-L (BH), 2008 WL 4878066, at *2 (N.D. Tex. July 25, 2008) (citing *Daubert*, 509 U.S. at 589), *report and recommendation adopted*, *Lofton v. McNeil Consumer & Specialty Pharm.* (*Lofton II*), 682 F. Supp. 2d 662 (N.D. Tex. 2010). Relevant evidence is that which has "any tendency to

make the existence of any fact that is of consequence to the determination of the action more probable or less probable that it would be without the evidence." Fed. R. Evid. 401. "Expert testimony which does not relate to any issue on the case is not relevant and, ergo, non-helpful." *Lofton I*, 2008 WL 4878066, at *2 (citing *Daubert*, 509 U.S. at 590).

III. ANALYSIS

A. Defendants' Challenge to Plaintiffs' Rule 26 Expert Reports

1. Defendants' Challenge to Plaintiffs' Rule 26 Expert Reports Under Rule 37(c)(1)

Defendants argue that the Court should strike the testimony and opinions of Dr. Cantilena, Dr. Mills, and Dr. Rusyniak under Rule 37(c)(1) of the Federal Rules of Civil Procedure[5] because their Rule 26 Expert Reports do not contain "a complete statement of all opinions . . . and the basis and reasons for them" and "the facts or data considered by the witness in forming them." (*See* Dr. Cantilena Mot. 6-9, ECF No. 184; Dr. Mills Mot. 6-8, ECF No. 186; Dr. Rusyniak Mot. 6-7, ECF No. 187).[6] Defendants contend that the Rule 26 Expert Reports "hardly disclose[] anything other than the proposed [experts'] ultimate, generalized conclusion[s] about causation." (*See* Dr. Cantilena Mot. 7, ECF No. 184; Dr. Mills Mot. 7, ECF No. 186; Dr. Rusyniak Mot. 7, ECF No. 187). Defendants maintain that Dr. Cantilena admitted in his deposition that he is not qualified to testify on labeling requirements for dietary supplements and

[5] Rule 37(c)(1) provides:

> If a party fails to provide information . . . as required by Rule 26(a) or (e), the party is not allowed to use that information or witness to supply evidence on a motion, at a hearing, or at a trial, unless the failure was substantially justified or is harmless. In addition to or instead of this sanction, the court, on motion and after giving an opportunity to be heard: (A) may order payment of the reasonable expenses, including attorney's fees, caused by the failure; (B) may inform the jury of the party's failure; and (C) may impose other appropriate sanctions, including any of the orders listed in Rule 37(b)(2)(A)(i)-(vi).

[6] Where there is a discrepancy between assigned ECF page numbers and internal document page numbers, the Court adopts ECF pagination. This proviso will not apply to the page numbers of the experts' depositions and the hearing transcript, which will be referred to by page and line, where appropriate.

that he will not be offering opinions as to the labeling or warnings, even though his Rule 26 Expert Report contains an opinion on that issue. (*See* Dr. Cantilena Mot. 8-9, ECF No. 184).

Defendants also claim that Plaintiffs' experts' testimony must be limited to the scope of their Rule 26 Expert Reports and any other testimony must be stricken. (*See* Defs.' Omnibus Reply 26, ECF No. 218). Defendants argue that the bases for the experts' opinions must be included in their Rule 26 Expert Reports rather than as exhibits or references. (*See id.* at 24-25). Defendants further contest Plaintiffs' Rule 26 Expert Reports on the ground that they do not use the term "mechanism of action." (*See id.*)

As to Dr. Cantilena's Rule 26 Expert Report, Defendants assert that he failed to include the specific outlier data he relied upon, the source of that data, and the scientific bases for using that data rather than other data. (*See* Dr. Cantilena Reply 3-4, ECF No. 219). Defendants further contend that Dr. Cantilena failed to include scientific evidence or an explanation regarding the "susceptible population" and how Decedent is part of that population. (*See id.* at 6-7).

Plaintiffs contend that the challenged Rule 26 Expert Reports are sufficient to meet the requirements of the Federal Rules. (*See* Pls.' Omnibus Resp. 48-53, 55-60, ECF No. 200). Plaintiffs argue that the Rule 26 Expert Reports contain: 1) opinions on the biological and pharmacological mechanism of action of DMAA (*see id.* at 49, 55-56, 58-59); 2) an explanation as to why no baseline toxic dose exists and information regarding the dose-response relationship (*see id.* at 50-51, 58-59); 3) opinions on the use of a differential diagnosis (*see id.* at 52-53, 55-56); and 4) an attached list of references to scientific literature relied upon by the experts. (*See id.* at 49, 57-58). Plaintiffs assert that Defendants deposed each of the experts for multiple hours in which they testified to their methodology and opinions. (*See id.* at 49, 56). Plaintiffs further contend that there is no requirement that each premise offered in every paragraph of the Rule 26 Expert Reports contain a citation and page number. (*See id.* at 49). Plaintiffs, however,

fail to respond to Defendants' argument regarding Dr. Cantilena's opinion as to the labeling and warnings on the Jack3d label.

Rule 26(a)(2) imposes a "duty to disclose information regarding expert testimony sufficiently in advance of trial [so] that opposing parties have a reasonable opportunity to prepare for effective cross examination and perhaps arrange for expert testimony from other witnesses." Fed. R. Civ. P. 26(a)(2) advisory committee's note (1993). "One of the purposes of the Rule 26 disclosure and report is to compel the proponent of the expert to be prepared for the remainder of the trial of the case by requiring the expert to finalize his opinions at least ninety days prior to trial." *Bro-Tech Corp. v. Purity Water Co. of San Antonio, Inc.*, Civ. No. SA-08-CV-0594-XR, 2009 WL 1748539, at *7 (W.D. Tex. June 19, 2009) (citations omitted). Expert reports should be "detailed and complete" so as "to avoid the disclosure of 'sketchy and vague' expert information." *Sierra Club v. Cedar Point Oil Co. Inc.*, 73 F.3d 546, 571 (5th Cir. 1996).

Although there is a question of whether Plaintiffs' Rule 26 Expert Reports satisfy the plain language of the Rule, the Court finds that any error by Plaintiffs is harmless and that a sanction of striking the testimony under Rule 37 would be too drastic a remedy. In evaluating whether a violation of Rule 26 is harmless, the Court examines four factors: 1) the importance of the evidence; 2) the prejudice to the opposing party of including the evidence; 3) the possibility of curing such prejudice by granting a continuance; and 4) the explanation for the party's failure to disclose. *See id.* at 572.

As for the first factor, it is clear that the evidence contained in the Rule 26 Expert Reports is of such high importance to Plaintiffs' case that, without it, Plaintiffs would lack causation testimony. As for the second factor, Defendants have not clearly explained how they will be prejudiced in light of the fact they have held multiple hours of depositions and were able to examine these experts further during the *Daubert* hearing. As to the third factor, "[w]hile the

Court is sympathetic to Defendants, who first identified the missing information in [the Rule 26 Expert Reports], supplementation may be proper . . . when it is in response to questions or challenges to the [experts'] opinion[s] raised by the opposing party." *Charles v. Sanchez*, No. EP-13-CV-00193-DCG, 2015 WL 808417, at *9 (W.D. Tex. Feb. 24, 2015) (Guaderrama, J.) (collecting cases). As for the fourth factor, Plaintiffs make clear that they have fully disclosed their experts' opinions in their Rule 26 Expert Reports and that the depositions of Plaintiffs' experts allowed Defendants to further explore any questions that arose during Defendants' review of Plaintiffs' Rule 26 Expert Reports. In considering the four factors together, the Court finds that the violation of Rule 26(a)(2)(B) was harmless and declines to strike the testimony of Dr. Cantilena, Dr. Mills, and Dr. Rusyniak under Rule 37. The Court will, however, strike the portions of Dr. Cantilena's Rule 26 Expert Report and testimony as it relates to labeling and warnings on the Jack3d label, because he admitted in his deposition that he was not offering an opinion on that subject. (*See* Dr. Cantilena Dep. 211:19-24, 218:7-19, 220:13-17, 350:16-351:11; Pls.' Ex. HH ¶ 10, ECF No 197-29).

2. Defendants' Challenge to Dr. Cantilena's Supplemental Rule 26 Expert Report

Defendants argue that Dr. Cantilena's Supplemental Rule 26 Expert Report was submitted untimely and that the Court should, therefore, strike it or prevent Plaintiffs from relying on the Coyle study.[7] (*See* Dr. Cantilena Mot. 8, ECF No. 184). Defendants contend that Dr. Cantilena discovered the "Coyle study as much as a month before his deposition" but did not disclose its existence or his reliance on that study until the middle of his deposition. (*See id.*) Based on that ground, Defendants argue that Dr. Cantilena failed to supplement "in a timely

[7] The parties refer to Ricardo Mora-Rodriguez et al., *Plasma Catecholamines and Hyperglycaemia Influence Thermoregulation in Man During Prolonged Exercise in the Heat*, 491 J. Physiology 529 (1996), as the Coyle study because Dr. Edward F. Coyle is the senior author. (Pls.' Ex. R-25, ECF No. 197-2).

manner" as required by Rule 26(e) of the Federal Rules of Civil Procedure. (*See id.*) Defendants state that Dr. Cantilena's untimely disclosure prejudiced Defendants by surprising them at Dr. Cantilena's deposition. (*See id.*) Defendants maintain that Dr. Cantilena failed to disclose his conversions and calculations mentioned in his Supplemental Rule 26 Expert Report or the scientific bases for the validity of those conversions and calculations.[8] (*See* Dr. Cantilena Reply 3, ECF No. 219).

Plaintiffs argue that Dr. Cantilena's Supplemental Rule 26 Expert Report was timely submitted on December 24, 2014.[9] (*See* Pls.' Omnibus Resp. 54, ECF No. 200). Plaintiffs assert that Dr. Cantilena's Supplemental Rule 26 Expert Report was submitted in conjunction with the Rebuttal Rule 26 Expert Report of Dr. Edward Coyle. (*See id.*) Plaintiffs contend that it became necessary to supplement Dr. Cantilena's Rule 26 Expert Report after Defendants' expert, Dr. Laci Alexander, introduced a theory that caused Plaintiffs to hire Dr. Coyle to rebut. (*See id.*) At that time, Plaintiffs further contend that Dr. Cantilena reviewed Dr. Coyle's studies and determined that one of Dr. Coyle's studies provided additional data regarding "the effects of equivalent doses of sympathomimetics on vasoconstriction and increased core temperature through heat retention." (*See id.*) Additionally, Plaintiffs argue that Dr. Cantilena testified about this research at his deposition on December 15, 2014, and timely submitted his Supplemental Rule 26 Expert Report in advance of the discovery cutoff and pretrial disclosure deadline. (*See id.*) Moreover, Plaintiffs state that they offered Defendants the opportunity to further depose Dr.

[8] The Court notes that Dr. Cantilena's conversions and calculations were first presented to Defendants on the Monday before the *Daubert* hearing. (Hr'g Tr. 199:10-18, Apr. 27, 2015 AM, ECF No. 321). A copy of Dr. Cantilena's handwritten conversions and calculations were presented at the *Daubert* hearing. (Pls.' Ex. 7, *Daubert* hearing).

[9] Although Plaintiffs argue that Dr. Cantilena's Supplemental Rule 26 Expert Report was submitted on December 24, 2014, a review of the Supplemental Report indicates that it is dated January 12, 2015. (*See* Pls.' Ex. QQ 3, ECF No. 197-38).

Cantilena on this issue. (*See id.*, citing Dr. Cantilena Dep. 146:5-13).

Supplemental Rule 26 Expert Reports are governed by Rule 26(e). Rule 26(e)(1)(A) requires that Rule 26 Expert Reports be supplemented "in a timely manner if the party learns that in some material respect the disclosure or response is incomplete or incorrect, and if the additional or corrective information has not otherwise been made known to the other parties during the discovery process or in writing." An expert's duty to provide a Supplemental Rule 26 Expert Report "extends both to information included in the report and to information given during the expert's deposition." The supplemental information "must be disclosed by the time the party's pretrial disclosures under Rule 26(a)(3) are due." As Rule 26(a)(3)(B) states that pretrial disclosures "must be made at least 30 days before trial," a Supplemental Rule 26 Expert Report is timely if submitted before that date.

The Court finds that Plaintiffs timely submitted Dr. Cantilena's Supplemental Rule 26 Expert Report and will consider Dr. Cantilena's use of the Coyle study in evaluating the reliability of his opinion. Plaintiffs designated Dr. Coyle on December 10, 2014. (*See* ECF No. 165). Dr. Cantilena's deposition took place on December 15, 2014. (*See* Dr. Cantilena Dep. 1). During his deposition, Dr. Cantilena testified that he discovered Dr. Coyle's study approximately one month to three weeks before his deposition. (*See id.* at 133:4-16). Dr. Cantilena's Supplemental Rule 26 Expert Report is dated January 12, 2015. (*See* Pls.' Ex. QQ 3, ECF No. 197-38). Although this date is after Dr. Cantilena's deposition, the Court notes that this date is well before pretrial disclosures were due when the trial was set for June 2015 and is well before pretrial disclosures are due for the new trial date in February. (*See* Text Order Sept. 24, 2014; Order 2-3, ECF No. 320). Therefore, the Court finds that Dr. Cantilena's Supplemental Rule 26 Expert Report was timely submitted and will not be stricken for that reason.

B. Applicability of *Havner*

Throughout their briefing, Defendants insist that the Court apply *Merrell Dow Pharmaceuticals, Inc. v. Havner*, 953 S.W.2d 706 (Tex. 1997), as the standard for relevance under *Daubert*. (*See* Dr. Cantilena Mot. 9-11, ECF No. 184; Dr. Mills Mot. 8-10, ECF No. 186; Dr. Rusyniak Mot. 8-10, ECF No. 187; Defs.' Omnibus Reply 13-14, ECF No. 218; Dr. Cantilena Reply 4-5, ECF No. 219; Dr. Mills Reply 3-4, ECF No. 220; Dr. Rusyniak Reply 3-5, ECF No. 221). In applying *Havner* as the standard for relevance under *Daubert*, Defendants argue that Plaintiffs must provide epidemiological studies that demonstrate more than a doubling of the risk of injury in order to prove general causation. (*See* Dr. Cantilena Mot. 9-11, ECF No. 184; Dr. Mills Mot. 8-10, ECF No. 186; Dr. Rusyniak Mot. 8-10, ECF No. 187; Defs.' Omnibus Reply 13-14, ECF No. 218; Dr. Cantilena Reply 4-5, ECF No. 219; Dr. Mills Reply 3-4, ECF No. 220; Dr. Rusyniak Reply 3-5, ECF No. 221). In *Havner*, the Texas Supreme Court addressed the issue of whether evidence was *legally sufficient* to support the judgment issued by the lower court. *See Havner*, 953 S.W.2d at 708, 711. The *Havner* court held that in order to establish some evidence of general causation, studies must meet certain requirements, including: 1) demonstrating "more than a doubling of the risk" due to exposure; 2) a confidence interval that does not include 1.0; and 3) a confidence level of at least ninety-five percent.[10] *See id.* at 718, 723-24. Additionally, the *Havner* court stated that the plaintiff must show that his circumstances are similar to the group analyzed in the study. *See id.*

[10] Confidence levels in epidemiological studies establish the boundaries of the relative risk in a study. *See Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706, 723 (Tex. 1997). The boundaries are called the confidence interval. *See id.* (citations omitted). Confidence intervals allow scientists to determine whether studies are statistically significant at a certain confidence level. *See id.* (citation omitted). The confidence interval demonstrates "a range of values within which the results of a study sample would be likely to fall if the study were repeated numerous times." *See id.* (internal quotation marks and citation omitted). If a confidence interval includes 1.0, a study is not statistically significant because the relative risk values demonstrate that "the null hypothesis (1.0)" should be both accepted and rejected. *See id.* (citations omitted). Therefore, a doubling of the risk refers to a relative risk greater than 2.0. *See id.* at 721, 724-25.

at 720. Thus, Defendants contend that Plaintiffs' experts fail to offer studies which meet the *Havner* standard and, therefore, are not relevant evidence of causation under *Daubert*. (*See* Dr. Cantilena Mot. 9-11, ECF No. 184; Dr. Mills Mot. 8-10, ECF No. 186; Dr. Rusyniak Mot. 8-10, ECF No. 187; Defs.' Omnibus Reply 13-14, ECF No. 218; Dr. Cantilena Reply 4-5, ECF No. 219; Dr. Mills Reply 3-4, ECF No. 220; Dr. Rusyniak Reply 3-5, ECF No. 221).

In support of the argument that *Havner* applies a standard requiring more than a doubling of the risk of injury, Defendants state that the Texas Supreme Court reaffirmed the *Havner* decision in *Bostic v. Georgia-Pacific Corp.*, 439 S.W.3d 332 (Tex. 2014). (*See* Dr. Cantilena Mot. 11, ECF No. 184; Dr. Mills Mot. 10, ECF No. 186; Dr. Rusyniak Mot. 9-10, ECF No. 187; Defs.' Omnibus Reply 14, ECF No. 218). Defendants argue that the *Bostic* court "held that in toxic tort cases expert testimony of causation must be *scientifically reliable* in the form of epidemiological studies showing that the defendant's product '*more than doubled* the plaintiff's risk of injury.'" (*See* Dr. Cantilena Mot. 11 & n.47, ECF No. 184; Dr. Mills Mot. 10 & n.45, ECF No. 186; Dr. Rusyniak Mot. 9-10 & n.42, ECF No. 87). In *Bostic*, the Texas Supreme Court addressed whether evidence was *legally sufficient* to support the judgment of the lower court, but this time in a case involving asbestos and mesothelioma. *See Bostic*, 439 S.W.3d at 336. The *Bostic* court acknowledged that "*Havner* is a foundational part of our jurisprudence[] [but] [w]e have *never* held that it applies universally *to all tort cases where causation is an issue*." *Id.* at 347 (emphasis added). The *Bostic* court further stated that "[*Havner*] offers an alternative method of establishing causation '[i]n the absence of direct, scientifically reliable proof of causation.'" *Id.* at 347-48. The *Bostic* court concluded that:

> [I]n the absence of direct proof of causation, establishing causation in fact against a defendant in *an asbestos-related disease case* requires scientifically reliable proof that the plaintiff's exposure to the defendant's product more than doubled his risk of contracting the disease. A more than doubling of the risk must be shown through reliable expert testimony that is based on epidemiological studies

or similarly reliable scientific evidence.

Id. at 350 (emphasis added). Defendants thus argue that the Court must apply a standard of more than a doubling of the risk of injury to determine whether Plaintiffs' experts have offered relevant evidence of causation, as reaffirmed in *Bostic*. (*See* Dr. Cantilena Mot. 11, ECF No. 184; Dr. Mills Mot. 10, ECF No. 186; Dr. Rusyniak Mot. 9-10, ECF No. 187; Defs.' Omnibus Reply 14, ECF No. 218).

In addition, Defendants cite *Cano v. Everest Minerals Corp.*, 362 F. Supp. 2d 814 (W.D. Tex. 2005) (Rodriguez, J.), as an example of a case from this district where the court applied *Havner* to determine that evidence was irrelevant and inadmissible under *Daubert*. (*See* Dr. Cantilena Mot. 10 & n.42, ECF No. 184; Dr. Mills Mot. 9 & n.40, ECF No. 186; Dr. Rusyniak Mot. 9 & n.37, ECF No. 187; Defs.' Omnibus Reply 13-14, ECF No. 218). The *Cano* court stated:

> If evidence is admissible under federal procedural law but fails to constitute "some evidence" under Texas substantive law, the Plaintiffs' victory on the admissibility question would be a hollow one, as the evidence would be deemed insufficient as a matter of law to survive summary judgment. Moreover, whether expert testimony will assist the trier of fact is governed in part by whether the testimony is relevant to the plaintiff's burden of proof under the substantive law, and testimony that will not assist the trier of fact by advancing an element of the plaintiff's case should be excluded. Thus, the Court concludes that *Havner* controls the issue of what evidence is required to establish causation in a toxic tort case and therefore what evidence is relevant.

362 F. Supp. 2d at 821-22 (internal citations omitted). Defendants thus argue that the Court should exclude Plaintiffs' experts because their proffered testimony fails to meet the standards set forth in *Havner* and applied in *Cano*. (*See* Dr. Cantilena Mot. 10 & n.42, ECF No. 184; Dr. Mills Mot. 9 & n.40, ECF No. 186; Dr. Rusyniak Mot. 9 & n.37, ECF No. 187; Defs.' Omnibus Reply 13-14, ECF No. 218).

Defendants further argue that if the Court declines to apply *Havner* to the relevance determination under *Daubert*, "federal common law" also applies the "doubling of the risk"

standard to satisfy the preponderance of the evidence burden in civil cases. (*See* Defs.' Omnibus Reply 20, ECF No. 218, citing *Daubert v. Merrell Dow Pharm., Inc.* (*Daubert II*), 43 F.3d 1311, 1320 (9th Cir. 1995); *Pozefsky v. Baxter Healthcare Corp.*, No. 92CV0314LEKRWS, 2001 WL 967608, at *3 (N.D.N.Y. Aug. 16, 2001)).

Plaintiffs contend that epidemiological studies showing a "doubling of the risk" are not necessary to determine the admissibility of experts under Rule 702 and *Daubert*. (*See* Pls.' Omnibus Resp. 38, ECF No. 200). Moreover, Plaintiffs argue that whether an expert's opinion is admissible is a matter of federal law rather than state law. (*See id.*) Although courts have stated that epidemiologic studies provide useful evidence, Plaintiffs argue that they are not required under Rule 702 and *Daubert*. (*See id.*, citing *Allen*, 102 F.3d at 195; *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 308 (5th Cir.), *modified on reh'g*, 884 F.2d 166 (5th Cir. 1989); *In re Norplant Contraceptive Prods. Liab. Litig.*, 215 F. Supp. 2d 795, 831 (E.D. Tex. 2002)). Plaintiffs, therefore, conclude that their lack of an epidemiological study showing a doubling of the risk "is of no consequence under Rule 702." (*See id.* at 40).

The Court relies on *Lofton II* in holding that the *Havner* requirements are not applicable to the question of whether Plaintiffs' experts' testimony is relevant under Rule 702 and *Daubert*. *See Lofton II*, 682 F. Supp. 2d at 668-69. In that case, the district court considered *Havner* and *Cano*, other decisions on the issue from the Northern District of Texas, and Fifth Circuit decisions which considered *Havner*. *See id.* The *Lofton II* court concluded that the Fifth Circuit has only considered the applicability of *Havner* in the context of legal sufficiency of the evidence supporting a jury verdict rather than in the context of Rule 702 and *Daubert*. *See id.* The Court has failed to find any decisions by the Fifth Circuit, subsequent to the *Lofton II* decision, in which *Havner* was applied in the context of Rule 702 and *Daubert*. Therefore, the

Court rejects Defendants' argument that *Havner* sets the standard for relevance in this context. The Court also rejects Defendants' argument that *Bostic* applies to all toxic tort cases as the *Bostic* court itself stated that their decision requiring proof of a doubling of the risk standard specifically applied to asbestos-related disease cases. *Bostic*, 439 S.W.3d at 350 (emphasis added). Moreover, the Court rejects Defendants' argument that the doubling of the risk standard is the standard under "federal common law" and the default standard under *Daubert*. (*See* Defs.' Omnibus Reply 20, ECF No. 218, citing *Daubert II*, 43 F.3d at 1320; *Pozefsky*, 2001 WL 967608, at *3). The precedent cited by Defendants applies the doubling of the risk standard in cases involving the substantive law on causation and, accordingly, the Court is persuaded by the *Lofton II* court's holding that the doubling of the risk standard does not apply in the context of Rule 702 and *Daubert*. (*See id.*; *see also Daubert II*, 43 F.3d at 1320 (birth defect case considering Rule 702 and motion for summary judgment and applying California substantive law on causation); *Pozefsky*, 2001 WL 967608, at *1-7 (breast implant case where, after granting in part and denying in part a motion for summary judgment, the court excluded the plaintiff's experts because the evidence and opinions offered were unreliable as compared to the defendant's evidence demonstrating a lack of a causal relationship between breast implants and systemic injuries)).

The Court further rejects all of Defendants' arguments as to the reliability and relevance of Plaintiffs' experts' opinions as challenged under state law. "Although state law governs the substance of the case, whether an individual is qualified to testify as an expert is a question of federal law." *Charles*, 2015 WL 808417, at *2 & n.3 ("The parties' briefing of admissibility of expert testimony under Texas law is therefore inapposite."). Finally, the Court rejects all of Defendants' arguments which concern whether Plaintiffs' experts have provided "sufficient" evidence to meet the ultimate burden of proof on causation because *Daubert* does not require this

Court to determine whether causation evidence is sufficient as a matter of law. While the Court is making this finding as to the current Rule 702 and *Daubert* challenge, the Court is not making a determination as to whether *Havner* applies for purposes of dispositive motions that are not currently before this Court.

C. The Relevant Scientific Literature for Dr. Cantilena, Dr. Mills, and Dr. Rusyniak

Dr. Cantilena, Dr. Mills, and Dr. Rusyniak rely on various studies in their attempt to set forth reliable evidence on whether DMAA can cause hyperthermia in the general population and whether DMAA caused hyperthermia in Decedent. Therefore, the Court briefly reviews the relevant scientific literature relied upon by these experts to form their opinions on causation.

1. The Bloomer Studies[11]

a. *Effects of 1,3-Dimethylamylamine and Caffeine Alone or in Combination on Heart Rate and Blood Pressure in Healthy Men and Women*[12]

In this study, Dr. Bloomer and his group sought to "determine the effect of [DMAA] intake at [two] different dosages of practical relevance, with and without the addition of caffeine, on [heart rate] and blood pressure." (Defs.' Ex. V 4, ECF No. 223). The study also included a caffeine-only condition for comparative purposes. (*See id.*) Each of the ten subjects, five men and five women, received one serving of each condition and was observed during a two hour period after ingestion. (*See id.*) The subjects reported for five test days in a ten hour fasted state and were instructed not to exercise twenty-four hours prior to the test. (*See id.*) Heart rate, systolic blood pressure, and diastolic blood pressure were measured pre-condition and at time

[11] The parties refer to several studies performed by the University of Memphis as the "Bloomer studies." (*See* Dr. Cantilena Mot. 20, ECF No. 184; Pls.' Omnibus Resp. 40 n.104, ECF No. 200; *see also* Defs.' Ex. V, ECF No. 223).

[12] Richard J. Bloomer et al., *Effects of 1,3-Dimethylamylamine and Caffeine Alone or in Combination on Heart Rate and Blood Pressure in Healthy Men and Women*, 39 Physician & Sportsmed. 111 (2011). (Defs.' Ex. V 3-12, ECF No. 223).

intervals of thirty, sixty, ninety, and one hundred twenty minutes post-ingestion. (*See id.* at 5). The conditions included two hundred fifty milligrams of caffeine, fifty milligrams of DMAA, seventy-five milligrams of DMAA, fifty milligrams of DMAA plus two hundred fifty milligrams of caffeine, and seventy-five milligrams of DMAA plus two hundred fifty milligrams of caffeine. (*See id.*)

The study indicated that oral DMAA use resulted in an increase in systolic blood pressure, diastolic blood pressure, and rate pressure product. (*See id.* at 7). The study further indicated that fifty milligrams of DMAA plus two hundred fifty milligrams of caffeine did not have as significant of an increase on systolic blood pressure or diastolic blood pressure as compared to seventy-five milligrams of DMAA alone. (*See id.* at 11). The study concluded that acute oral ingestion of DMAA results in a significant increase in blood pressure without impacting heart rate. (*See id.*) It also concluded that the effect seems to be dose dependent, in particular on systolic blood pressure, with greater blood pressure increases with seventy-five milligrams of DMAA versus fifty milligrams of DMAA. (*See id.*) Finally, the study concluded that the addition of caffeine to fifty milligrams of DMAA caused a percentage change in rate pressure product but did not increase the other measures in a statistically significant manner. (*See id.*)

b. *Effect of Caffeine and 1,3-Dimethylamylamine on Exercise Performance and Blood Markers of Lipolysis and Oxidative Stress in Trained Men and Women*[13]

This study investigated "the effect of caffeine and [DMAA] on exercise performance and blood markers of lipolysis and oxidative stress in a sample of exercised-trained men and women." (*Id.* at 14). In this study, twelve subjects, six men and six women, reported to the

[13] Richard J. Bloomer et al., *Effect of Caffeine and 1,3-Dimethylamylamine on Exercise Performance and Blood Markers of Lipolysis and Oxidative Stress in Trained Men and Women*, 1 J. Caffeine Res. 169 (2011). (Defs.' Ex. V 13-22, ECF No. 223).

laboratory on four test days in a ten hour fasted state and were instructed not to exercise twenty-four hours prior to the test. (*See id.*) Soon after arriving to the laboratory, heart rate, systolic blood pressure, and diastolic blood pressure were measured and a blood sample was drawn. (*See id.*) Sixty minutes after ingestion of the condition, the study participants underwent a second round of testing and began a ten kilometer run. (*See id.*) The same measures were taken five minutes and thirty minutes after the run ended. (*See id.*) The tests were performed once per week for four weeks. (*See id.*) Running temperatures ranged from 44°F to 68°F. (*See id.*) The conditions in this study were a placebo (thirty grams of carbohydrate), caffeine (thirty grams of carbohydrate plus caffeine at a dose of four mg/kg body mass^{-1}), DMAA (thirty grams of carbohydrate plus DMAA at a dose of one mg/kg body mass^{-1}), or caffeine and DMAA (thirty grams of carbohydrate plus caffeine at a dose of four mg/kg body mass^{-1} and DMAA at a dose of one mg/kg body mass^{-1}). (*See id.*)

The study found

> that (1) ingestion of caffeine or [DMAA] alone or in combination [did] not improve exercise performance as measured by run time; (2) ingestion of [DMAA] results in the greatest increase in postexercise glycerol and [free fatty acids] concentrations; (3) caffeine or [DMAA] alone or in combination [did] not differently effect oxidative stress biomarkers pre- or postexercise; (4) caffeine and [DMAA] alone increase [systolic blood pressure].

(*Id.* at 16). The study also found that there was no statistically significant effect on run time when comparing the conditions versus placebo. (*See id.* at 17). The study demonstrated that while there was a higher systolic blood pressure with caffeine alone and DMAA alone compared to placebo, the combination of caffeine and DMAA did not result in a higher systolic blood pressure compared to placebo. (*See id.* at 19). This study noted that although the *Effects of 1,3-Dimethyamylamine* study, discussed above, demonstrated there was an additive effect for the combined condition of caffeine and DMAA, this study did not reach the same findings.

(*See id.* at 7, 11, 19).

c. *Safety Profile of Caffeine and 1,3-Dimethylamylamine Supplementation in Healthy Men*[14]

The present study tested caffeine and DMAA alone and in combination at dosages that are used in dietary supplements, for a period of twelve weeks, and performed tests for body mass and composition, resting respiratory rate, electrocardiography, urinalysis with microscopic examination, and oxidative stress, inflammatory, and cardiac biomarkers. (*See id.* at 24). This study had sixty-six participants but only fifty men completed the study. (*See id.*) Three testing days occurred in this study: pre-condition, after six weeks, and after twelve weeks. (*See id.* at 25). The participants arrived following an eight hour fast. (*See id.*) On arrival, participants turned in a diet log, voided, had an electrocardiography test, had their respiratory rate, blood pressure, and body mass and composition measured, and had a blood sample drawn. (*See id.*) The four conditions in the study were placebo (cellulose), two hundred and fifty milligrams of caffeine, fifty milligrams of DMAA, and two hundred and fifty milligrams of caffeine plus fifty milligrams of DMAA. (*See id.*) The participants were to consume one capsule per day during the first week and were then to consume two capsules per day for the remaining weeks; however, no capsules were consumed on the morning of a test day. (*See id.*)

The investigators found that "[twelve] weeks of supplementation with caffeine and DMAA, whether alone or in combination, [did] not result in statistically significant changes in any measured variables." (*Id.* at 26). The study concluded that the data is specific to "a sample of healthy, young men consuming daily dosage of caffeine [two hundred fifty milligrams] and DMAA [fifty milligrams] that are considered moderate and recommended." (*Id.* at 31-33).

[14] Richard J. Bloomer et al., *Safety Profile of Caffeine and 1,3-Dimethylamylamine Supplementation in Healthy Men*, 32 Hum. & Experimental Toxicology 1126 (2011). (Defs.' Ex. V 13-22, ECF No. 223).

d. *Hemodynamic and Hematologic Profile of Healthy Adults Ingesting Dietary Supplements Containing 1,3-Dimethylamylamine and Caffeine*[15]

In this study, the investigators "sought to determine the hemodynamic and hematologic profile of two different dietary supplements containing [DMAA and caffeine] before and after two weeks of daily intake." (*Id.* at 35). The investigators hypothesized that acute intake would cause an increase in blood pressure but chronic intake would not cause significant changes. (*See id.*)

Thirteen people participated in the study and took two servings of OxyElite Pro or Jack3d. (*See id.* at 36). Of the thirteen participants, four men and two women took OxyElite Pro and seven men took Jack3d. (*See id.*) When subjects arrived for testing, they rested for ten minutes and then had their heart rate, systolic blood pressure, and diastolic blood pressure readings taken along with a blood sample. (*See id.*) Subjects then ingested a condition and had heart rate and blood pressure measurements taken at thirty, sixty, ninety, and one hundred twenty minutes post-ingestion. (*See id.*) The subjects reported to the laboratory between 6:00 am and 9:00 am on two days for testing and were told not to exercise twenty-four hours prior to testing. (*See id.* at 35-36).

The investigators stated that their study demonstrated that "acute ingestion of the tested dietary supplements result[] in an increase in myocardial work (as measured specifically by [systolic blood pressure])." (*Id.* at 42). As to the results of the chronic portion of their study, the investigators stated that there was "no increase in resting [heart rate] or blood pressure . . . nor are bloodborne variables negatively impacted with either supplement." (*Id.* at 42-43). The study concluded that use of OxyElite Pro and Jack3d "[did] not elevate resting [heart rate, systolic

[15] Tyler M. Farney et al., *Hemodynamic and Hematologic Profile of Healthy Adults Ingesting Dietary Supplements Containing 1,3-Dimethylamylamine and Caffeine*, 2012 Nutrition & Metabolic Insights 1 (2012). (Defs.' Ex. V 34-45, ECF No. 223).

blood pressure, diastolic blood pressure, and rate pressure product] when ingested daily for [fourteen] days." (*Id.* at 44). Additionally, the study concluded that acute ingestion increased systolic blood pressure but did not have statistically significant increases in diastolic blood pressure or rate pressure product. (*See id.*)

e. *A Finished Dietary Supplement Stimulates Lipolysis and Metabolic Rate in Young Men and Women*[16]

This study tested six men and six women in order to determine "the acute effects of [OxyElite Pro] on blood markers of lipolysis, as well as metabolic rate." (*Id.* at 47). In this study, subjects reported to the laboratory on two occasions, separated by three or four days, between 6:00 am and 9:00 am. (*See id.* at 48). Subjects received placebo (cellulose) or OxyElite Pro. (*See id.*) Subjects came to the laboratory in a ten hour fasted state and were asked not to exercise twenty-four hours prior to testing. (*See id.*) After ten minutes of rest, the participants had their heart rate, systolic blood pressure, and diastolic blood pressure measured and a blood sample was drawn. (*See id.*) After the condition was ingested, subjects were measured again at thirty, sixty, ninety, and one hundred twenty minutes post-ingestion. (*See id.*) Blood collections occurred at the sixty and one hundred twenty minute time points. (*See id.*)

The investigators concluded that two capsules of OxyElite Pro resulted in an increase in plasma glycerol and free fatty acids as well as metabolic rate. (*See id.* at 53). They also noted that there was an increase in heart rate and blood pressure. (*See id.*)

[16] Cameron G. McCarthy et al., *A Finished Dietary Supplement Stimulates Lipolysis and Metabolic Rate in Young Men and Women*, 2012 Nutrition & Metabolic Insights 23 (2012). (Defs.' Ex. V 46-54, ECF No. 223).

f. *Biochemical and Anthropometric Effects of a Weight Loss Dietary Supplement in Healthy Men and Women*[17]

The purpose of this study was to determine the effects of OxyElite Pro "on weight loss and associated markers following an eight week intervention, using a randomized, placebo controlled, double blind design." (*Id.* at 56). The investigators hypothesized "that subjects in the supplement group would experience more favorable changes in weight loss and associated parameters compared to subjects in the placebo group." (*Id.*) In this study, thirty-two subjects reported to the laboratory on two occasions between 5:00 am and 11:00 am in a ten hour fasted state. (*See id.* at 56-57). After ten minutes of rest, the participants had their heart rate, systolic blood pressure, and diastolic blood pressure measured and a blood sample was drawn. (*See id.* at 57). Subjects had their height, weight, waist and hip circumference, skinfold thickness, and body composition measured. (*See id.*) Subjects were randomly assigned placebo or OxyElite Pro. (*See id.*) Sixteen subjects, eight men and eight women, were assigned to the two conditions. (*See id.*) They were required to ingest one capsule per day and, starting on day four, were allowed to ingest a second capsule. (*See id.*) They were asked not to perform any exercise two days prior to each test day. (*See id.* at 58).

The investigators found that OxyElite Pro "may assist in weight and body fat loss, while improving selected markers of the blood lipid panel." (*Id.* at 59). They noted that "the supplement [did] not result in any adverse effects pertaining to bloodborne markers of safety (eg, liver function)." (*Id.* at 60). The investigators further stated that "the supplement [did] result in an increase in resting heart rate of approximately six beats per minute[,] [a]lthough this increase in heart rate was not accompanied by a significant increase in systolic or diastolic blood pressure

[17] Cameron G. McCarthy et al., *Biochemical and Anthropometric Effects of a Weight Loss Dietary Supplement in Healthy Men and Women*, 2012 Nutrition & Metabolic Insights 13 (2012). (Defs.' Ex. V 55-64, ECF No. 223).

. . . ." (*Id.*)

g. *Physiological and Pharmacokinetic Effects of Oral 1,3-Dimethylamylamine Administration in Men*[18]

The Schilling study sought to "determine the pharmacokinetic profile of a single [twenty-five milligram] oral dosage of DMAA alone through [twenty-four] hours post-ingestion." (*Id.* at 66). The study recruited eight men to participate. (*See id.*) The subjects reported in the morning following an eight hour fast, were asked not to use any dietary supplements containing DMAA for seventy-two hours prior to testing and to refrain from exercise thirty-six hours prior to testing. (*See id.*) The subjects had their resting heart rate and blood pressure measured, cutaneous temperature measured via forehead, and a blood sample drawn. (*See id.* at 66-67). Subjects then received a twenty-five milligram dose of DMAA and the same measurements were taken over a twenty-four hour period at intervals of fifteen minutes, thirty minutes, forty-five minutes, one hour, one and one half hours, two hours, two and one half hours, three hours, four hours, five hours, six hours, eight hours, twelve hours, and twenty-four hours. (*See id.* at 67). Subjects remained in the laboratory for the first eight hours of testing and were given meals after the three and six hour mark, between the eight and twelve hour mark, and between the twelve and sixteen hour mark. (*See id.*) The subjects were instructed to have minimal physical activity and return to the laboratory eight hours fasted for the last blood draw. (*See id.*)

The investigators noted that one subject had extremely high levels of DMAA in his blood and, therefore, his data was excluded from the results. (*See id.* at 68-69). The investigators stated that "[t]he most important finding in this investigation is the relatively low plasma concentrations of DMAA corresponding to the [twenty-five milligram] oral dose, and the lack of

[18] Brian K. Schilling et al., *Physiological and Pharmacokinetic Effects of Oral 1,3-Dimethylamylamine Administration in Men*, BMC Pharmacology & Toxicology, Oct. 2013, at 1. (Defs.' Ex. V 65-74, ECF No. 223).

meaningful physiologic effects associated with the single dose." (*Id.* at 70). The study determined that DMAA has a longer half-life than caffeine. (*See id.* at 71). The authors indicated that "[w]hile a significant increase in temperature at [twelve] hours post-ingestion is noted in our data, *the values are still within normal range* of 36.1 to 37.8°C, *suggesting little meaningful effect is present*." (*Id.* (emphasis added)) The authors speculated that the increase in temperature "is likely attributable to the fact that subjects were out of the laboratory and reported back for testing, and that activity associated with leaving and returning to the laboratory slightly [their] elevated temperature." (*Id.*) They stated that "[f]urther study of DMAA effects on temperature in the context of exercise and heat exposure is warranted." (*Id.* at 72). On blood pressure, the study found that blood pressure values were "within normal clinical ranges." (*Id.* at 73). The investigators concluded that "it appears that the concern over adverse health-related effects of DMAA is specific to the dosage ingested by the individual." (*Id.*) The investigators further concluded "our data indicate minimal to no change in heart rate, blood pressure, or body temperature, and no adverse effects were noted." (*Id.*)

h. *Impact of a Dietary Supplement Containing 1,3-Dimethylamylamine on Blood Pressure and Bloodborne Markers of Health: A 10-Week Intervention Study*[19]

In this study, the authors sought to "extend our prior findings related to the use of [DMAA] in a combined product by using a [ten] week intervention trial to determine the change in selected markers of health and safety in a sample of healthy men." (*Id.* at 76). Here, there were thirty men who were instructed to maintain their current exercise routine and diet but were asked to refrain from exercise forty-eight hours prior to coming to the laboratory. (*See id.*) The subjects reported to the laboratory in the morning following an eight hour fasted state.

[19] Paul N. Whitehead et al., *Impact of a Dietary Supplement Containing 1,3-Dimethylamylamine on Blood Pressure and Bloodborne Markers of Health: A 10-Week Intervention Study*, 2012 Nutrition & Metabolic Insights 33 (2012). (Defs.' Ex. V 75-81, ECF No. 223).

(*See id.* at 77). After resting for ten minutes, subjects had their heart rate and blood pressure measured and a blood sample was drawn. (*See id.*) Subjects were assigned a placebo powder or Jack3d and were instructed to consume one to three servings on each workout day, thirty minutes prior to their exercise. (*See id.*) Subjects had a mean of four workout days per week and did not consume the supplement on non-workout days. (*See id.*) Twenty-five subjects completed the testing and were included in the results. (*See id.*)

The investigators found that DMAA "[did] not significantly increase resting heart rate or blood pressure (although systolic blood pressured increased [approximately six millimeters of mercury] with the supplement." (*Id.* at 79). They further found that "the supplement [did] not adversely impact bloodborne biomarkers of health." (*Id.*) They note, however, that "additional well-designed experiments of similar scope, inclusive of larger sample sizes, are needed to extend the findings presented within. It is only through such work that our ability to generalize these findings to the population at large will be possible." (*Id.*) The investigators concluded that "a dietary supplement contained [DMAA] consumed for a period of [ten] weeks [did] not result in a statistically significant increase in resting heart rate or blood pressure in a sample of healthy men, nor [did] the supplement negatively impact bloodborne biomarkers of health." (*Id.* at 81).

2. The Marsh Study[20]

This study was primarily conducted on dogs in order to determine the pharmacology of various compounds and their effects on blood pressure. (*See* Pls.' Ex. R-14 2, ECF No. 196-38). Sixty-three adult dogs were anesthetized and injected with various compounds tested; five dogs per compound. (*See id.*) Seven unanesthetized dogs received one compound every four days. (*See id.* at 2-3). Records of pithed dogs were also measured. (*See id.* at 3). The investigators

[20] David F. Marsh, *The Comparative Pharmacology of the Isomeric Heptylamines*, 94 J. Pharmacology & Experimental Therapeutics 225 (1948). (Pls.' Ex. R-14, ECF No. 196-38).

also studied tissue segments from five rabbits. (*See id.* at 6).

As a result of the Marsh study, an "epinephrine-agent equivalence" table was created. (*Id.* at 3-4). This study examined the amount of epinephrine it took in each type of dog to have the same blood pressure increases. (*See id.* at 3). The study reported that DMAA was the most potent of the compounds tested. (*See id.* at 5).

The study also tested DMAA, one of three of the most active agents, in one human participant, two and one half hours after a morning meal. (*See id.* at 6). The investigators measured blood pressure and heart rate every fifteen minutes for three hours while the subject remained sitting. (*See id.*) The dose given was two milligrams per kilogram in an individual who weighed seventy-nine kilograms. (*See id.*) For DMAA, "blood pressure slowly rose from a normal of 112/68 (70) to 134/90 in [ninety] minutes after the ingestion of [DMAA]." (*Id.*) The study found that blood pressure and heart rate returned to normal after one hundred fifty to one hundred eighty minutes. (*See id.* at 7). The study noted that a three milligram per kilogram dose was not well tolerated. (*See id.*) The study found that DMAA had a long duration of action similar to amphetamine. (*See id.*) The study concluded that DMAA had pressor action that "is about 1/200 as active as epinephrine." (*Id.* at 8). Additionally, the study stated that "[o]rally, in man, the [compounds] have but little pressor action." (*Id.*)

3. The Coyle Study

The Coyle study was "designed to determine whether manipulation of plasma catecholamine levels during exercise could alter [cutaneous vascular conductance] and subsequently core temperature." (Pls.' Ex. R-25 3, ECF No. 197-2). The study was also designed "to determine whether adrenaline or glucose infusion can alter the decline in stroke volume, cardiac output[,] or blood pressure that we have observed previously during exercise with concomitant dehydration." (*Id.*)

In this study, epinephrine was intravenously infused along with saline in one trial in a dose of 0.1 micrograms per kilogram per minute. (*See id.*) In another trial, only saline was infused. (*Id.*) In the third trial, glucose was infused. (*See id.*) The authors hypothesized "that levels of circulating catecholamines achieved with our treatments (i.e. glucose infusion < saline infusion < adrenaline infusion) will result in corresponding differences in [cutaneous vascular conductance] and core temperature." (*Id.*)

The study included seven cyclists who were acclimated to the heat in the testing conditions by cycling at least four times within ten days. (*See id.*) The environment was set at approximately 33.1°C.[21] (*See id.*) The subjects were intravenously infused during their exercise with the conditions, however, when they were administered epinephrine, they exercised for only one hundred twenty minutes rather than one hundred fifty minutes to prevent heat injury. (*See id.*) The subjects reported in a euhydrated and twelve hour fasted state and were given the same diet forty-eight hours prior to testing. (*See id.*) The subjects were fitted with a heart monitor and had catheters inserted into their arms for blood sampling and infusion. (*See id.*) The subjects entered the environmental chamber and instruments were attached to measure blood flow. (*See id.*) After fifteen minutes of rest, temperatures were recorded and a blood sample was drawn before exercise began. (*See id.*) While the subjects were exercising, heart rate, temperature, oxygen uptake, cardiac output, blood pressure, and blood flow were measured. (*See id.*)

The study found that epinephrine caused "a rapid and maintained decrease" in cutaneous blood flow and cutaneous vascular conductance, a 0.3-0.4°C increase in oesophageal

[21] Dr. Coyle agreed that this was between approximately 90.1°F and 92.8°F. (*See* Dr. Coyle Dep. 104:4-9).

temperature,[22] and an increase in heart rate. (*Id.* at 10). The authors concluded that "these observations indicate that large increases in plasma catecholamine levels cause hyperthermia during exercise by vasoconstricting the skin. This suggests a possible role of adrenergic vasoconstriction of skin in causing hyperthermia during prolonged exercise in the heat." (*Id.* at 12).

4. The Department of Defense Study[23]

The Department of Defense conducted a study on DMAA after removing all DMAA-containing products from military bases. (See Pls.' Ex. R-16 4, ECF No. 196-40). The study contains three phases: 1) a literature review and survey of adverse events; 2) patient interviews and a review of FDA reported adverse events; and 3) a case control study on the association between DMAA and adverse health effects. (*See id.* at 7). In Phase I, the investigators reviewed forty adverse medical event reports and "reviewed the likelihood of DMAA being casually linked with the events using a Naranjo Scale methodology."[24] (*Id.*) The investigators also reviewed available case reports in the scientific literature, the Bloomer Studies, FDA warning letters, and literature on whether DMAA is naturally-occurring in geranium plants and oils. (*See id.* at 8-9). In Phase II, the Armed Forces Medical Examiner System reviewed autopsy data and fluid samples from service members who died from exercise and heat-related injuries. (*See id.* at 9). In Phase III, the authors reviewed responses of 1,789 soldiers to determine if DMAA-containing supplements caused various adverse injuries. (*See id.*) The study determined

[22] Dr. Coyle agreed that this was approximately a 0.6°F to a 1°F increase in temperature. (Dr. Coyle Dep. 163:22-168:6).

[23] Col. John Lammie et al., *Report of the Department of Defense 1,3 Dimethylamylamine (DMAA) Safety Review Panel* (2013). (Pls.' Ex. R-16, ECF No. 196-40).

[24] The study admits that there can be flaws with the use of this methodology because if any alternative potential causes exist, that cause becomes the accepted explanation. (*See* Pls.' Ex. R-16 22, ECF No. 196-40).

that the soldiers were no more likely to have used DMAA than controls with an adjusted odds ratio of 0.85. (*See id.*) There was a ninety-five percent confidence interval including 1.0, which means that the results were not statistically significant. (*See id.* at 9-10). The study stated that "[t]he existing evidence does not conclusively establish that DMAA-containing substances are causally-associated with adverse events." (*Id.* at 11). The study also stated that "a consistent theme among the studies is that DMAA use potentially affects cardiovascular function, just as other sympathomimetic stimulants." (*Id.*)

D. Defendants' Rule 702 and *Daubert* Challenges to Dr. Cantilena, Dr. Mills, and Dr. Rusyniak

Dr. Cantilena, Dr. Mills, and Dr. Rusyniak are Plaintiffs' experts on causation.[25] (*See* Pls.' Omnibus Resp. 19-20, ECF No. 200; Hr'g Tr. 103:5-13, Apr. 28, 2015, ECF No. 324). Although the Court is not tasked with determining whether the evidence offered through Plaintiffs' experts is sufficient to meet their ultimate burden of proof, Plaintiffs' experts must establish reliable methodology for their opinions on general and specific causation. "General causation is whether a substance is capable of causing a particular injury or condition in the general population." *Johnson*, 685 F.3d at 468 (quoting *Knight*, 482 F.3d 347, 351 (5th Cir. 2007)). "[S]pecific causation is whether a substance caused a particular individual's injury." *Id.* (quoting *Knight*, 482 F.3d at 351).

In this case, Dr. Cantilena and Dr. Mills offer opinions on general causation: that DMAA can cause hyperthermia in susceptible individuals. (*See* Pls.' Ex. HH ¶ 20, ECF No. 197-29;

[25] Plaintiffs use a large portion of their brief arguing the collective opinions of Dr. Cantilena, Dr. Mills, and Dr. Rusyniak. (*See* Pls.' Omnibus Resp. 19-31, ECF No. 200). The Court must, however, evaluate each expert individually to determine if their opinions are relevant and reliable.

Pls.' Ex. JJ ¶ 14, ECF No. 197-31). Dr. Cantilena and Dr. Rusyniak[26] offer opinions on specific causation: that DMAA was a substantial contributing factor in Decedent's hyperthermia and death.[27] (*See* Pls.' Ex. HH ¶ 21, ECF No. 197-29; Pls.' Ex. II ¶ 9, ECF No. 197-30).

1. Dr. Cantilena

Dr. Cantilena opined that DMAA is a sympathomimetic compound of the aliphatic amine class. (*See* Pls.' Ex. HH ¶ 8, ECF No. 197-29). He further opined that sympathomimetic compounds cause vasoconstriction and increased blood pressure, and that increased blood pressure is a surrogate marker for increased heat production. (*See id.* at ¶ 15; Dr. Cantilena Dep. 154:6-8; Hr'g Tr. 107:4-109:16, Apr. 27, 2015 PM, ECF No. 323). Dr. Cantilena connected DMAA to other sympathomimetics by stating that scientific literature showed DMAA demonstrates a biologically plausible mechanism of action that DMAA "can" cause adverse events or harm in susceptible individuals. (*See* Pls.' Ex. HH ¶¶ 15, 20, ECF No. 197-29; Pls.' Ex. QQ, ECF No. 197-38; Dr. Cantilena Dep. 46:2-6, 46:20-25, 99:3-21, 132:15-19; Hr'g Tr. 202:14-206:13, Apr. 27, 2015 AM, ECF No. 321; Hr'g Tr. 31:12-21, 35:6-23, Apr. 27, 2015 PM, ECF No. 323). He explained that the notion that certain individuals are susceptible to DMAA is based on outliers in the Bloomer studies,[28] which are individuals who

[26] Plaintiffs stated in their Omnibus Response and at the *Daubert* hearing that the "thrust" of Dr. Rusyniak's opinion relates to specific causation but he is qualified to testify about general causation. (*See* Pls.' Omnibus Response 55, ECF No. 200; Hr'g Tr. 103:8-14, Apr. 28, 2015, ECF No. 324). As to Dr. Rusyniak's differential diagnosis in his specific causation opinion, the Court will only consider the parties' arguments and Dr. Rusyniak's opinion on general causation as they pertain to Dr. Rusyniak's ability to rule in DMAA as a cause.

[27] Defendants argue that Decedent died of heat stroke rather than hyperthermia and that Plaintiffs have to show that the amount of DMAA that Decedent took more likely than not caused his core body temperature to go above 104°F. (*See* Defs.' Omnibus Reply 45, ECF No. 218). Although the Court is not considering the sufficiency of the evidence offered by Plaintiffs' experts, the Court notes that the cause of death listed in Decedent's autopsy is "hyperthermia." (*See* Defs.' Ex. W 3, 8-9, ECF No. 215-5).

[28] Dr. Cantilena identified two individuals out of ten in the *Effects of 1,3 Dimethylamylamine* study by Bloomer and three individuals out of twelve in the *A Finished Dietary Supplement* study by McCarthy. (*See* Dr. Cantilena Dep. 78:18-98:12; Hr'g Tr. 225:4-7, Apr. 27, 2015 AM, ECF No. 321; *see also* Defs.' Ex. V 3-12, 46-54, ECF No. 223).

had a more dramatic increase in systolic blood pressure than the mean. (*See* Dr. Cantilena Dep. 59:18-60:1, 78:18-98:12; Hr'g Tr. 209:4-212:24, 213:8-215:6, 217:8-219:13, 222:10-234:2, Apr. 27, 2015 AM, ECF No. 321; Hr'g Tr. 4:7-7:9, Apr. 27, 2015 PM, ECF No. 323). He also relied on the Schilling study to state that DMAA significantly increases cutaneous temperature as measured at the forehead, although forehead temperature likely underestimated core temperature by approximately 2°C. (*See* Pls.' Ex. HH ¶ 15, ECF No. 197-29; Hr'g Tr. 205:22-206:6, Apr. 27, 2015 AM, ECF No. 321).

To further confirm his theory of a biologically plausible mechanism of action, Dr. Cantilena relied on the Coyle study, measuring core temperature and cutaneous vasoconstriction from an infusion of epinephrine in seven humans, and the Marsh study, measuring the blood pressure effects of epinephrine and DMAA, among other compounds, injected into dogs. (*See* Pls.' Ex. QQ, ECF No. 197-38; Dr. Cantilena Dep. 99:3-21, 101:1-7, 262:15-263:9, 265:1-7; Hr'g Tr. 12:14-43:5, 107:7-108:23, 120:1-135:23, 143:21-154:22, Apr. 27, 2015 PM, ECF No. 323). Using these two studies, Dr. Cantilena converted the level of DMAA found in Decedent's antemortem blood to an epinephrine equivalent and determined that the converted level was close enough to the plasma levels of epinephrine found in the Coyle study. (*See* Hr'g Tr. 12:14-43:5, Apr. 27, 2015 PM, ECF No. 323). Although there was an approximately thirty percent difference between the values,[29] Dr. Cantilena opined that DMAA can increase core temperature, just as epinephrine did in the Coyle study, and he ultimately concluded that "DMAA can cause adverse events in susceptible individuals." (*See id.* at 35;

[29] After making the conversions and calculations based on the Coyle and Marsh studies, Dr. Cantilena found that the DMAA found in Decedent's antemortem blood was equivalent to 0.97 micrograms of epinephrine per liter. (Pls.' Ex. 7, *Daubert* hearing). Dr. Cantilena then compared 0.97 to 1.30 micrograms of epinephrine per liter, the converted and calculated plasma concentrations of epinephrine found in the subjects of the Coyle study. (Pls.' Ex. 7, *Daubert* hearing). His conversions and calculations concluded that 0.97 micrograms of epinephrine per liter is approximately equivalent to 1.30 micrograms of epinephrine per liter. (Pls.' Ex. 7, *Daubert* hearing).

Pls.' Ex. HH ¶ 15, ECF No. 197-29).

The Court recognizes that "[t]rained experts commonly extrapolate from existing data" and that "an expert may extrapolate data from studies of similar chemicals." *Johnson*, 685 F.3d at 460 (quoting *Joiner*, 522 U.S. at 146). However, "[s]everal post-*Daubert* cases have cautioned [against] leaping from an *accepted* scientific premise to an *unsupported* one." *Moore*, 151 F.3d at 279 (emphasis added) (collecting cases). "To support a conclusion based on such reasoning, the extrapolation or leap from one chemical to another must be reasonably and scientifically valid." *Id.* (collecting cases). That is because "nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence which is connected to existing data only by the *ipse dixit* of the expert. A court may conclude that there is simply too great of an analytical gap between the data and the opinion proffered." *Id.* at 277 (quoting *Joiner*, 522 U.S. at 146).

A close examination of Dr. Cantilena's opinion demonstrates that "there is simply too great an analytical gap between the data and the opinion proffered." *Id.* (quoting *Joiner*, 522 U.S. at 146). Plaintiffs state that a class effects theory would be "merely asserting that because DMAA is a sympathomimetic and because other sympathomimetics have been demonstrated to increase core body temperature leading to heat stroke, DMAA can also increase core body temperature leading to heat stroke." (Pls.' Omnibus Resp. 31, ECF No. 200). Plaintiffs, however, argue that their theory is that

> DMAA causes heat stroke because other sympathomimetics . . . have been proven to cause heat stroke through a mechanism of action that involves the exact same effects DMAA has been demonstrated to have (vasoconstriction and metabolic heat) in peer-reviewed, randomized clinical trials in humans. Further, that mechanism is not theoretical but has been clinically proven at comparable levels (accounting for potency and pharmacokinetics) to that found in [Decedent's] antemortem blood. It is not the class alone that connects DMAA to these other drugs but the shared mechanism of action while accounting for differences in potency and pharmacokinetics.

(*Id.*) While Plaintiffs argue that Dr. Cantilena's opinion offers more than a class effects theory, it is clear to the Court from Dr. Cantilena's Rule 26 Expert Report, his Supplemental Rule 26 Expert Report, his deposition testimony, and his *Daubert* hearing testimony that all he is asserting is a class effects theory. Furthermore, because the Court finds below that Dr. Cantilena's conversions and calculations are unreliable, Plaintiffs' argument that Dr. Cantilena accounted for differences in potency and pharmacokinetics to support his mechanism of action theory is ultimately tainted.

a. Class Effects

Dr. Cantilena's methodology demonstrates that he is extrapolating from the class effects of other sympathomimetics to form his opinion on DMAA without accounting for any of the differences in sympathomimetics. *See, e.g.*, *Wells v. SmithKline Beecham Corp.*, 601 F.3d 375, 377-80 (5th Cir. 2010) (rejecting expert testimony analogizing between drugs in the class of dopamine agonists and their ability to cause impulsive behaviors); *Huss v. Gayden*, 571 F.3d 442, 458-59 (5th Cir. 2009) (rejecting expert's class of chemical theory as to sympathomimetic drugs); *see also McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1244-46 (11th Cir. 2005) (rejecting expert's analogy between ephedrine and PPA for failing to show the reliability of each step of expert's analysis). Dr. Cantilena testified in his deposition that "it is very impossible for me to divorce DMAA from the *known class effect of sympathomimetics*" and later opined that "[i]f it is established that DMAA is sympathomimetic, there is more than adequate evidence to say *that sympathomimetics* are a known risk factor for drug-induced hyperthermia." (Dr. Cantilena Dep. 210:18-21, 211:15-18 (emphasis added)). Dr. Cantilena's opinion that DMAA, like other sympathomimetics, increases blood pressure, heart rate, rate pressure product, causes vasoconstriction, and increases cutaneous temperature, is similar to the expert's opinion excluded in *Huss*, which stated that sympathomimetics as a class tend to "rev the individual up."

Huss, 571 F.3d at 458-59. Dr. Cantilena even used the same phrase by stating that "[t]he Jack3d product has been shown to increase energy consumption by studies that were . . . conducted with this specific product, and so that sort of increased energy use, if you will, *revving up* the body's metabolism, result[s] in an increased heat generation." (Hr'g Tr. 204:15-20, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)).

Dr. Cantilena explained that his use of studies of other sympathomimetics to make conclusions that DMAA causes hyperthermia was based on "*a class effect*, a known pharmacologic situation." (Dr. Cantilena Dep. 210:25-211:8 (emphasis added)). He explained that "human data from Schilling and from McCarthy . . . supports what the molecule is expected to do by virtue of its pharmacologic category . . . an alpha agonist." (Hr'g Tr. 204:15-20, Apr. 27, 2015 AM, ECF No. 321). However, Dr. Cantilena also acknowledged that sympathomimetics can have dissimilar effects. (*See id.* at 136:13-15; 138:5-140:12). Given that Dr. Cantilena's opinion merely compares sympathomimetics as a class, without accounting for any differences, the Court concludes that his opinion is unreliably extrapolating from the class effects of sympathomimetics to reach his conclusion that DMAA can cause hyperthermia in susceptible individuals.

b. The Bloomer Studies

Dr. Cantilena relies on the Bloomer studies to provide a biologically plausible mechanism of action demonstrating that DMAA can cause hyperthermia in the general population. Although the Bloomer studies represent the majority, if not the totality, of clinical studies where participants took DMAA and had various health parameters measured, the studies lack a significant sample size which may affect the statistical significance of their outcomes. *See, e.g., Orthoflex, Inc. v. ThermoTek, Inc.*, 986 F. Supp. 2d 776, 801-02 (N.D. Tex. 2013) (citing *Overton v. City of Austin*, 871 F.2d 529, 544 (5th Cir. 1989) (Jones, J., concurring)

("Whether a given [test result] should be regarded as statistically significant must be determined on a case by case basis since the value signifying statistical significance is dependent upon sample size."). In fact, in the Whitehead study, *Impact of a Dietary Supplement Containing 1,3-Dimethylamylamine on Blood Pressure and Bloodborne Markers of Health: A 10-Week Intervention Study*, the authors stated

> Due to the fact that our sample size is small, additional well-designed experiments of similar scope, inclusive of larger sample sizes, are needed to extend the findings presented within. It is only through such work that our ability to generalize these findings to the population at large will be possible.

(Def.'s Ex. V. 79, ECF No. 223). Moreover, *all* of the Bloomer studies indicate that further studies need to be performed in order to better understand the effects of DMAA on humans. (*See id.* at 9-10, 19, 33, 44, 53, 63, 73, 81).

It is clear from Dr. O'Brien, Plaintiffs' expert biostatistician, that the scientific literature on DMAA presents insufficient data to conclude that DMAA is safe or that DMAA causes harm because the sample sizes are too small. (*See* Pls.' Ex. TT ¶¶ 5-9, ECF No. 197-41; Dr. O'Brien Dep. 32:25-33:3, 72:18-77-6, 78:19-81:25, 128:21-129:12). In line with Dr. O'Brien's testimony and the Court's concern with relating small study populations to the general population, Dr. Cantilena's Rule 26 Expert Report opined that "*no adequately powered study has ever been performed on DMAA to accurately assess the occurrence of uncommon or relatively rare adverse health outcomes in the general population*." (Pls.' Ex. HH ¶ 8, ECF No. 197-29 (emphasis added)). Dr. Cantilena admitted that the Bloomer studies do not conclude that DMAA causes harm in individuals. (*See* Dr. Cantilena Dep. 59:5-9, 60:20-23). In reference to the Schilling study, Dr. Cantilena testified that "it would be *really unrealistic to expect* that seven subjects in one trial *would truly characterize the entire population*." (Hr'g Tr. 219:4-10, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)). Thus, the Court finds that Dr. Cantilena fails to

reliably use scientific methods to reach his conclusions on a biologically plausible mechanism. *See Knight*, 482 F.3d at 354 ("Where an expert *otherwise reliably utilizes scientific methods* to reach a conclusion, the lack of textual support may go to the weight, not the admissibility of the expert's testimony." (emphasis added) (internal quotation marks and citations omitted)).

Moreover, the Court finds that it is improper for Dr. Cantilena to extrapolate from the Bloomer studies that DMAA causes hyperthermia in the general population because DMAA demonstrated an acute increase in systolic blood pressure in a small number of individuals. *See Anderson v. Bristol Myers Squibb Co.*, No. Civ. A. H-95-0003, 1998 WL 35178199, at *1 (S.D. Tex. Apr. 20, 1998) (holding that an expert may not use studies purporting to prove one issue in order to infer that the same study proves a different issue because the inferential process is subject to *Daubert*); *see also McClain*, 401 F.3d at 1246 ("As the court stated, '[e]vidence suggest[ing] that [a chemical] may cause ischemic stroke does not apply to situations involving hemorrhagic stroke. This is a 'leap of faith' supported by little more than the fact that both conditions are commonly called strokes.'" (quoting *Rider v. Sandoz Pharm. Corp.*, 295 F.3d 1194, 1202 (11th Cir. 2002)). The Bloomer studies simply do not make conclusions about DMAA causing hyperthermia. *See Huss*, 571 F.3d at 459 ("It is axiomatic that causation testimony is inadmissible if an expert relies upon studies or publications, the authors of which were themselves unwilling to conclude that causation had been proven." (collecting cases)). Dr. Cantilena admitted that he had not relied on any studies indicating that an increase in blood pressure related to an increase in cutaneous vasoconstriction in humans. (*See* Hr'g Tr. 108:20-23, Apr. 27, 2015 PM, ECF No. 323). Thus, Dr. Cantilena's attempt to go from vasoconstriction and increased blood pressure to increased heat production, thereby causing harm to susceptible individuals, ultimately has too many analytical gaps in the causal chain. Therefore, the Court finds that Dr. Cantilena is not exhibiting the rigors of science that he would use in the laboratory

but rather, he is attempting to use unscientific speculation to support his opinion.

c. Outliers

Dr. Cantilena's used outliers in the raw data from the *Effects of 1,3-Dimethyamlamine* study by Bloomer and the *A Finished Dietary Supplement* study by McCarthy to support his causation opinion. Dr. Cantilena admitted that these studies did not measure cutaneous vasoconstriction or core temperature in the subject participants. (*See* Hr'g Tr. 106:18-107:3, Apr. 27, 2015 PM, ECF No. 323). He also readily acknowledged that the outliers only demonstrate safety signals rather than a causal relationship between DMAA and hyperthermia in the general population. (*See* Dr. Cantilena Dep. 58:16:21, 74:10-19; Hr'g Tr. 58:3-11, 70:21-23, 75:19-77:7, Apr. 27, 2015 PM, ECF No. 323). Dr. Cantilena further testified that "I *never* make the statement that this is *proof of a definitive causal safety issue*." (Hr'g Tr. 90:6-7 (emphasis added)). He opined that outliers are "not proof that it is a true safety finding, but it's *suggestive* of a signal." (*Id.* at 89:113-14 (emphasis added)). Dr. Cantilena conceded that "[a]ssociation is the initial step in the attempt for causation." (Dr. Cantilena Dep. 47:18-19).

Despite his reliance on outliers, Dr. Cantilena was unable to explain how the outlier data related to the general population. He testified that the outliers demonstrated adverse hemodynamic outcomes "for those individuals" and that "to see if those individuals would be represented in [twenty] percent or [twenty-five] percent of a population taking the product . . . *would be an area of concern*." (Dr. Cantilena Dep. 150:19-151:3 (emphasis added)). He explained that he was not relying on statistical analysis to identify outliers and that he did "not believe a statistical definition [was] required in [this] setting as we don't require that in the context of drug review for safety signals." (Hr'g Tr. 62:6-17, Apr. 27, 2015 PM, ECF No. 323). While the outliers Dr. Cantilena relies on may represent safety signals that could be important in the regulatory context, the courtroom is not the place to develop the research necessary to create

a causal connection in the tort context. *See Allen*, 102 F.3d at 198 (a regulatory agency's "threshold of proof is reasonably lower than that appropriate in tort law, which 'traditionally makes more particularized inquiries into cause and effect'"). As Dr. Cantilena's outlier testimony demonstrates, a relationship merely suggestive of association is not scientifically reliable evidence of causation. *See id.* at 197.

When the Court asked Dr. Cantilena to explain what literature supports the use of outliers for his causation opinion, he discussed the difference between statistical definitions and clinically meaningful differences. (*See* Hr'g Tr. 67:9-69:25, Apr. 27, 2015 PM, ECF No. 323). He further explained that with small clinical trials, "[y]ou don't know the generalized ability through the whole world. It's probably not [twenty] percent, but because you had such a small sample size, it just happened to be [twenty] percent." (*Id.* at 69:16-19). When the Court again asked Dr. Cantilena to state what literature supported his opinion, he stated "there are guidances at FDA that sort of talk about, you know, safety." (*Id.* at 70:1-13). He also opined that "*the FDA would never accept such . . . a small sample to characterize the population*. But this is all we had." (Hr'g Tr. 218:24-219:3, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)). Despite this testimony, Plaintiffs ask the Court to find Dr. Cantilena's opinion reliable but the Court simply cannot oblige.

Furthermore, Dr. Cantilena failed to explain how Decedent is similar to the outliers and dissimilar to the remaining study population but admits that Decedent "has characteristics that are similar to both groups." (Dr. Cantilena Dep. 162:17-22). He testified that he could not use the raw data in isolation to reach the conclusion that Decedent was similar to the outliers rather than the other study participants. (*See id.* at 163:22-164:5). Of the five outliers that Dr. Cantilena recognized, three were female. (*See* Hr'g Tr. 228:25-232:6, Apr. 27, 2015 AM, ECF No. 321; Hr'g Tr. 6:15-7:9, Apr. 27, 2015 PM, ECF No. 323). Furthermore, the outliers

from the *A Finished Dietary Supplement* study by McCarthy used OxyElite Pro rather than Jack3d, the difference of which Dr. Cantilena found to be irrelevant because both substances contain DMAA. (*See* Hr'g Tr. 233:21-234:2, Apr. 27, 2015 AM, ECF No. 321; Hr'g Tr. 77:13-78:18, Apr. 27, 2015 PM, ECF No. 323). Additionally, the outliers from the *Effects of 1,3-Dimethylamylamine* study by Bloomer ingested seventy-five milligrams of DMAA plus two hundred fifty milligrams of caffeine, almost double the amount of DMAA ingested by Decedent, which is assumed to be forty milligrams. (*See* Hr'g Tr. 4:7-7:9, 81:17-82:6, Apr. 27, 2015 PM). The Court finds that these unexplained gaps further demonstrate the unreliability of Dr. Cantilena's opinion.

d. Conversions and Calculations

Even given Plaintiffs' argument that Dr. Cantilena asserts more than a class effects theory and assuming without deciding that Dr. Cantilena's conversions and calculations were timely submitted,[30] the Court finds that they are unreliable. It is clear that Dr. Cantilena's conversions and calculations do not stem from research conducted independent of this litigation but rather, they have been developed for the purpose of his testimony in this case.[31] *See Newton*, 243 F. Supp. 2d at 678 (citing *Daubert II*, 43 F.3d at 1317 ("One very significant fact to be considered is whether the experts are proposing to testify about matters growing naturally and directly out of research they have conducted independent of the litigation, or whether they have

[30] On the Friday before the *Daubert* hearing, Defendants filed a "Pre-Hearing Motion to Exclude Untimely Disclosures by Plaintiffs' Expert Louis Cantilena" (ECF No. 258), to challenge the timeliness of Dr. Cantilena's conversions and calculations that were produced on the Monday before the *Daubert* hearing. At the hearing, the Court made clear that it would hear testimony from Dr. Cantilena on his conversions and calculations, which were a subject set forth in the Court's Order, and would reserve ruling on Defendants Motion until a later time. (*See* Hr'g Tr. 199:7-200:13, Apr. 27, 2015 AM, ECF No. 321; Order 2, ECF No. 235). As the Court ultimately finds Dr. Cantilena's conversions and calculations to be unreliable, the Court will deny Defendants' Pre-Hearing Motion as moot.

[31] Although it is unclear when exactly Dr. Cantilena created his conversions and calculations, it appears to the Court that they may have been prepared sometime in December 2014 after he received the Coyle study. (Hr'g Tr. 12:22-17:7, Apr. 27, 2015 PM, ECF No. 323).

developed their opinions expressly for purposes of testifying.")). Moreover, this methodology has not been tested or subject to peer review. *See Daubert II*, 43 F.3d at 1318 ("Peer review and publication do not, of course, guarantee that the conclusions reached are correct . . . . But the test under *Daubert* is not the correctness of the expert's conclusions but the soundness of his methodology." (citation omitted)). Dr. Cantilena provides no indication that other experts in his field use similar methodologies to extrapolate between sympathomimetics and he pointed to no literature making these comparisons to validate his approach. *See id.* at 1317 ("That the testimony proffered by an expert is based directly on legitimate, preexisting research unrelated to the litigation provides the most persuasive basis for concluding that the opinions he expresses were 'derived by the scientific method.'"). Compounding the concern the Court has with Dr. Cantilena's development of his conversions and calculations solely for the purpose of this litigation, the conversions and calculations are supported merely by his assurances amounting to too large of an analytical gap to allow the trier of fact to hear this testimony.

In support of his conversions and calculations in translating from one sympathomimetic to another, Dr. Cantilena testified that

> [t]he pharmacologic principles and methodology are standard when you look at receptors specific actions for which there is known potency. Any drug reference will give you, for example, morphine equivalence, to allow you to transfer from one opiate analgesic to another based on relative potency, so that you don't change pain medicine and inadvertently overdose a patient, because the different molecule has a different potency. This is a [well-established] pharmacologic method that is actually used in clinical practice.

(Dr. Cantilena Dep. 134:24-135:10). He further explained that he had support from

> clinical teaching material from my lectures. It's also in handbooks of pharmacy, handbooks of pharmacotherapeutics. And the example was the translation to equal potent analgesics, based on morphine equivalence. That is, being able to compare different chemical molecules that have the same receptor effect, analogous to sympathomimetics, to be able to translate their differential potency.

(*Id.* at 190:17:191:14). Other than the morphine equivalence technique used in clinical situations

and Dr. Cantilena's assurances that unidentified literature exists on the topic, Dr. Cantilena provided no other literature to support his conversions and calculations from epinephrine to DMAA. Therefore, the Court finds that Dr. Cantilena's blanket assurances are an insufficient basis for the Court to accept regarding the reliability of his conversions and calculations. *See Moore*, 151 F.3d at 276 ("The expert's assurances that he has utilized generally accepted scientific methodology is insufficient." (citation omitted)).

Dr. Cantilena began his conversions and calculations with the Marsh study which measured blood pressure effects in barbitalized, unanesthetized, and pithed dogs following the injection of various compounds, including DMAA. (*See* Hr'g Tr. 15:15-16:4, 20:6-23:25, 119:12-123:18, Apr. 27, 2015 PM, ECF No. 323). Although animal studies are not per se inadmissible, the Fifth Circuit has "previously recognized the 'very limited usefulness of animal studies when confronted with questions of toxicity.'" *Johnson*, 685 F.3d at 463 (quoting *Allen*, 102 F.3d at 197). Moreover, "studies of the effects of chemicals on animals must be carefully qualified in order to have explanatory potential for human beings." *Id.* (quoting *Allen*, 102 F.3d at 197). Dr. Cantilena explained that he was being "very conservative" in choosing the epinephrine equivalence calculated in the Marsh study for barbitalized dogs. (*See* Hr'g Tr. 21:4-23:23, Apr. 27, 2015 PM, ECF No. 323). Given that he did not choose unanesthetized dogs because they have confounding factors that can have an effect on the results and that he declined to use pithed dogs because they are an unrealistic model, the Court finds that Dr. Cantilena was essentially left with barbitalized dogs by default. (*See id.* at 22:4-23:21, 123:2-18). While Dr. Cantilena stated that dog models are "the most used" by the FDA and that the dog model is still "valid," he provided no support for his extrapolation from the dog data to human data other than his assurances that literature existed on the subject. (*See id.* at 20:16-21:1, 126:25-127:18, 133:2-137:22). Moreover, even if it did exist, Dr. Cantilena freely admitted that

he did not rely on that material to form his opinion. (*See id.* at 135:5-136:11).

Dr. Cantilena then used the Coyle study, which injected epinephrine into seven cyclists exercising in a temperature-controlled chamber at 92°F, to find the blood levels of epinephrine that resulted in a change in body temperature. (*See id.* at 12:22-13:14, 144:3-8, Apr. 27, 2015 PM, ECF No. 323). Dr. Cantilena stated that "it really would have been ideal to have an infusion of DMAA and [perform] the same type of experiment, but for a lot of, you know, reasons, that was not feasible." (*Id.* at 13:22-24). Thus, Dr. Cantilena compared plasma concentrations found in the subjects in the Coyle study and blood levels of DMAA in Decedent, which Dr. Cantilena stated "removes all the bioavailability variables" from his analysis. (*See* Dr. Cantilena Dep. 139:14-22, 140:24-141:8). The Court finds that Dr. Cantilena ignored the fact that the Coyle study subjects were injected with epinephrine and that the dogs in Marsh were injected with epinephrine and DMAA, as compared to Decedent who took DMAA orally. The Court finds that Dr. Cantilena's extrapolation from the Coyle study to prove a causal relationship between DMAA and hyperthermia in the general population is based on nothing more than his *ipse dixit*, which is an insufficient basis for the Court to find that his opinion is reliable.

Moreover, the Court finds that Dr. Cantilena failed to explain how a thirty percent difference in the epinephrine equivalence of Decedent's DMAA blood levels impacted his conversions and calculations.[32] (*See* Hr'g Tr. 35:6-23, Apr. 27, 2015 PM, ECF No. 323). Dr. Cantilena does not explain why he chose 7.1 pmol ml^{-1} rather than 7.9 pmol ml^{-1}, which is the higher value in the range that caused the same temperature increase. Dr. Cantilena also failed to explain how the same temperature increase shown in the Coyle study, of approximately 1°F, would be the same as Decedent's temperature increase with a thirty percent lower level of

[32] As mentioned above, the values were 0.97 micrograms of epinephrine per liter and 1.30 micrograms of epinephrine per liter. (Pls.' Ex. 7, *Daubert* hearing).

epinephrine. Thus, the Court finds that Dr. Cantilena's conversions and calculations are "not an exercise in scientific logic but in the fallacy of *post-hoc propter-hoc* reasoning, which is as unacceptable in science as in law." *Black*, 171 F.3d at 308.

e. Case Reports

Finally, insofar as Dr. Cantilena used case reports to support his opinion, the Court finds this methodology to be unreliable. Dr. Cantilena's Rule 26 Expert Report cites to a variety of case reports including FDA MedWatch reports and a case study by Gee et al. to demonstrate evidence of causation. (*See* Pls.' Ex. HH 17, ECF No. 197-29; Pls.' Ex. R-1-R-9, R-26, ECF Nos. 196-26-196-33, 197-3). A review of the case reports indicate that some of them deal with products other than Jack3d, involve much higher doses of DMAA, and have injuries which are different than those that Decedent suffered. (*See, e.g.*, Pls.' Ex. R-3, ECF No. 196-27 (injured male took Jack3d, "Pro Performance AMP, and Mega Men Sport" and was diagnosed with hepatitis); Pls.' Ex. R-5, ECF No. 196-29 (injured party "[a]woke one morning with intense muscle aches" and was diagnosed with "hypokalemic myopathy"); Pls.' Ex. R-8, ECF No. 196-32 (injured male took seven scoops of Jack3d and had palpitations and weakness); Pls.' Ex. R-26, ECF No. 197-3 (explaining that three individuals who took pills or a powder containing various doses of DMAA and also drank alcohol experienced cerebral hemorrhaging)). Moreover, courts have rejected the use of case reports to form opinions on causation. *See Newton*, 243 F. Supp. 2d at 680 & n.11 ("The Fifth Circuit and many other courts have soundly rejected case reports as an acceptable basis for causation." (footnote omitted)); *Castellow v. Chevron USA*, 97 F. Supp. 2d 780, 787 (S.D. Tex. 2000). Even Dr. Cantilena stated that "individual case reports *would not be able to establish causation* and would be hypothesis-generating signals *requiring further confirmation*." (Dr. Cantilena Dep. 56:1-9 (emphasis added)). Therefore, the Court rejects Dr. Cantilena's opinion based on the cited case reports.

f. Dr. Cantilena's Second Supplemental Rule 26 Expert Report and Declaration

Despite the unreliability of Dr. Cantilena's opinion established above, he sets forth a theory discussing the quantity and properties of the ingredients that form OxyElite Pro and Jack3d in order to demonstrate that "without DMAA [Decedent's] mild dehydration would not have caused his hyperthermia or death."[33] (Dr. Cantilena Second Supplemental Report & Decl. ¶¶ 5-15, ECF No. 327-1). Assuming *arguendo* that his Second Supplemental Rule 26 Expert Report and Declaration is timely,[34] the Court finds that Dr. Cantilena's opinion is unreliable. After reviewing the ingredients and their quantities found in OxyElite Pro, Dr. Cantilena opines that "DMAA and caffeine account[] for a substantial part of the increase metabolic activity found in the McCarthy study, *A Finished Dietary Supplement*." (*Id.* at ¶ 9 (emphasis added)). Likewise, for Jack3d, Dr. Cantilena states that the other ingredients and their quantities are "not capable of causing vasoconstrictive reduction of heat dissipation and/or the heat generation caused by DMAA." (*Id.* at ¶ 11). However, the Court finds that Dr. Cantilena assumes, without providing any further scientific bases, that DMAA can cause "increased heat generation" and "cutaneous vasoconstriction causing hyperthermia and death," which the Court has already deemed unreliable. (*Id.* at ¶¶ 9, 11). Therefore, Dr. Cantilena's Second Supplemental Rule 26 Expert Report and Declaration should also be excluded.

g. Conclusion on General Causation

In conclusion, the Court finds that Dr. Cantilena's entire general causation opinion is unreliable. Dr. Cantilena failed to account for any of the differences in extrapolating between

[33] As mentioned above, Dr. Cantilena's Second Supplemental Rule 26 Expert Report and Declaration was filed on July 24, 2015. (ECF No. 327-1).

[34] Dr. Cantilena's Second Supplemental Rule 26 Expert Report and Declaration is dated May 1, 2015. (Dr. Cantilena Second Supplemental Report & Decl. 4, ECF No. 327-1). As the Court ultimately finds Dr. Cantilena's Second Supplemental Rule 26 Expert Report and Declaration unreliable, the Court will deny Defendants' Pre-Hearing Motion as moot. (*See* Def.'s Reply 3, 10-11, ECF No. 303).

the class effects of other sympathomimetics and DMAA. Moreover, he provides no reliable grounds to support his conclusion of a biologically plausible mechanism of action because the Bloomer studies' small sample sizes do not allow Dr. Cantilena to translate their results to the general population and the studies do not reach the same conclusions drawn by Dr. Cantilena. In addition, Dr. Cantilena's use of outliers is not reliable as he admitted that these outliers did not provide evidence of causation and would not translate to the general population. Furthermore, the biggest gap in Dr. Cantilena's testimony comes from his conversions and calculations, prepared for the purposes of litigation, which are ultimately only supported by his blanket assurances that literature exists on similar methodology. Turning to the *Daubert* factors, the Court finds that Dr. Cantilena's conversions and calculations were not tested or subject to peer review. Moreover, Dr. Cantilena failed to provide a rate of error or evidence that his conversions and calculations are generally accepted in the scientific community and, therefore, the Court finds that his conversions and calculations are entirely speculative. Additionally, Dr. Cantilena relies on animal studies without qualifying how they apply differently to humans. As admitted by Dr. Cantilena, his use of case reports cannot establish causation. Finally, Dr. Cantilena's Second Supplemental Rule 26 Expert Report and Declaration fail to provide reliable evidence that DMAA can cause "increased heat generation" and "cutaneous vasoconstriction causing hyperthermia and death." Dr. Cantilena's opinion must provide more than studies "showing correlation, accompanied by unrelated anecdotes and quasi-scientific musings about how a class of drugs affects the human body." *Huss*, 571 F.3d at 459. Based on the foregoing, the Court finds that Dr. Cantilena's entire general causation opinion is excluded.

h. Differential Diagnosis

Given the Court's finding that Dr. Cantilena's general causation opinion is excluded and, as discussed below, the Court's decision to exclude Dr. Mills' opinion and Dr. Rusyniak's

opinion on general causation, the Court further concludes that Dr. Cantilena cannot rule in DMAA as a potential cause in his differential diagnosis.[35] *See Johnson*, 685 F.3d at 468-69 (holding that the district court did not abuse its discretion in excluding an expert's differential diagnosis because the expert failed to satisfy general causation requirements). Without being able to rule in DMAA as a potential cause, Dr. Cantilena's differential diagnosis is unreliable. The *Johnson* court explained that "*Moore* illustrates that an expert may not rely on a differential diagnosis to circumvent the requirement of general causation." *Id.* at 468 (citing *Moore*, 151 F.3d at 278). Therefore, the Court rejects Dr. Cantilena's specific causation testimony.

2. Dr. Mills

Dr. Mills opined that DMAA is a sympathomimetic in the aliphatic amine class. (*See* Pls.' Ex. JJ ¶ 4, ECF No. 197-31; Dr. Mills Dep. 18:25-19:6; Hr'g Tr. 5:15-6:4, Apr. 28, 2015, ECF No. 324). He further opined that generally sympathomimetics cause vasoconstriction, increased heart rate and blood pressure, decreased gut motility, increased body temperature, and increased lipolysis, plasma glycerol, and free fatty acids. (*See* Pls.' Ex. JJ ¶ 4, ECF No. 197-31; Dr. Mills Dep. 28:24-29:12, 62:22-63:7, 155:2-256:8; Hr'g Tr. 5:15-6:4, Apr. 28, 2015, ECF No. 324). Dr. Mills maintained that DMAA can cause the same effects as other sympathomimetics based on its development as a nasal decongestant and various animal and human studies demonstrating a biologically plausible mechanism of action. (*See* Pls.' Ex. JJ ¶ 4, ECF No. 197-31; Dr. Mills Dep. 35:18-22, 135:6-22, 148:5-149:24; Hr'g Tr. 5:15-6:4, 23:4-

[35] A differential diagnosis is defined as "the determination of which one of two or more diseases or conditions a patient is suffering from, by systematically comparing and contrasting their clinical findings." *Dorland's Illustrated Medical Dictionary* 507 (32d ed. 2012). One court within this district has defined differential diagnosis as "the distinguishing of a disease or condition from others presenting similar symptoms." *Johnson v. United States*, No. EP-02-CA-580-PRM, 2005 WL 1605822, at *3 n.21 (W.D. Tex June 30, 2005) (citation omitted). Additionally, another court observed that a differential diagnosis is "accomplished by determining the possible causes for the patient's symptoms and then eliminating each of these potential causes until reaching one that cannot be ruled out or determining which of those that cannot be excluded is the most likely." *Johnson v. Arkema, Inc.*, 685 F.3d 452, 468 (5th Cir. 2012) (quoting *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 262 (4th Cir. 1999)).

18, Apr. 28, 2015, ECF No. 324).

Dr. Mills explained that sympathomimetics cause hyperthermia through a number of processes that include the stimulation of the sympathetic nervous system, the uncoupling of proteins, heat production, and heat dissipation through vasoconstriction. (*See* Pls.' Ex. JJ ¶¶ 6-10, ECF No. 197-31; Dr. Mills Dep. 121:21-122:1; Hr'g Tr. 5:15-7:23, 62:18-63:9, Apr. 28, 2015, ECF No. 324). He relied on the Schilling study to support his opinion that DMAA has hyperthermic properties based on those subjects' increase in forehead temperature, which was within normal limits, but he noted that no other study supported this position. (*See* Pls.' Ex. JJ ¶ 4, ECF No. 197-31; Dr. Mills Dep. 44:2-54:8, 55:6-14; 58:15:64:12, 79:22-80:7; Hr'g Tr. 6:13-7:23, 38:1-6, Apr. 28, 2015, ECF No. 324). He also relied on his education, training, and experience and his "knowledge of pharmacology that suggests that [DMAA] is . . . likely, very likely [a] sympathomimetic agent." (Dr. Mills Dep. 107:23-108:8).

Dr. Mills opined that "Jack3d and OxyElite are preparations containing DMAA, a drug that possesses the structural, functional, and pharmacologic properties that define the classical sympathetic neurotransmitters and their sympathomimetic acting agents. Like ingestion of other sympathomimetics DMAA can be a substantial contributing cause of hyperthermia." (Pls.' Ex. JJ ¶ 13, ECF No. 197-31). He explained "that based upon [DMAA's] sympathomimetic properties and the fact that in a peer-reviewed paper . . . [which] showed that a rise in body temperature can occur by DMAA, . . . I think DMAA poses a significant risk to the development of hyperthermia in susceptible individuals." (Dr. Mills Dep. 121:21-122:1). Dr. Mills concluded that "[c]onfirmed use of DMAA in mild to moderate temperature conditions with low humidity, moderate exercise and absence of use of anesthetics is a setting where DMAA can be a substantial contributing factor causing hyperthermia, related complications and death." (Pls.' Ex. JJ ¶ 4, ECF No. 197-31).

a. The Schilling Study

It is clear that the only clinical study specific to DMAA and body temperature that Dr. Mills relies on is the Schilling study. In assessing that study, Dr. Mills testified that the results showed a statistically significant increase in body temperature over the baseline temperature after twelve hours. (*See* Dr. Mills Dep. 47:18-48:2; Hr'g Tr. 6:13-7:23, 38:1-19, Apr. 28, 2015, ECF No. 324). While that increase was within normal body temperature limits and Dr. Mills indicated that the results do "[n]ot necessarily" represent an adverse medical event, he opined that "anything that increases body temperature to any degree can be a substantial risk factor for causing hyperthermia under certain conditions." (Dr. Mills Dep. 81:3-13). Furthermore, Dr. Mills opined that the core temperature of the subjects was likely higher than their forehead temperature. (*See* Hr'g Tr. 44:7-23, Apr. 28, 2015, ECF No. 324). Although the subjects were allowed to leave the laboratory for several hours before the final blood draw, Dr. Mills found it "remarkable" that there was still a rise in body temperature. (*See* Dr. Mills Dep. 49:15-21, 50:7-53:21, 59:16-64:12, 84:22-25, 86:1-18; Hr'g Tr. 39:14-41:22, 42:23-43:5, Apr. 28, 2015, ECF No. 324). Thus, Dr. Mills opined that the Schilling study supported his opinion on general causation. (*See* Dr. Mills Dep. 49:10:14, 51:3-53:21, 59:16-64:12, 84:13-21, 86:1-18; Hr'g Tr. 41:14-42:3, 46:14-17, 47:11-22, Apr. 28, 2015, ECF No. 324).

Dr. Mills disagreed with the authors' conclusions in the Schilling study that the change in body temperature was not "meaningful." (*See* Dr. Mills Dep. 45:7-54:8, 59:11-15, 76:19-79:6; Hr'g Tr. 33:21-35:7, 47:17-22, Apr. 28, 2015, ECF No. 324). He admitted that he was interpreting the data from the Schilling study in a way that differed from what the authors concluded. (*See* Hr'g Tr. 47:11-48:9, Apr. 28, 2015, ECF No. 324). Dr. Mills explained that he "thinks it's likely that DMAA would substantially raise the risk of exercise induced hyperthermia." (Dr. Mills Dep. 145:13-15). He further testified that the Schilling study

demonstrates that "taking DMAA gives rise to a substantial risk that in certain susceptible individuals, their body temperatures can increase." (*Id.* at 87:19-22). However the authors of the Schilling study stated that "[f]urther study of DMAA effects on temperature in the context of exercise and heat exposure is warranted." (Defs.' Ex. V 72, ECF No. 223).

Like Dr. Cantilena's use of several of the other Bloomer studies to make conclusions about the general population, Dr. Mills' use of the Schilling study also lacks a significant sample size to make causal conclusions about DMAA's ability to increase body temperature in the general population. Dr. Mills acknowledged Dr. O'Brien's testimony that the Schilling study does not provide a large enough sample size to make a conclusion as to whether DMAA elevates body temperature. (*See* Hr'g Tr. 58:20-23, Apr. 28, 2015, ECF No.; *see also* Dr. O'Brien Dep. 72:18-74:20). Dr. Mills further acknowledged that the Department of Defense study concluded that there was no relationship between DMAA and heat injuries. (*See* Hr'g Tr. 59:1-18, Apr. 28, 2015, ECF No. 324). Dr. Mills' only real attempt to justify his reliance on the Schilling study, despite the lack of a significant sample size, was his explanation that with "positive" scientific results "one can feel fairly confident . . . that [a] statistically significant event may have actually occurred" but when there are "negative" scientific results, the "small number of people" could create error. (Dr. Mills Dep. 56:3-19). He testified that more studies were needed to make conclusions about DMAA. (*See id.* at 55:2-5, 64:21-22, 68:23-69:20, 128:20-129:4, 138:11-23; Hr'g Tr. 56:2-5, 94:12-22, Apr. 28, 2015, ECF No. 324). When asked if the opinion he had formed about DMAA and body temperature was "more of a correlation than causation," he responded "I don't think in this case that matters." (Dr. Mills Dep. 65:4-6). In determining whether DMAA was the cause of hyperthermia, Dr. Mills opined that "would require a properly controlled, randomized, case controlled study, and that's difficult to do in humans with a suspected toxi[n]." (*Id.* at 144:20-145:3).

Only evidence demonstrating a causal relationship between a product and an alleged injury is admissible as relevant and reliable. *See Moore*, 151 F.3d at 274, 276; *see also Brumley*, 200 F.R.D. at 602. However, Dr. Mills does not opine that the Schilling study, or any other evidence, establishes a causal relationship which can be demonstrated in the general population. Instead, he relies on the Schilling study to opine that susceptible individuals can have increased body temperature. When the Court asked Dr. Mills how to determine who is susceptible to DMAA, he indicated that scientists do not know what causes people to have idiosyncratic responses. (*See* Hr'g Tr. 63:20-64:10, Apr. 28, 2015, ECF No. 324). He defined idiosyncratic as a "response [that] occurs rarely in individuals . . . with an underlying susceptibility factor that may or may not be understood yet." (Dr. Mills Dep. 157:11-18). Dr. Mills fails to explain how susceptible individuals relate to the general population. His failure to do so ultimately makes his use of the Schilling study and his opinion on DMAA's ability to increase body temperature unreliable.

Assuming *arguendo* that Dr. Mills' Supplemental Rule 26 Expert Report and Declaration is timely,[36] the Court addresses the reliability of his "statistical regression analysis" using the individual subject data from the Schilling study.[37] (Dr. Mills Supplemental Report & Decl. ¶ 6, ECF No. 327-22). While it appears to the Court that Dr. Mills' analysis of the data is "supportive" of his opinion "that changes measured at the forehead were caused by the ingestion of 25 mg of DMAA," this presumes the reliability of the Schilling study, which the Court has already found unreliable above. (*Id.*) Moreover, like Dr. Cantilena, Dr. Mills' "statistical

[36] Dr. Mills' Supplemental Rule 26 Expert Report and Declaration is dated April 30, 2015. (Dr. Mills Supplemental Report & Decl. 3 ECF No. 327-22). As the Court ultimately finds Dr. Mills' "statistical regression analysis" to be unreliable, the Court will deny Defendants' Pre-Hearing Motion as moot. (*See* Def.'s Reply 3, 10-11, ECF No. 303).

[37] As mentioned above, Dr. Mills' Supplemental Rule 26 Expert and Declaration was filed on July 24, 2015. (ECF No. 327-22).

regression analysis" has been developed solely for the purposes of his testimony in this case, further undercutting its reliability. *See Newton*, 243 F. Supp. 2d at 678 (citing *Daubert II*, 43 F.3d at 1317). Accordingly, the Court finds that Dr. Mills' Supplemental Rule 26 Expert Report and Declaration should be excluded.

b. Class Effects

Dr. Mills not only fails to bridge the gap in his extrapolation from the class effects of sympathomimetics to DMAA, he relies on animals studies using other sympathomimetics to form his opinion on DMAA. In discussing the Schilling study, it appears to the Court that Dr. Mills relied on the class effects of sympathomimetics. He testified that "I would *suspect* that they would have seen a rise in body temperature, as we do *in animals with sympathomimetics* . . . like DMAA[,] that we all agree . . . induce[s] vasoconstriction." (Hr'g Tr. 44:11-19, Apr. 28, 2015, ECF No. (emphasis added)). In further discussing the Schilling study, Dr. Mills stated that "it is likely that DMAA caused a metabolic heat production, which is the *effect of a class* of sympathomimetics, *all of which* induce metabolic rate and heat production, and also decrease heat dissipation and release from the body, because they induce vasoconstriction." (*Id.* at 7:4-10 (emphasis added)).

Dr. Mills further maintained that "MDMA is the prototypical sympathomimetic resulting in increased heat production, [which] . . . is *an effect that is shared by all drugs in its class*." (Pls.' Ex. JJ ¶ 9, ECF No. 197-31 (emphasis added)). He stated that "[t]he strongest demonstration of a molecular mechanism of sympathomimetic-induced hyperthermia was revealed by our work published . . . in 2003 showing that mice lacking the gene encoding [Uncoupling Protein 3] were profoundly resistant to hyperthermia and completely protected from death induced by MDMA." (*Id.*) Dr. Mills testified that "most" sympathomimetics pose safety risks for heat stroke and that "[t]hese are *class effects*." (Dr. Mills Dep. 146:23-148:4 (emphasis

added)). However, he further testified that not all sympathomimetics cause increased core body temperature, vasoconstriction, or hyperthermia. (*See id.* at 27:19-24, 71:15-22). Thus, it is clear that despite Dr. Mills' generalizations, there are many differences in sympathomimetics and their potential effects which Dr. Mills fails to account for in his opinion.

Dr. Mills defined sympathomimetics broadly, stating that sympathomimetics include "*any agent* that bears structural and functional / pharmacologic similarity with [norepinephrine and epinephrine]." (Pls.' Ex. JJ ¶ 4, ECF No. 197-31 (emphasis added)). He acknowledged that DMAA lacked an aromatic or benzene ring in its chemical structure. (*See* Dr. Mills Dep. 18:25-19:23; Hr'g Tr. 27:1-28:4, Apr. 28, 2015, ECF No. 324). He also opined that "[a]ny chemical structure difference *can cause* a variation either up or down in the efficacy potency of any agent on its receptor systems including sympathomimetics." (Dr. Mills Dep. 24:23-25:1 (emphasis added)).

Furthermore, Dr. Mills recognized that sympathomimetics have "class effects" as well as "distinctive properties." (*See id.* at 103:19-104:1). He testified that sympathomimetics have different chemical structures, dose-response relationships, and potency. (*See id.* at 15:22-16:6; Hr'g Tr. 21:6-24, 25:23-28:21, Apr. 28, 2015, ECF No. 324). When asked if he would want to take into account the distinctive properties of sympathomimetics in performing an analysis, Dr. Mills responded "[w]ell, that you could, you would want to do everything possible to understand, giving your findings' levels and opportunities to do so." (Hr'g Tr. 32:9-14, Apr. 28, 2015, ECF No. 324). Dr. Mills also opined that in order to compare sympathomimetics, "[y]ou have to do a *variety of studies* . . . to *fully characterize the sympathomimetic effects*." (Dr. Mills Dep. 28:5-12 (emphasis added)).

Dr. Mills also relied on class comparisons to support his opinion on dose-response and potency. He explained that there is data "on all sympathomimetics as a class" that "suggest[s]

that the [dose-response] relationships . . . are weak." (*Id.* at 156:25-157:9). Analogizing to ecstasy and MDMA, Dr. Mills stated that sympathomimetics do not have a clear dose-response. (*See* Hr'g Tr. 65:4-15, Apr. 28, 2015, ECF No. 324). He noted that no literature demonstrates a dose-response curve for DMAA and cutaneous vasoconstriction. (*See id.* at 21:18-22:23). He also explained that "[w]ith respect to DMAA especially and heat . . . that is also *a class effect* that is misunderstood by most of the sympathomimetics." (Dr. Mills Dep. 158:7-11 (emphasis added); Hr'g Tr. 63:13-65:15, Apr. 28, 2015, ECF No. 324).

Dr. Mills further opined that while case-controlled studies on DMAA would be helpful, "there's information out there to suggest that [DMAA] *can be* a substantial causing risk factor for hyperthermia." (Hr'g Tr. 65:24-66:2, Apr. 28, 2015, ECF No. (emphasis added)). He stated that "my opinion would be that it's a risk factor *because of its alpha-agonist activities* that are undisputed. It's a sympathomimetic. *Like all sympathomimetics*, they *could* lead to hyperthermia. Exercise increases that risk. I think this is a risk factor for hyperthermia and death." (*Id.* at 66:13-21 (emphasis added)). When asked if DMAA was only a potential risk, Dr. Mills agreed and stated it was "[t]he *same* with MDMA." (*Id.* at 66:25-67:3 (emphasis added)).

Despite all the differences that Dr. Mills acknowledged in his Rule 26 Expert Report, his deposition testimony, and his *Daubert* hearing testimony about sympathomimetics, Dr. Mills still attempted to compare sympathomimetics to DMAA to demonstrate that the same effects would occur in the general population. The Court cannot accept this leap in logic as support for his expert opinion. Dr. Mills lacks scientific literature as a basis for each step in his analysis as it relates to extrapolating from sympathomimetics as a class to the effects that DMAA has on the general population.

c. Animal Studies

The only information Dr. Mills relies on to show dose-response for DMAA is a dog study

which used a dose of six to ten milligrams per kilogram in a dog, as opposed to a human dose of approximately one half milligram per kilogram. (*See* Dr. Mills Dep. 39:20-40:15). Although he testified that "[t]he dog is the undisputed, gold standard model in thousands and thousands of papers and laboratories for human cardiovascular biology, and especially in terms of drug development and the predictive effects of toxicity in humans related to cardiovascular function and blood pressure," he presents no clear evidence other than his assurances. (Hr'g Tr. 10:17-21, Apr. 28, 2015, ECF No. 324).

Dr. Mills conceded that data from animal studies does not always extrapolate accurately to effects in humans. (*See* Dr. Mills Dep. 99:22-100:4, 101:11-102:2). Dr. Mills' mere assurances that dogs are a good model to predict human effects are insufficient to establish reliability and even so, his opinion on dose-response fails to account for the different doses used in animals and humans. *See, e.g.*, *Joiner*, 522 U.S. at 144-45 (holding that it was not an abuse of discretion to reject experts' reliance on animal studies where infant mice were injected with large doses of chemicals because the study was distinguishable from the facts presented in the litigation); *Gulf S. Insulation v. U.S. Consumer Prod. Safety Comm'n*, 701 F.2d 1137, 1146 (5th Cir. 1983) (rejecting rat study based on the small sample size, the large dose given to the rats, and the difficulty extrapolating the study results to humans).

d. Biologically Plausible Mechanism of Action

Insofar as Dr. Mills relies on a biologically plausible mechanism of action to form his opinion, the Court rejects that theory as well. Dr. Mills testified that dosage, time, route of administration, species, sex, and environmental conditions are important factors for toxicologists to review in determining the adverse effects of chemicals on living organisms. (*See* Dr. Mills Dep. 7:25-10:25, 112:21-113:18; Hr'g Tr. 12:7-11, 15:3-18:24, 28:17-21, Apr. 28, 2015, ECF No. 324). He further testified that while "those [factors] aren't required to conclude .

. . that a drug can cause hyperthermia, those [factors] are required to *understand how it is happening*." (Dr. Mills Dep. 71:23-72:7 (emphasis added)). He further opined that it was unnecessary to know the pharmacodynamics or pharmacokinetics of a drug to reach a causal conclusion but they are only necessary "*to understand how the drug caused the effect*." (*Id.* at 108:24-111:19 (emphasis added)). Moreover, when Dr. Mills was given the chance to discuss DMAA's biologically plausible mechanism of action to cause heat-related injuries, he instead chose to discuss how DMAA is a sympathomimetic and "has clear undisputed alpha-1-agonistic activity." (*See* Hr'g Tr. 5:9-6:4, Apr. 28, 2015, ECF No. 324).

The Court finds that there is a glaringly large gap in Dr. Mills' theory of a biologically plausible mechanism of action. Dr. Mills is extrapolating from other sympathomimetics, such as amphetamine, methamphetamine, ecstasy, and MDMA, and how they cause heat-related injury, to form his opinion that DMAA also causes heat-related injury. (Pls.' Ex. JJ ¶ 8, ECF No. 197-31). In making this comparison, Dr. Mills fails to examine the factors listed above which would allow him to form an opinion on how DMAA causes heat-related injury. Instead, Dr. Mills compares DMAA to other sympathomimetics and speculates that DMAA has the same biologically plausible mechanism of action. Dr. Mills fails to rely on scientific literature to explain the various factors mentioned above which would allow him to opine as to how DMAA acts on the human body or, in essence, DMAA's biologically plausible mechanism of action. Dr. Mills testified that there has been no study performed on DMAA demonstrating cutaneous vasoconstriction in humans and he agreed that he did not find any studies that indicated that DMAA caused hyperthermia. (*See id.* at 25:4-19, 32:24-33:2). Dr. Mills also agreed that no peer reviewed scientific study involved the measurement of core temperature after acute ingestion of Jack3d. (*See id.* at 69:12-17, 94:24-95:7). He further agreed that there were no studies on absorption rates of DMAA. (*See id.* at 64:11-13). Therefore, the Court finds that Dr.

Mills' theory of a biologically plausible mechanism of action is nothing more than pure speculation which the Court cannot allow to reach the trier of fact.

e. Conclusion on General Causation

The Court finds that Dr. Mills' opinion suffers from many of the same flaws as Dr. Cantilena's opinion and, therefore, should be excluded. Dr. Mills' opinion is full of contradictions and equivocations that ultimately weaken the value of his opinion and make his methodology unreliable. The Schilling study's small sample size, which Dr. Mills acknowledged, does not allow Dr. Mills to translate the results to the general population. He further admitted that whether the Schilling study demonstrated causation rather than correlation does not matter, yet, at the same time, he contradicts himself by requiring case controlled studies to show a causal relationship between DMAA and hyperthermia. Moreover, in using the Schilling study, Dr. Mills simply opined that DMAA could affect susceptible people without explaining who is susceptible and how that translates to the general population. Dr. Mills' "statistical regression analysis," which was created for his Supplemental Rule 26 Expert Report and Declaration, presumes the reliability of the Schilling study and is ultimately improper for that reason. Dr. Mills also acknowledged that the Department of Defense study found that there was no relationship between DMAA and heat injuries. Like Dr. Cantilena, Dr. Mills failed to account for any of the differences in extrapolating between the class effects of other sympathomimetics and DMAA. He further relies on animal studies for his opinion without qualifying how they apply differently to humans or accounting for the differences in dose between dogs and humans. Finally, Dr. Mills admittedly failed to rely on several important factors used to determine the adverse effects of chemicals on living organisms, which would have allowed him to determine a biologically plausible mechanism of action. Accordingly, the Court finds that Dr. Mills' assurances as to the reliability of his opinion are not enough to satisfy

the standard under *Daubert* and, therefore, his entire general causation opinion is excluded.

3. Dr. Rusyniak

In order to rule in DMAA as a potential cause, Dr. Rusyniak relied on his research, training, and experience, the scientific literature on DMAA, the opinion of Dr. Cantilena on the pharmacologic properties of DMAA, and the opinion of Dr. Mills on the hyperthermic properties of DMAA. (*See* Pls.' Ex. II ¶¶ 4-5, ECF No. 197-30; Dr. Rusyniak Dep. 12:2-4, 32:23-36:9, 38:2-39:4, 64:25-67:10; 86:23-87:17, 100:11-25, 102:15-103:8). In order to rule out other potential causes, Dr. Rusyniak reviewed Decedent's medical records, statements made by Benjamin Tuthill, Decedent's toxicology report, Decedent's autopsy, temperature, humidity, and wet bulb data for Fort Bliss on the day of Decedent's death, statements regarding the time and nature of physical training, and statements made by those rendering care on the scene. (*See* Pls.' Ex. II ¶ 6, ECF No. 197-30; Dr. Rusyniak Dep. 32:20-23, 135:5-12, 220:1-223:9; Hr'g Tr. 58:16-60:1, Apr. 27, 2015 AM, ECF No. 321). He opined that Decedent was well-conditioned, acclimated to the environment, and not dehydrated. (*See* Pls.' Ex. II ¶¶ 7-8, ECF No. 197-30; Dr. Rusyniak Dep. 32:10-14, 132:18-134:16, 135:13-21; Hr'g Tr. 57:10-58:15, 63:21-65:3, 106:19-107:23, 108:24-109:1, 148:19-149:4, 164:23-165:10, Apr. 27, 2015 AM, ECF No. 321). He found that Decedent did not have other medications, stimulants, or sympathomimetics in his system, sickle cell trait, an infection, or a family history of malignant hyperthermia. (*See* Dr. Rusyniak Dep. 21:4-25, 32:15-19; Hr'g Tr. 56:24-57:9, 63:1-20, Apr. 27, 2015 AM, ECF No. 321). Dr. Rusyniak further opined that DMAA, combined with exertion and other contributing factors, such as temperature, humidity, acclimation, and physical fitness, led to Decedent's hyperthermia and death. (*See* Dr. Rusyniak Dep. 53:7-55:18; Hr'g Tr. 60:18-22, Apr. 27, 2015 AM, ECF No. 321). Dr. Rusyniak considered the temporal association

between Decedent's DMAA use and his hyperthermia,[38] whether it was biologically plausible that DMAA could cause hyperthermia, and whether other factors could have increased Decedent's risk of hyperthermia. (*See* Hr'g Tr. 54:22-58:15, Apr. 27, 2015 AM, ECF No. 321). After taking these various factors into consideration, Dr. Rusyniak concluded that DMAA was a substantial factor in causing Decedent's hyperthermia and ultimate death. (*See* Pls.' Ex. II ¶¶ 4, 9, ECF No. 197-30).

a. Class Effects

1) Sympathomimetics Generally

The Court first reviews whether Dr. Rusyniak is merely relying on the class effects of sympathomimetics in order to come to conclusions about DMAA. Dr. Rusyniak testified that the literature he reviewed demonstrated that DMAA "caused similar physiologic and clinical responses that we see with drugs that are in the *sympathomimetic class*." (Dr. Rusyniak Dep. 87:18-25 (emphasis added)). When questioned about literature relating to sympathomimetics and their ability to increase temperature during exertion in warm environments, Dr. Rusyniak explained that he was referring to "MDMA, amphetamine or dextroamphetamine, methamphetamine. A variety of, and I'll say variety because I can't name them all, of substituted phenylethylamines, synthetic cathinones." (*Id.* at 67:13-18). He explained that "drugs that *are sympathomimetics all increase body temperature* when given to animals in a warm environment while they're exerting themselves." (*Id.* at 68:5-14 (emphasis added)). However, Dr. Rusyniak admitted that methamphetamine, ecstasy, and DMAA "are structurally

[38] "[T]he Fifth Circuit has rejected expert testimony that relies 'substantially on the temporal proximity between exposure and symptoms.'" *Newton v. Roche Labs., Inc.*, 243 F. Supp. 2d 672, 683 (W.D. Tex. 2002) (citing *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278 (5th Cir. 1998) (en banc); *Black v. Food Lion, Inc.*, 171 F.3d 308, 313 (5th Cir. 1999) (rejecting reliance on temporal proximity as 'not an exercise in scientific logic but in the fallacy of *post-hoc propter-hoc* reasoning, which is as unacceptable in science as in law'); *Lassiegne v. Taco Bell Corp.*, 202 F. Supp. 2d 512, 517 (E.D. La. 2002) (rejecting opinion based on 'temporal proximity alone' as 'present[ing] no scientific basis for [the] ultimate conclusion")).

different compounds." (*Id.* at 68:22-69:4). He also admitted that not all sympathomimetics have the same dose-response curve, effects on blood pressure, vasoconstriction, and body temperature, and that they are not absorbed at the same rate in the body. (*See* Hr'g Tr. 88:13-20, 90:10-93:7, Apr. 27, 2015 AM, ECF No. 321). Contrary to Dr. Cantilena, Dr. Rusyniak explained that route of entry and rate of absorption would have an effect on blood concentrations of various sympathomimetic substances. (*See id.* at 92:13-93:7).

Although Dr. Rusyniak testified that the sympathomimetics that he was familiar with are capable of causing vasoconstriction, he admitted that he had not studied the relative effects of DMAA and other sympathomimetics. (*See* Dr. Rusyniak Dep. 105:13-25, 110:18-25). He opined that, based on animal studies with MDMA, methamphetamine, and amphetamine, and human studies with cocaine, "[d]rugs that cause vasoconstriction . . . impair your ability to dissipate heat because you would not be able to dilate." (*Id.* at 118:20-119:5). He explained that "[d]rugs that are vasoconstrictors, like DMAA, are going to interfere with the cutaneous blood vessels, and as such, you are going to decrease your ability to dissipate heat." (Hr'g Tr. 52:9-12, Apr. 27, 2015 AM, ECF No. 321). Dr. Rusyniak based this explanation on an Eli Lilly study that demonstrated that DMAA was a "potent vasoconstrictor." (*See* Dr. Rusyniak Dep. 119:20-24; *see also* Hr'g Tr. 53:20-22, Apr. 27, 2015 AM, ECF No. 321). He opined that DMAA impairs the dissipation of heat at the cutaneous blood vessels "[b]ecause it *shares similar sympathomimetic properties* to . . . other drugs that do that." (Dr. Rusyniak Dep. 119:25-120:6 (emphasis added)). He added that he would expect "*all drugs that share those common pharmacologic and physiologic properties* . . . to impair the dissipation of heat." (*Id.* at 122:8-17 (emphasis added)).

Dr. Rusyniak testified that if an increase in blood pressure is due to an alpha agonist working on alpha receptors, the substance will result in an increase in cutaneous

vasoconstriction, however, he admitted that no studies demonstrated this specifically about DMAA. (*See* Hr'g Tr. 135:1-18, Apr. 27, 2015 AM, ECF No. 321). While Dr. Rusyniak pointed to no literature on DMAA, he explained that his opinion was "*based on properties that drugs that work like sympathomimetics and alpha receptors share*." (*Id.* at 135:17-18 (emphasis added)). He opined that "[i]n terms of dissipation, cutaneous blood flow would be probably the biggest single factor that a drug like DMAA[,] . . . a vasoconstrictor[,] is going to hit." (*Id.* at 52:21-23). Dr. Rusyniak admitted that increased blood pressure does not necessarily correspond with an increase in body temperature. (*See* Dr. Rusyniak Dep. 105:3-12). He further stated that "based on its *pharmacologic properties*, I would *infer* that . . . DMAA contributed to [heat stroke] in part through increased heat production by uncoupling." (*Id.* at 116:16-21 (emphasis added)).

Dr. Rusyniak opined that "if you give somebody a drug *that works like a sympathomimetic*, *very often* that will mask the delay or onset of fatigue and allow people to exert themselves longer, and that usually comes at the expense of body temperature." (Hr'g Tr. 53:13-19, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)). In response to a question from the Court, Dr. Rusyniak stated that no study investigated whether DMAA causes a delay or masks the onset of fatigue. (*See id.* at 132:9-17). This testimony demonstrates to the Court that Dr. Rusyniak is simply relying on the class effects of sympathomimetic drugs to extrapolate to his opinion on DMAA in order to rule it in as a potential cause.

2) Lack of Relevant Scientific Experience

While Dr. Rusyniak has experience studying various sympathomimetics, including amphetamine, methamphetamine, and MDMA in animals, he lacks scientific experience with DMAA, which is a factor that weighs against the reliability of his opinion. (*See* Dr. Rusyniak Dep. 67:19-25). Dr. Rusyniak stated that he has not conducted research on DMAA in humans or

animals and has no personal experience, which he is aware of, in treating someone who used DMAA. (*See id.* at 43:3-19, 68:20-21). He further explained that he has not performed a direct comparison between methamphetamine, amphetamine, and MDMA in any experiments in order to determine heat production or temperature changes and agreed that these types of studies had not been performed with respect to DMAA. (*See id.* at 70:4-71:10). In forming the opinion that Decedent's dose of DMAA was sufficient to cause his exertional heat stroke, Dr. Rusyniak testified that he had not performed a scientific comparison between Decedent's dose of DMAA, in milligrams per kilogram, to the doses used in animal studies on DMAA. (*See id.* at 151:16-153:7). He explained that while no studies indicate the dosage at which DMAA can cause a hyperthermic response or increase in core temperature, there are studies indicating that DMAA is a vasoconstrictor "*suggest[ing]* it would contribute to hyperthermia." (Hr'g Tr. 69:13-17, 93:8-14, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)). Despite Dr. Rusyniak's experience studying animals, the Court finds that he fails to demonstrate how he can extrapolate from animals to humans in any of the steps of his analysis.

3) Lack of Reliable Scientific Literature

Moreover, Dr. Rusyniak testified that he knew of no peer reviewed epidemiological studies or any studies, outside of case reports, that demonstrate causal links between DMAA and death, heat stroke, hyperthermia, and increased core temperature. (*See* Dr. Rusyniak Dep. 39:5-40:8, 41:7-21, 42:5-12, 47:15-48:11). While Dr. Rusyniak explained that "the case reports . . . ultimately determine whether something caused harm," he stated that even a single case report would make it hard to determine a cause. (*Id.* at 49:22-50:22). Dr. Rusyniak also opined that while some of the case reports reported an association between DMAA and various injuries, "[f]rom a pure scientific standpoint, you'd need to do a randomized placebo-controlled trial *to properly determine causation*." (*Id.* at 88:1-91:5 (emphasis added)). He further stated that in

order to quantify the percentage or likelihood that DMAA caused Decedent's death, it "would take a large controlled trial to determine what the incident rate is for [DMAA]." (Hr'g Tr. 104:19-21, Apr. 27, 2015 AM, ECF No. 321).

Additionally, Dr. Rusyniak testified that the increase in temperature demonstrated in the Schilling study did not meet the definition of hyperthermia and that the study was not accurate because the sample size was too small. (*See* Dr. Rusyniak Dep. 40:17-41:6, 141:3-145:7). He testified that "*none* of the human studies . . . were set up or designed to properly detect whether [DMAA] would *cause* hyperthermia." (Hr'g Tr. 69:7-9, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)). He further stated that the Bloomer studies had no "significant bearing" on his opinion. (*See* Dr. Rusyniak Dep. 161:22-162:6). However, he agreed that the literature on DMAA demonstrates a "*trend* that . . . acutely . . . DMAA and caffeine increase . . . systolic blood pressure in individuals." (Hr'g Tr. 155:23-156:4, Apr. 27, 2015 AM, ECF No. 321 (emphasis added)). He clearly admits that the scientific evidence on DMAA is lacking and that the only studies he is familiar with on DMAA had no "significant bearing" on his opinion, yet he agrees that the literature demonstrates a trend that would be helpful for his opinion. It appears to the Court that Dr. Rusyniak is not using the same scientific rigor in the courtroom as he would in the laboratory. Thus, the Court finds that Dr. Rusyniak's testimony fails to bridge the analytical gap between the generalized nature of the class of sympathomimetics and the specific characteristics of DMAA.

Even in Dr. Rusyniak's attempt to explain that a susceptible population has an increased risk to DMAA use, he testified that "I'm not sure we, from a scientific standpoint, know all the features that might increase somebody's susceptibility." (Dr. Rusyniak Dep. 125:2-8; *see also* Hr'g Tr. 146:12-147:15, Apr. 27, 2015 AM, ECF No. 321). He also testified that "I think *everyone* can develop exertional heat stroke, yes, absolutely." (Dr. Rusyniak Dep. 129:15-

20 (emphasis added)). He explained that he meant that everyone is susceptible "[u]nder the right conditions." (*Id.* at 129:21-130:6). He further testified that "it is my belief that if you took people and randomized them to DMAA or not and had them run in a warm environment, you would have significant[ly] more cases of exertional heat stroke in those getting DMAA." (*Id.* at 130:23-131:2). Dr. Rusyniak admitted that he was not aware of any peer reviewed literature that identified a susceptible population with regard to DMAA and heat stroke and hyperthermia. (*See* Hr'g Tr. 128:13-16, Apr. 27, 2015 AM, ECF No. 321). Although a scientist can have doubts in the scientific literature available, it is hard for the Court to find Dr. Rusyniak's opinion on DMAA reliable when he admits that science has not found explanations for a portion of his theory. As "[l]aw lags science," the Court finds that this portion of Dr. Rusyniak's opinion is entirely speculative.

b. Reliance on Other Experts

As to Dr. Rusyniak's reliance on Dr. Cantilena and Dr. Mills, the Court finds that, insofar as Dr. Rusyniak blindly adopts those experts' opinions, Dr. Rusyniak's opinion is unreliable. *See Lightfoot v. Hartford Fire Ins. Co.*, Civ. A. No. 07-4833, 2011 WL 39010, at *4 (E.D. La. Jan. 4, 2011) ("Rule 703 does not allow 'the wholesale adoption of another expert's opinions without attempting to assess the validity of the opinions relied on." (citation omitted)). Dr. Rusyniak attempted to explain that the phrasing used in his Rule 26 Expert Report that he was "relying" on Dr. Cantilena and Dr. Mills meant that he found them reliable. (*See* Hr'g Tr. 161:1-12, Apr. 27, 2015 AM, ECF No. 321). Although Dr. Rusyniak stated that he agreed with Dr. Mills' conclusions on the hyperthermic and sympathomimetic properties of DMAA and that they confirmed his own conclusions, he testified that "it would have been *important* to know from pharmacologists that this drug or compound, chemical, *was a sympathomimetic or at least had sympathomimetic properties*." (Dr. Rusyniak Dep. 86:23-87:10

(emphasis added)). Dr. Rusyniak specifically stated that he relied on Dr. Mills' explanation of the expected pharmacologic properties of DMAA and the role of uncoupling proteins, fatty free acids, and plasma glycerol. (*See id.* at 100:11-25, 102:15-103:8). In relying on Dr. Mills' opinion, Dr. Rusyniak testified he did so because Dr. Mills continues to perform research in areas relating to sympathomimetics that Dr. Rusyniak no longer focuses on. (*See* Hr'g Tr. 61:13-16, Apr. 27, 2015 AM, ECF No. 321). As to Dr. Cantilena, Dr. Rusyniak explained that he does not "routinely" work with outliers and he relied on Dr. Cantilena for that opinion. (*See id.* at 62:10-15). Dr. Rusyniak stated that he "deferred in terms of having [Dr. Mills and Dr. Cantilena] discuss the pharmacology [of DMAA]." (*Id.* at 141:11-15).

c. Conclusion on Ruling in DMAA as a Potential Cause

The Court finds that Dr. Rusyniak's opinion is unreliable as it suffers from many of the same pitfalls as the opinions of Dr. Cantilena and Dr. Mills. Dr. Rusyniak failed to account for any of the differences in extrapolating between the class effects of other sympathomimetics and DMAA. Furthermore, Dr. Rusyniak relies on animal studies without qualifying how they apply differently to humans. He lacks experience studying DMAA and does not account for the differences in dose in his extrapolations. Moreover, Dr. Rusyniak relied almost exclusively on animal studies and case reports, as he found that the Bloomer studies had no significant bearing on his opinion. He also could not explain how he could translate any of the studies to the general population. Finally, the reliability of Dr. Rusyniak's entire opinion is significantly undermined by his reliance upon the already excluded opinions of Dr. Cantilena and Dr. Mills, thus, the Court finds that Dr. Rusyniak's entire opinion purporting to rule in DMAA as a potential cause is excluded.

d. Differential Diagnosis

In light of the Court's findings as to Dr. Cantilena and Dr. Mills on general causation and

Dr. Rusyniak's opinion on ruling in DMAA as a potential cause, the Court finds Dr. Rusyniak's differential diagnosis to be unreliable. Without being able to rule in DMAA as a potential cause, Dr. Rusyniak cannot find that DMAA was a substantial factor in Decedent's hyperthermia and ultimate death. *See Johnson*, 685 F.3d at 468-69. Accordingly, the Court rejects the entirety of Dr. Rusyniak's specific causation testimony.

E. Defendants' Rule 702 and *Daubert* Challenges to Dr. ElSohly

Defendants challenge the reliability and relevance of Dr. ElSohly's opinion. (*See* Dr. ElSohly Mot. 8, 10, ECF No. 185; Dr. ElSohly Reply 3, ECF No. 213; Hr'g Tr. 42:1-45:1, 47:8-49:1, Apr. 27, 2015 AM, ECF No. 321). Dr. ElSohly offers an opinion on whether DMAA is naturally-occurring in geranium plants and oils. He explained that he has studied geranium plants and oils and published a peer-reviewed study on that subject, which found that the geranium plants and oils he studied did not have detectable amounts of DMAA despite using methods with various detection levels. (*See* Pls.' Ex. VV ¶ 3, ECF No. 197-43). Dr. ElSohly has since published a second peer-reviewed study which was performed in conjunction with three other laboratories, again failing to detect the presence of DMAA in geranium plants and oils.[39] (*See id.* at ¶ 9; Dr. ElSohly Dep. 25:19-28:4). He noted that he reviewed four other published, peer-reviewed studies that performed a similar analysis and did not find the presence of DMAA in geranium. (*See* Pls.' Ex. VV ¶ 4, ECF No. 197-43). He criticized the Ping, Li, and Fleming

[39] Neither party produced Dr. ElSohly's 2012 study, or a finalized version of his 2014 study. A pre-publication version of Dr. ElSohly's 2014 study is attached to his Rule 26 Expert Report. (*See* Pls.' Ex. VV 24-53, ECF No. 197-44).

studies, which purported to find DMAA in geranium plants, as unreliable scientific literature.[40] (*See id.* at ¶¶ 5-6, 12). He also opined that "[e]ven if the studies reportedly finding the presence of DMAA naturally occurring in geranium were valid at the levels they found DMAA it would be impossible to naturally extract DMAA from the natural source to be used at levels commonly found in products like Jack3d and OxyElite Pro." (*Id.* ¶ 12). Therefore, Dr. ElSohly concluded that "it is my opinion to a reasonably degree of scientific probability that DMAA is not a natural constituent of *Pelargonium graveolens*, *Pelargonium graveolens* oil, geranium or geranium oil."[41] (*Id.* at ¶ 13).

1. Reliability

Defendants challenge to Dr. ElSohly's testimony is based on Dr. ElSohly's failure to use the same methodology (detection level and samples) as used in the Ping, Li, and Fleming studies and his criticisms of those studies. (Dr. ElSohly Mot. 8-10, ECF No. 185; Dr. ElSohly Reply 3-5, ECF No. 213). It is clear to the Court that Defendants are not attacking the reliability of Dr. ElSohly's use of ten parts-per-billion as a detection level in his studies in the sense that in analytic and forensic chemistry, scientists use a different standard detection level. Dr. ElSohly testified that "we don't select the limits of detection. . . . You start doing the analytical process, you start doing all the work, and as a result of that work, you come up with the limit of detection." (Hr'g Tr. 14:9-18, 35:23-36:19, Apr. 27, 2015 AM, ECF No. 321). According to Dr.

[40] Dr. ElSohly's Rule 26 Expert Report lists all three studies among those he reviewed and provided with his Report. (*See* Pls.' Ex VV ¶ 11, ECF No. 197-43). The Ping study is Zang Ping et al., *A Study on the Chemical Constituents of Geranium Oil*, 25 J. Guizhou Inst. Tech. 83 (1996). (Defs.' Ex. 2, *Daubert* hearing). The Li study is J.S. Li. Et al., *Identification and Quantification of Dimethylamylamine in Geranium by Liquid Chromatography Tandem Mass Spectrometry*, 2012 Analytical Chemistry Insights 47 (2012). (Defs.' Ex. 1, *Daubert* hearing). The Fleming study is Heather L. Fleming et al., *Analysis and Confirmation of 1,3-DMAA and 1,4-DMAA in Geranium Plants Using High Performance Liquid Chromatography with Tandem Mass Spectrometry at ng/g Concentrations*, 2012 Analytical Chemistry Insights 59 (2012). (Defs.' Ex. F., ECF No. 178-9).

[41] Dr. ElSohly testified that pelargonium and geranium are used synonymously. (*See* Hr'g Tr. 35:16-22, Apr. 27, 2015 AM, ECF No. 321).

ElSohly a standard detection level is "not set by any organization." (*Id.* at 36:16). Furthermore, it is also clear that Defendants are not attacking the reliability of Dr. ElSohly's collection and authentication of geranium plants and oils in the sense that he did not provide collection information about the plants and oils in his studies.

Insofar as Defendants' argument is an argument about the reliability of Dr. ElSohly's testimony, the Court rejects it because Dr. ElSohly's failure to use the same methodology as the authors of other studies who have detected the presence of DMAA in geranium plants does not show that Dr. ElSohly's methodology is inherently flawed. Defendants' challenge equates to an attack on the conclusions of Dr. ElSohly's studies, i.e., that DMAA does not occur naturally in geranium plants and oils, which is not the type of challenge considered in the context of Rule 702 and *Daubert*. The Court recognizes that it is possible for multiple scientists to conduct tests on the same or similar plants and oils and reach different conclusions. Indeed, that much is clear from the literature referenced in the Fleming study. (*See* Defs.' Ex. F 3-4, ECF No. 178-9). Thus, Defendants' challenge fails to attack the methodology used by Dr. ElSohly as required in the context of Rule 702 and *Daubert*.

Dr. ElSohly testified that in his conducting his studies, he had no "preconceived idea whether [DMAA] was there or not [in geranium plants and oils]." (Hr'g Tr. 10:8-9, Apr. 27, 2015 AM, ECF No. 321). In fact, while Dr. ElSohly would not agree that it is "impossible that DMAA will be present in geranium plants," he testified that the scientific literature that he was relying on did not find the presence of DMAA in geranium plants. (Dr. ElSohly Dep. 28:5-21). Furthermore, Dr. ElSohly acknowledged that there are factors that would affect whether DMAA is present in geranium plants, including: the environment, the time of collection, the age of the plant, the altitude, the soil composition, the exposure to light, the ambient temperature, the time that the plant sample was picked, the storage conditions after it was picked, whether the sample

was dry or fresh, and the water moisture level. (*See id.* at 29:2-30:14; Hr'g Tr. 16:23-18:18, 27:19-28:6, Apr. 27, 2015 AM, ECF No. 321). Dr. ElSohly testified that while these conditions may affect the amount of DMAA in geranium plants, it could not have an effect on whether or not DMAA existed at all. (*See* Dr. ElSohly Dep. 31:6-14; Hr'g Tr. 16:23-18:18, 27:19-28:6, Apr. 27, 2015 AM, ECF No. 321). He expanded upon that answer as well:

> Q. Is it possible, then, for a plant from one part of a country or the world could have a different chemical composition as a plant from another part?
>
> . . .
>
> A. If it's the same species, the differences would be [a] quantitative difference, not [a] qualitative [difference].
>
> Q, So, you are saying . . . you couldn't find the presence of certain trace oils or minerals or compounds in a plant of the same species that was grown in China versus Africa?
>
> A. I'm saying the amount would be different.
>
> Q. But you're saying there couldn't be different ones at all?
>
> A. It might be because of the level of detection, or you know . . . some of the components that are not, you know, photosynthesized. For example, if you are looking for the presence of rare metal cadmium or some kind of element that will have something to do with the soil of that plant, where that plant is growing. You might find it in one area but not in another area. But as far as compounds that are photosensitized by the plant, by the enzyme machine of the plant - -
>
> Q. Sure.
>
> A. - - will be there, though it might be a different quantity, so you have - - you know, I've done some work, like I say again, with cannabis to determine the country of origin based on the chemical composition.
>
> Q. Because there's a signature - -
>
> A. Based on the chemical composition. Based on the signature. Based on that chemical composition . . . it's not that you're going to find, you know, a whole set of different compounds here but not here. All of them are there, it's just the ratio of those different compounds in there change from one location to the other.

(Dr. ElSohly Dep. 31:21-33:10). Dr. ElSohly admitted that he had not studied all of the same samples as the Ping, Li, and Fleming studies, in which those authors discovered the presence of DMAA in geranium plants. (*See id.* at 35:5-38:14, 63:3-24, 73:17-77:3, 78:16-79:22;

Hr'g Tr. 20:9-18, 26:9-15, Apr. 27, 2015 AM, ECF No. 321). Moreover, when he did, in fact, test one sample from the same province that Dr. Li tested and purportedly found DMAA at a different detection level, Dr. ElSohly failed to detect DMAA at ten parts-per-billion. (*See* Hr'g Tr. 16:4-22, 18:10-18, 26:11-12, Apr. 27, 2015 AM, ECF No. 321). Dr. ElSohly testified that though he thought the analytics of the Li and Fleming studies were sound, he felt that their findings, that DMAA was present in geranium plants, were based on the samples being contaminated. (*See id.* at 28:22-24).

Furthermore, Dr. ElSohly testified that "it would take so much [geranium] to get [enough DMAA] to put in one bottle . . . [that the DMAA used in Jack3d could not] be . . . under any circumstances . . . from the natural source." (*See id.* at 31:12-21). Specifically, he explained that even taking the "highest level" of DMAA that Dr. Li purported to find in geranium plants, "180,000 kilos of plant material [would be needed] to produce one bottle of [Jack3d]." (*Id.* at 31:12-18). Given the amount of DMAA used in Jack3d, Dr. ElSohly stated that the DMAA used in Jack3d could not be from a natural source and had to be synthetic.[42] (*See id.* at 31:19-32:24). Furthermore, Dr. ElSohly added that the Li and Fleming studies demonstrated that the DMAA they purported to find in geranium matched the composition of synthetic DMAA. (*See id.* at 32:7-13). Dr. ElSohly clarified that natural products do not exist in nature in the same composition as synthetic products. (*See id.* at 32:14-24).

Although Dr. ElSohly did not test the same samples using the same detection levels as those used in the Ping, Li, and Fleming studies, the Court finds that the work Dr. ElSohly performed and his opinion based on that work is reliable because, in applying the *Daubert* factors, the Court finds that Dr. ElSohly's technique has been tested, subject to peer-review and

[42] Dr. ElSohly testified that he tested a bottle of Jack3d to determine the amount of DMAA in the product. (*See* Hr'g Tr. 33:18-35:8, Apr. 27, 2015 AM, ECF No. 321).

publication, with a potential rate of error, and his technique is generally accepted in the scientific community. (*See* Dr. ElSohly Dep. 25:6-28:4, 38:25-39:8, 49:8-53:1, 59:23-62:8, 63:17-24, 73:17-77:3, 96:10-99:25; Hr'g Tr. 9:16-18:18, Apr. 27, 2015 AM, ECF No. 321; *see also Daubert*, 509 U.S. at 592-94). The Court further finds that because Dr. ElSohly's first study was published before this litigation commenced, it adds to the reliability of Dr. ElSohly's opinion. In the context of Rule 702 and *Daubert*, the Court also finds that Defendants cannot merely attack Dr. ElSohly's conclusion that DMAA is not naturally-occurring in geranium plants and oils instead they must challenge whether or not the methodology that he used in forming that opinion is reliable. The Court concludes that whether DMAA is naturally-occurring substance in geranium plants and oils is a proper subject for cross-examination and, therefore, Dr. ElSohly's expert opinion should not be excluded.

2. Relevance

The Court finds that Dr. ElSohly's opinion is relevant to assist the trier of fact in determining Plaintiffs' causes of action. While it may be that none of Plaintiffs' claims "turn on" whether DMAA occurs naturally in geranium plants and oils, the Court believes that Dr. ElSohly's opinion may assist the trier of fact in making determinations about causes of action, such as products liability and misrepresentation. Plaintiffs First Amended Complaint alleges that Defendants advertised and marketed Jack3d as a natural supplement, when they knew it was not. (*See* Am. Compl. ¶ 80, ECF No. 98). Plaintiffs further allege that misrepresentations about whether Jack3d was a natural product "falsely reassure[d] consumers that Jack3d is a safe product." (*Id.*) Plaintiffs also contend that the FDA warned companies that synthetically-produced DMAA is not a "dietary ingredient" and could not be used in a dietary supplement. (*See id.* at ¶ 81). Moreover, Plaintiffs allege that Dr. ElSohly's study refutes claims that synthetic DMAA is identical to naturally-occurring ingredients because geranium plants and oils

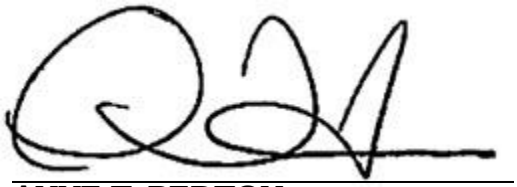
do not have DMAA at detectable levels. (*See id.* at ¶ 84). Accordingly, the Court finds that Dr. ElSohly's opinion is relevant and, therefore, denies Defendants' Motion to Strike his opinion.

IV. CONCLUSION

For the reasons stated above, it is **HEREBY ORDERED** that Defendants' Motions to Strike Dr. Cantilena (ECF No. 184), Dr. Mills, (ECF No. 186), and Dr. Rusyniak (ECF No. 187) are **GRANTED** and Defendants' Motion to Strike Dr. ElSohly (ECF No. 185) is **DENIED**.

Accordingly, Defendants' Motion to Exclude Untimely Disclosures (ECF No. 258) is **DENIED AS MOOT**.

SIGNED and **ENTERED** this 27th day of July, 2015.



ANNE T. BERTON
U.S. MAGISTRATE JUDGE