IN THE STATE COURT OF COBB COUNTY
STATE OF GEORGIA

TOM MUTZ, *et. al*,

Plaintiffs,

vs.

STERIGENICS U.S., LLC, *et.al.*,

Defendants.

CIVIL ACTION NO.: 20-A-3448

This document relates to:

1. Roxana Gil and Adam Gil, as Natural Parents of Olivia and Roslyn Gil; Civ. Action No. 20-A-3012
2. Matthew Cassacia as Personal Representative of the Heirs and Estate of Barry Goppman and Patricia Goppman; Civ. Action No. 21-A-151
3. Chad Stephens and Karen Stephens; Civ. Action No. 21-A-2425
4. Ralph Stephen Franks and Deborah D. Franks; Civ. Action No. 21-A-2414
5. Lyndsey Hayes; Civ. Action No. 21-A-2462

ORDER

Introduction

This case has a long history with the Court. The first Sterigenics-related cases began populating the Court's docket in January 2020. To date, there are hundreds of Plaintiffs seeking compensation from Defendants. There have been innumerable conferences,

scheduling, and substantive orders. There have been four attempts for interlocutory review. Two of those applications have been successful. In 2022, Defendants appealed the Court's denial of severance of the close to 400 plaintiffs. In 2024, both parties appealed the Court's November ruling on General Causation.

On October 31, 2025, the Court of Appeals vacated the November 2024 ruling of this Court and remanded the referenced cases[1] for further consideration. *Sterigenics US, LLC, et al. v. Mutz, et. al.*, 923 S.E.2d 176 (Ga. App. 2025). In its Order the Court of Appeals specifically directed this Court to:

1) Apply, in the first instance, the Eleventh Circuit standard as enunciated in *McClain v. Metabolife Intl., Inc.*, 401 F3d 1233 (11th Cir. 2005) and its progeny. *Sterigenics US, LLC, et al.* at 179-180.
2) Determine whether the medical community routinely and widely recognizes that the drug or chemical at issue is both toxic and causes the type of harm alleged by the plaintiffs. *Id.* at 185
3) If so, the battleground shifts to specific causation. *Id.*
4) If not, the court must address the reliability of the methodology used by the plaintiff's general causation experts. *Id.*
5) When addressing the reliability of methodology used by plaintiffs' general causation experts, "the court must pay careful attention to the experts' testimony about the dose-response relationship and whether an expert has identified a harmful level at which the drug or chemical could cause the harms alleged. The trial Court also should consider whether the expert can establish general causation through the alternative methodologies of epidemiology and background risk of disease." *Id.*

[1] Although the Court of Appeals decision was also entered for case numbers 20-A-3448 (*Mutz*), 21-A-2420 (*Bonner*), and 21-A-4396 (*Harrell*), this Court in its order dated October 17, 2025, granted summary judgment to Defendants in Phase 2. Plaintiffs have filed a notice of appeal in those cases.

And this is where the Court begins.

First, this Court determines that ethylene oxide (EtO) is a Category 2 substance because, using the *McClain* standard, the medical community does not routinely and widely recognize that EtO causes the types of harm Plaintiffs allege. Second, this Court concludes that Plaintiffs' experts are unable to provide a threshold dose-response relationship that satisfies the requirements of the *McClain* standard. Third, this Court finds Plaintiffs' experts lack sufficient reliability to establish general causation under O.C.G.A. § 24-7-702, *Daubert*, and the standard articulated in *McClain* and its progeny as to alternative methodologies. Therefore, Plaintiffs' expert opinions are inadmissible and Defendants' motion for summary judgment is granted.

1. *McClain* and its progeny.

Pursuant to the directive of the Court of Appeals, this Court must apply *McClain* and its progeny. The Court conducted a comprehensive review of that case law. Although the review is not exhaustive, it is intended to satisfy the directive of the Court of Appeals. These cases primarily concern pharmaceuticals and medical devices rather than toxins. The limited number of cases involving toxins are either related to workplace exposure or are otherwise distinguishable from the present matter.

This case is entirely unique. These Plaintiffs seek damages from alleged exposure to a naturally occurring toxin whose molecular structure and effects are present in all humans.[2] And Plaintiffs' expert Dr. Felsher agrees: there is no difference between naturally occurring EtO and synthetic or non-natural EtO.[3] It is no surprise then that Plaintiffs' experts cannot meet O.C.G.A. § 24-7-702, *Daubert,* or *McLain* standards as to the dose-response

[2] It is formed from ethylene conversion during metabolic processes and is present in our bodies.

[3] Plaintiff's expert Dr. Felsher acknowledges that the human body naturally produces EtO. Dr. Felsher Dep. July 2024 at 108:24:110:25. He further concedes that EtO molecules are physically and chemically identical, regardless of their source. *Id.* at 90:5-10, 91:2-3, 93:9-10.

relationship. In other words, measurability and reliability are as elusive as the molecules that Plaintiffs allege caused their harm.

In *McClain*, the substance at issue was a combination of ephedrine and caffeine. *McClain,* 401 F.3d at 1234. Because the medical community did not generally recognize ephedrine or caffeine, either individually or in combination, as causing the harm alleged by the *McClain* plaintiffs (heart attacks and strokes), that court conducted a comprehensive *Daubert* inquiry addressing both general and specific causation. *Id.* at 1236, 1239.

Where "the medical community does not generally recognize the agent as both toxic and causing the injury plaintiff alleges," a court must conduct a two-part *Daubert* analysis that "covers not only the expert's methodology for plaintiff-specific questions about individual causation but also the general question of whether the drug or chemical can cause the harm plaintiff alleges." *Id.* at 1239. "General causation is concerned with whether an agent increases the incidence of disease in a group and not whether the agent caused any given individual's disease." *Id.* When analyzing an expert's methodology in toxic tort cases, the court should "pay careful attention to the expert's testimony about the dose-response relationship." *Id.* at 1241.

One of the first 11th Circuit cases after *McClain* is *Cleveland v. United States*, 2006 WL 5334601 (N.D. Ga. 2006). In that workplace exposure case, the plaintiffs argued that the defendant breached its duty by failing to provide an expectant mother with a safe work environment during her pregnancy and by failing to warn of potential harm from exposure to hazardous substances. *Id.* at *1. Those plaintiffs claimed that the chemicals involved were teratogenic and/or mutagenic, and that the defendant's breach led to the child developing a brain tumor (glioblastoma multiforme) along with other related health issues. *Id.*

The *Cleveland* court conducted a brief analysis of *McClain* but ultimately accepted plaintiffs' alternative argument: that even if the expert testimony did not meet all *McClain* criteria, it should be permitted because the case was a bench trial. *Id.* at *4. The court explained the Federal Rules of Evidence allow district courts to admit a broader range of

scientific testimony than would have been admissible under *Frye*. *Id*. The court noted, "'There is less need for the gatekeeper to keep the gate when the gatekeeper is keeping the gate only for himself.'" *Id*. at *5, citing *United States v. Brown*, 415 F.3d 1257, 1268-69 (11th Cir.2005).

The standard applied in *Cleveland* is materially distinct from the present case. While some facts may initially seem pertinent, *Cleveland* addressed workplace exposure. Furthermore, the reliability requirements under Rule 702 and *McClain* were not at issue, as judges in bench trials possess greater expertise in evaluating expert testimony than laypersons. Consequently, the analysis in *Cleveland* offers limited guidance for the facts of this case.

The next case reviewed by this Court involved denture wearers who sued a denture adhesive manufacturer, alleging the adhesive caused them to suffer zinc-induced, copper-deficiency myelopathy (CDM). *Chapman v. Procter & Gamble Distrib.,* 766 F.3d 1296 (11th Cir. 2014). The plaintiff in *Chapman* argued the substance was a *McClain* Category 1 substance, claiming there was a general consensus in the medical community that ingesting zinc causes copper deficiency myeloneuropathy (CDM). *Id*. Plaintiff also referenced medical textbooks, journals, and testimony from her experts as well as those of defendant. All recognized an association between excess zinc and copper deficiency. *Id*.

However, that court determined the experts failed to prove the zinc compound in the substance qualified as a *McClain* Category 1 medically accepted, cause-and-effect toxin, similar to asbestos causing asbestosis or cigarette smoking causing lung cancer and heart disease. *Id*. at 1314, *citing Rider v. Sandoz Pharm. Corp*., 295 F.3d 1194, 1199. That court ruled the expert testimony failed to show "dose-response, epidemiological evidence, and background risk of disease, methodologies [that] this circuit has recognized as indispensable to proving the effect of an ingested substance." *Id*. at 1308. Those plaintiffs' general causation experts, and the articles upon which they relied, didn't determine "how much" of the substance needed to "be used for how long" to cause harm. *Id*. at 1307.

The application of the *McClain* standard in *Chapman* offers significant guidance for the present case. Although *Chapman* was in the pharmaceutical context, the principle regarding the relevance of exposure to a substance remains applicable. The *Chapman* court clarified that Category 1 toxins are defined as those recognized as medically accepted cause-and-effect toxins.

In the present case, for EtO to be classified as a Category 1 toxin, Plaintiffs' experts are required to demonstrate a specific cause-and-effect relationship, which they have not established. Additionally, *Chapman* emphasized that expert testimony must reliably employ at least one of the three primary methodologies to establish causation. *Id.* at 1307. As discussed in detail below, Plaintiffs' experts did not reliably utilize any of the three primary methodologies to establish causation.

Finally, the *Chapman* court determined that general causation experts must specify the quantity of the substance and the duration of exposure necessary to cause harm. *Id.* In both *Chapman* and the present case, Plaintiffs' experts failed to satisfy this requirement.

A 2018 case further supports the requirement for a specific link between a pharmaceutical and the cause of an adverse effect. *In re Abilify (Aripiprazole) Prods. Liab. Litig.*, 299 F. Supp. 3d 1291, 1306 (N.D. Fla 2018). In *Abilify*, patients sued manufacturers and marketers of the prescription drug used in patients with mental disorders. They alleged that, after taking the drug as prescribed, they developed impulsive and irrepressible urges to engage in certain harmful behaviors. *Id.*

The 11th Circuit held " there are three primary methodologies which are indispensable for proving whether a drug can cause a specific adverse effect: epidemiological studies, dose-response relationship, and background risk of disease." *Id.* at 1306, *citing Chapman*, 766 F.3d at 1308. "A general causation opinion that is not supported by at least one of these primary methodologies is unreliable as a matter of law." *See id.*

An expert who has reliably applied primary methodologies may bolster his general causation opinion with evidence from "secondary" methodologies, such as:

1. biological plausibility,
2. case studies,
3. adverse event reports,
4. extrapolations from animal and *in vitro* studies, and/or
5. extrapolations from analogous drugs.

See id. Importantly, the flaws inherent in the secondary methodologies limit their reliability under *Daubert*. *See id.* For this reason, secondary methodologies alone, even in the aggregate, cannot establish general causation. *See id.*

The *Abilify* court makes it clear, unreliability is untenable. Plaintiffs' expert opinions in this case are not supported by any one of the three primary methodologies, *see infra*. Therefore, according to *Abilify* and *Chapman*, their opinions are unreliable as a matter of law.

Another case that deserves discussion is *Williams v. Mosaic Fertilizer, LLC*, 889 F.3d 1239 (11th Cir. 2018). In *Williams*, a homeowner sued a factory operator, alleging that emissions of toxic substances from the factory caused or worsened various medical conditions, including the homeowner's pulmonary hypertension and other lung problems. *Id.*

Following *McClain*, the *Williams* court determined that the main downfall of the plaintiff's experts was their reliance on regulatory data and studies. What mattered in *Williams* was whether the EPA data offered a reasonably specific calculation of the exposure levels needed to cause the plaintiff's conditions and could the expert reasonably rely on them when forming his opinions. *Id.* The court ultimately rejected reliance on regulatory standards, even when they were "based on human studies", to establish causation in a toxic tort case involving airborne emissions. *Id.* at 1246–47. The court found such regulatory standards are protective rather than predictive of causation. *Id.*[4]

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[4] *See also*, *McClain* at 1250, *citing Glastetter v. Novartis Pharmaceuticals Corporation*, 252 F.3d 986, 991(2001 8th Cir Ct. Appeals) (internal cites omitted:

Additionally, the *Williams* court determined that the plaintiff's expert did not properly assess dose-response and failed to account for background risk. *Id.* at 1245-1246. The court further explained that the plaintiff bore the burden of demonstrating that the expert's determinations were methodologically sound. *Id.* at 1248. As the plaintiff failed to establish the methodological soundness of the expert's opinions, that court concluded the district court did not abuse its discretion in excluding the testimony. *Id.* at 1245.

Plaintiffs' experts, including Dr. Stayner, a former regulatory scientist, applied scientific methodologies in a cursory and conclusory manner. Their primary reliance on regulatory studies, as analyzed in *Williams*, is inadequate. In other words, public policy protections and courtroom causation are not the same.

In sum, *Williams* guides this Court when evaluating the testimony of Plaintiffs' experts in this airborne emissions case. The Court finds these Plaintiffs experts' opinions inadequate and unreliable.[5]

The next case, *Taylor v. Mentor Worldwide, LLC*, 940 F.3d 582 (11th Cir. 2019), involves a medical device. A patient who received an allegedly defective polypropylene mesh sling made by the defendant filed a products liability lawsuit. After a jury verdict in favor of the plaintiff, the defendant moved for judgment as a matter of law or, in the alternative, for a new trial. The trial court concluded, "[i]n this case, which is focused on the physiological response to a design defect in a medical device, the dose-response relation is not implicated." *Id.* at 596. Nonetheless, that court stated that, "[g]iven the 'importance of

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> The FDA's approach differs from ours in another critical aspect. The FDA will remove drugs from the marketplace upon a lesser showing of harm to the public than the preponderance-of-the-evidence or the more-like-than-not standard used to assess tort liability. The methodology employed by a government agency results from the preventive perspective that the agencies adopt in order to reduce public exposure to harmful substances.

[5] *See Henderson, infra.* The *Henderson* court found "regulatory standards are not enough standing alone to establish general causation on a full *Daubert* inquiry, they certainly are not enough to show that these substance/illness pairings fall into category one." *Henderson v. Lockheed Martin Corp., 2023 U.S. Dist. LEXIS 237286 (M.D. Fla. Sept. 18, 2023)* at *19, 20 and n.3, 21, 23.

individual responses to toxins,' a plaintiff must demonstrate both [1] the level of exposure to the allegedly harmful chemical that is hazardous to a human being and [2] the amount of the chemical to which plaintiff was exposed." *Id.* 595 (quoting *McClain*, 401 F.3d at 1241.)

Unlike *Taylor*, the present case concerns airborne emissions. Even if this Court, as the Court in *Taylor*, accepts Plaintiffs' assertion that dose-response is not measurable and cannot be determined for these Plaintiffs, it remains— as established in *McClain* and subsequent cases— general causation requires more than speculation and conclusory opinions.[6] *Taylor* identified, as did *Chapman* and *Abilify*, epidemiology and background risk as other primary methodologies available to plaintiffs for supporting expert opinions on general causation. In this case, Plaintiffs' experts, even when disregarding dose response, fail to provide reliable opinions using either alterntive methodology. Instead, Plaintiffs' experts rely primarily on regulatory findings and conclusory statements, which this Court finds insufficient, unreliable, and inadequate to satisfy O.C.G.A. § 24-7-702, *Daubert,* and *McClain* standards.

Here, Plaintiffs' experts have offered little beyond regulatory findings and conclusory statements, which this Court finds unhelpful, unreliable and unable to meet O.C.G.A. § 24-7-702, *Daubert* and *McClain* standards.

In *Pinares v. Raytheon Technologies,* 2023 WL 2661521 (11th Cir. 2023), the plaintiff alleged that a Raytheon facility leaked chemicals that contaminated the water supply and ultimately caused her to develop kidney cancer. *Id.* The plaintiff appealed the exclusion of her causation expert. The 11th Circuit upheld the exclusion because the expert's hypothesis asserted that no safe threshold existed for the chemicals in question. The expert cited no supporting authority and failed to explain why a model that disregarded dosage was appropriate. *Id.* at *4-5. As a result, the model lacked a reliable baseline. Without such

[6] *See also*, *In re Deepwater Horizon Belo Cases*, U.S. Dist. LEXIS 48865 (11th Cir. 2024), "...general causation in a category two case must address the dose-response relationship."

baseline, the 11th Circuit was unable to assess whether the expert's conclusion regarding causation was scientifically valid. *Id.*, *citing Chapman*, 766 F.3d at 1307; *Williams,* 889 F.3d at 1246–48.

Even though *Pinares* is an unreported and therefore non-binding opinion, it too follows *McClain*, *Chapman,* and *Williams*. This Court, while not relying on the holding in *Pinares*, finds its analysis instructive.

Another case involving workplace exposure is *Henderson v. Lockheed Martin Corp.*, 2023 U.S. Dist. LEXIS 237286 (M.D. Fla. Sept. 18, 2023). The *Henderson* court rejected the plaintiff's Category 1 classification of the substances allegedly causing harm. The plaintiff's experts argued that because the toxins were "genotoxic and carcinogenic," they should be classified as Category 1. *Id.* The court emphasized the need for a "widespread medical consensus" of an "undeniable causal link between the substances and the types of illnesses alleged," including "the particular types of cancer alleged." *Id.* The court also highlighted that because "regulatory standards are not enough standing alone to establish general causation on a full *Daubert* inquiry, they certainly are not enough to show that these substance/illness pairings fall into category one." *Id.* at *19, 20 and n.3, 21, 23.

At the hearing on remand held on March 18, 2026, Plaintiffs' counsel advanced the same argument as the plaintiffs in *Henderson*. Plaintiffs' counsel asserted that, because EtO is "genotoxic and carcinogenic," it must be classified as a Category 1 toxin. It is not that simple. The Court of Appeals directed this Court to determine whether the "medical community routinely and widely recognizes that the drug or chemical at issue is both toxic and causes the type of harm alleged by plaintiffs." *Sterigenics US, LLC, et al. v. Mutz, et. al.*, 923 S.E.2d 176, 185 (Ga. App. 2025). That is an exacting standard. Upon review of all the studies, depositions and expert opinions presented in this case, this Court finds that the medical community does not routinely and widely recognize EtO as both toxic and causative of the alleged harm(s). Therefore, EtO is not a Category 1 toxin.

The next case, *In re Deepwater Horizon BELO Cases (DH Belo)*, 119 F.4th 937 (11th Cir. 2024), was analyzed in this Court's November 2024 order. However, certain points warrant reiteration. In *DH Belo*, the 11th Circuit affirmed the district court's exclusion of the plaintiffs' proffered toxicology expert under O.C.G.A. § 24-7-702. *Id.* As a result, that court upheld summary judgment in favor of the defendants thereby dismissing the plaintiffs' claims arising from the Deepwater Horizon oil spill. *Id.* at 943

The *DH Belo* court found the expert's failure to consider relevant exposure data, combined with reliance on data from locations distant from the plaintiffs' residences, rendered the opinion unhelpful and not a proper fit for the circumstances. *Id.* at 941. Additionally, the *DH Belo* trial court concluded that the expert's excessive reliance on EPA benchmarks, use of a conclusory Bradford Hill analysis, failure to independently evaluate the limitations and biases of the epidemiological studies relied upon (including not acknowledging the limited value of cross-sectional studies for establishing causation), and lack of distinction between acute symptoms and chronic conditions were critical shortcomings given the nature of the injuries at issue. *Id.* at 943.

As discussed in detail below, Plaintiffs' experts Dr. Stayner and Dr. Felsher, are similar to the expert in *DH Belo*. They almost exclusively rely on regulatory studies and documents. Furthermore, Dr. Stayner and Dr. Felsher did not perform a comprehensive and objective Bradford Hill analysis, the lack of which was fatal to the expert in *DH Belo*. These failings, together with the failures previously identified further undermine the reliability of Plaintiffs' experts' general causation opinions.

The final and most recent decision this Court reviewed is that of *Perrotti v. Lockheed Martin Corporation*, 2025 WL 2554425 (Slip Op.). This workplace exposure case was brought by a widow on behalf of her husband who passed away from gastroesophageal cancer after a two decade career as a Lockheed engineer. What caused his cancer was the central matter in dispute.

The plaintiff's expert in *Perrotti*, Dr. Sahu, is the same expert these Plaintiffs proffered for case numbers 20-A-3448 (*Mutz*), 21-A-2420 (*Bonner*), and 21-A-4396 (*Harrell*), in Phase 2 specific causation. The court in *Perrotti* completed an exhaustive review of *McClain* and its progeny and concluded as the courts did in *Williams* and *Taylor*. It stated, "[c]ausation in drug cases is not the same as in atmospheric exposure cases. That is a scientifically grounded distinction and one the general state of the law in this Circuit supports." *Id.* at *16. This Court concurs but does not disregard all precedent solely because this is an atmospheric exposure case. The Court of Appeals has specifically instructed this Court to avoid such an approach. *Sterigenics US, LLC, et al. v. Mutz, et. al.*, 923 S.E.2d 176, 179-180 (Ga. App. 2025).

Perrotti is easily distinguishable from the case at bar. *Perrotti* addresses workplace exposure, whereas the present case concerns the alleged exposure of property owners residing miles away from Sterigenics' facility. In *Perrotti*, the experts established background exposure, whereas Plaintiffs' experts in this case did not. Here, the toxin at issue, EtO, is produced and expelled by the human body, unlike the toxins in *Perrotti*. Furthermore, the substances in *Perrotti* were not ubiquitous, whereas EtO is. The *Perrotti* court, in its conclusion, cautioned, "[s]training precedent beyond its factual context leads to unintended consequences." *Perrotti* at *16. This Court applies the caution described in *Perrotti* and refrains from extending precedent beyond its intended scope, particularly in light of the unique facts of this case. This Court remains resolute in its findings.

Having completed the *McClain* and its progeny analysis, this Court now addresses the remaining directives of the Court of Appeals.

2. Is EtO a Category 1 toxin?

Throughout the history of this litigation, this Court has proceeded on the understanding that EtO is classified as a category two substance.[7] Nearly three years ago,

[7] First Consolidated Case Management Order (June 11, 2021).

this Court stated, "[T]oxic tort cases usually come in two broad categories: first, those cases in which the medical community generally recognizes the toxicity of the drug or chemical at issue, and second, those cases in which the medical community does not generally recognize the agent as both toxic and causing the injury plaintiff alleges." This case falls into the second category."[8] This Court reaffirms its original classification of EtO as a category two substance.

However, Plaintiffs assert that EtO should be classified under *McClain* Category 1. Plaintiffs maintain that EtO is genotoxic and carcinogenic, meaning it can damage human cells and cause cancer, and therefore does not require linkage to specific illnesses to qualify for Category 1. The Court disagrees. The purpose of Category 1 is to recognize that the causal relationship between a substance and an illness is so well established that it eliminates the need for a comprehensive *Daubert* general causation analysis. *McClain* at 1239-1241. Even if a substance is generally recognized as harmful to humans, the Court must determine whether scientific evidence establishes a definitive causal link between the substance and the specific illnesses alleged. *See McClain*, 401 F.3d at 1239; cf. *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 198 (5th Cir. 1996) (whether a substance has "genotoxic capabilities ... is the beginning, not the end of the scientific inquiry and proves nothing about causation without other scientific evidence.")

Specifically, Plaintiffs allege that EtO is generally accepted as a cause of Plaintiffs' injuries. In support of these assertions, Plaintiffs point to various findings by regulatory agencies. But the 11th Circuit has repeatedly found that regulatory findings and standards alone are insufficient to reliably establish general causation, as regulatory bodies are focused on protecting public health and thus err on the side of caution in determining what is safe. *See McClain*, 401 F.3d at 1248–50. Public health rules are not altogether excluded from consideration, but courts must exercise caution in examining their methodology. *See*

[8] March 29, 2023 Order.

McClain, 401 F.3d at 1249; *cf. Waite v. AII Acquisition Corp.*, 194 F. Supp. 3d 1298, 1313 (S.D. Fla. 2016). Where experts heavily relied on regulatory authorities and findings were usually *McClain* Category 2 substances. But the same reasoning is even more relevant when assessing whether substance/illness pairs belong in Category 1.[9]

No court has previously classified EtO as a Category 1 substance. Similarly, other carcinogens frequently addressed in toxic tort litigation have not been designated for Category 1 treatment. *In re: Zantac (Ranitidine) Prods. Liab. Litig.*, 644 F. Supp.3d 1075 (S.D. Fla. 2022). EtO is a naturally occurring and ubiquitous substance found in both the human body and the atmosphere. If classification as a "carcinogen" alone were sufficient for Category 1 designation, the number of Category 1 toxins would significantly exceed that of Category 2 toxins. *Id.* at 1105-06 (rejecting as "untenable" that a component "known to be a carcinogen" fell into Category 1 because it "is a ubiquitous substance found in trace amounts in air, water and food.")

Dr. Felsher also opined that endogenous EtO created by the body, cannot cause cancer.[10] Subsequently, he claimed that inhalation of endogenous EtO can cause cancer.[11] This endogenous EtO is identical to the substance responsible for certain non-anthropogenic background levels that Dr. Felsher considers safe.[12] However, Dr. Felsher is unable to provide supporting studies. Consequently, the Court cannot rely on Dr. Felsher's opinion that exposure to EtO causes the cancers alleged by some of these Plaintiffs. Therefore, EtO does not meet the criteria for classification as a Category 1 toxin.

Plaintiffs must prove a connection between the substance and the type of illness alleged. Plaintiffs rely heavily on regulatory standards to support their argument that EtO

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[9] *See also*, *Williams v. Mosaic Fertilizer, LLC*, 889 F.3d 1239 (11th Cir. 2018) and *Taylor v. Mentor Worldwide, LLC*, 940 F.3d 582 (11th Cir. 2019).

[10] Dr. Felsher Dep. at 114:17-120:15.

[11] Dr. Felsher Dep. at 132:8-23.

[12] *Id.*

falls into *McClain* Category 1, but this evidence is insufficient. EtO and the diseases alleged by Plaintiffs must be evaluated in a full *Daubert* inquiry under *McClain* Category 2.

3. Because EtO is a Category 2 toxin, methods of proving general causation must be reviewed.

3.1. Dose-Response

3.1.1. The dose-response relationship is the best way to establish general causation in a Category 2 substance.[13]

Generally, when assessing the toxicity of a Category 2 substance, a dose-response relationship is required to establish general causation. *McClain* is clear: the gold standard as to general causation is the dose-response relationship as it is the foremost principle of general causation.[14] The dose-response relationship as "the hallmark of the science of toxic torts,"[15] and the "...hallmark of basic toxicology."[16] Plaintiffs' expert Dr. Salem agrees. "What we do, as scientists, is we look at different doses and then we look at different levels of incidence and correlate dose to incidence."[17]

As stated in this Court's November 22, 2024 Order, Plaintiffs' experts Dr. Salem and Dr. Felsher fail in this regard.[18] The question before this Court now is: Can Dr. Stayner's opinion satisfy the *McClain's* general causation standards for a Category 2 substance by way of a dose-response relationship. This Court finds that he cannot. "When analyzing an expert's methodology in toxic tort cases, the court

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[13] An "expert who avoids or neglects the dose-response principle of toxic torts without justification casts suspicion on the reliability of his methodology." *Kilpatrick v. Breg, Inc.*, 613 F.3d 1329, 1339 (11th Cir. 2010) (alteration adopted) (quoting *McClain*, 401 F.3d at 1242).

[14] *McClain* at 1242.

[15] *Mc Clain* at 1240.

[16] *McClain* at 1242.

[17] Salem Dep. 86:7-10

[18] The Court adopts and reiterates its findings in its November 22, 2024 order as to the testimony of Dr. Salem and Dr. Felsher and its findings as to birth defects.

should pay careful attention to the expert's testimony about the dose-response relationship."[19] In fact, Dr. Stayner does not even try to give a threshold level at which EtO can cause the harm alleged.

Dr. Stayner's testimony is almost verbatim the testimony of Dr. O'Donnell, the expert in *McClain* who failed the *Daubert* analysis in that seminal, controlling case. "Although [Dr. O'Donnell] agreed that a drug's effect is dose-driven, he offered no testimony about the dose of Metabolife required to injure Plaintiffs or anyone else. He could not say how much is too much."[20] Like Dr. Stayner, "...O'Donnell could not provide any opinions about the general dose-response levels for Metabolife's toxicity, i.e., the dose or level of exposure at which it causes harm."[21] Dr. Stayner refused to opine on the dose-response relationship even though he states that there is strong evidence of the exposure and dose response. "[W]e do, of course, consider whether there's evidence of dose response, it's a very strong evidence if we can show the relationship between exposure response or dose response. But I'm not going to be offering any opinion about what is dose or duration required to produce these cancers."[22] He doubled down. "My opinion is independent of dose."[23] And apparently, he disagrees with the 11th Circuit's decision in *McClain* when he states general causation is "not a matter of...dose."[24]

The Court of Appeals specifically rejected this any-exposure-creates-risk analysis this Court articulated in its November 22, 2024 Order. "[T]he 11th Circuit does not recognize an exception to its two-tier classification system for experts testifying that 'any exposure' can cause harm, and *McClain* makes clear that experts

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[19] *McClain* at 1241.
[20] *Id.*
[21] *Id.*
[22] Stayner Dep. 30:3-9.
[23] Stayner Dep. 32:4.
[24] Stayner Dep. 29:24.

who employ a dose-response methodology to prove general causation must identify a harmful level at which the toxin could cause the harm alleged."[25]

The dose or level of exposure at which EtO causes harm is important because, at low doses, an otherwise toxic substance may not cause the harm alleged by Plaintiffs. "Often 'low dose exposures -- even for many years -- will have no consequence at all, since the body is often able to completely detoxify low doses before they do any damage.' Furthermore, 'for most types of dose-response relationships following chronic (repeated) exposure, thresholds exist, such that there is some dose below which even repeated, long-term exposure would not cause an effect in any individual.'"[26]

Dr. Felsher explicitly states that Plaintiffs **cannot** meet this standard in this case. "The data to date does not set a threshold but demonstrates the EtO is genotoxic and cancer causing. Population studies have shown that when EtO is considered as a continuous variable there is no threshold level of exposure that would prevent a risk for cancer. ...It is important to note that no study is capable of identifying, or could be designed in a way to identify, a threshold."[27] Dr. Salem agrees. In fact, all studies relied upon by Dr. Salem reference the dose-response relationship. "I have not read a paper that specified that there is a safe amount of ethylene oxide that does not create chromosomal aberrations. The data that I have read, and so the only thing that I can draw my conclusions on, are that there is a relationship between dose and DNA adducts and chromosomal aberrations."[28]

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[25] *Sterigenics, US, LLC, et al. v. Mutz, et. al.*, 377 Ga. App. 624, 633 (2025).
[26] *McClain* at 1242. (Citations omitted.)
[27] Felsher Report p. 28.
[28] Salem Dep. 77:7-13.

Without such testimony, Plaintiffs' experts cannot show a threshold level of dose required, and their testimony must be excluded under a O.C.G.A. § 24-7-702 analysis.

3.1.2. Studies referred to by Plaintiffs' experts are dose-dependent at high levels of exposure.

Plaintiffs' experts' reliance on dose-response in studies puts the lie to their opinions that "any exposure" or "any exposure over background" can cause the harm alleged. The studies show that—generally—the higher the dose, the more likely the harm. And the doses in these studies are high. "[T]he majority of positive studies evaluated exposure conditions in which higher concentrations were present."[29] "The results show that relatively high exposures to EtO were required to induce detectable increases in chromosome aberrations."[30] "It's my opinion that chromosomal aberrations increase as a function of dose-response."[31] Even Stayner's study relies on dose-response.[32] And it found increased risk of breast cancer at "higher cumulative exposures to EtO."[33]

In fact, Stayner's study is titled: "Exposure-Response Analysis of Cancer Mortality in a Cohort of Workers exposed to Ethylene Oxide."[34] And Stayner agrees that dose response is important. "So in terms of general causation, which we're interested in here, dose response is a key factor, and that's what we see convincingly in the tables in this paper."[35]

Dr. Salem confirms the dose-response relationship at high levels of exposure. The occupational breast cancer studies are dose-dependent and they involve high

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[29] Salem Dep. 167:15-18.
[30] Salem Dep. 178:7-17.
[31] Salem Dep. 84:23-25.
[32] Salem Dep. 229:2-230:3.
[33] Salem Dep. 255:1-5.
[34] Salem Dep. 226:21-24. (Emphasis added.)
[35] Stayner Dep. 237 :13-17.

exposure to EtO. "[I]ncreased exposure leads to increased incidence of breast cancer."[36] Animal studies are dose-dependent as well. They subject laboratory animals to high doses.[37]

In other words, the higher the dose, the more likely the harm. But there is no evidence that the harm begins at a minimal level. Dr. Salem agrees that studies show low levels do not increase chromosomal aberrations.[38] Unfortunately, this is the requirement set out in *McClain*[39] and one which Plaintiffs do not meet.

The Court's analysis does not stop after finding a lack of dose-response relationship in this case. The Court of Appeals stated that other methodologies could establish general causation in a Category 2 toxic tort case. It asked this Court to consider epidemiological studies and the background risk of disease. And although Plaintiffs' experts do refer to these alternative methodologies, the experts do not carry the day in this regard.

4. Epidemiology

Although a dose-response relationship may not be attainable in this case, the 11th Circuit has recognized two other primary methodologies as reliable: epidemiological studies and background risk of the disease in the general population. *See McClain*, *Chapman v. Procter & Gamble Distrib., LLC*, 766 F.3d 1296, 1306 (11th Cir. 2014); *cf. In re Abilify (Aripiprazole) Prods. Liab. Litig.*, 299 F. Supp. 3d 1291, 1306 (N.D. Fla 2018). The Court now turns to the epidemiological evidence, or lack thereof, in this case.

"Epidemiology is the branch of science that studies the incidence, distribution, and cause of disease in human populations and examines the pattern of disease in human populations." *In re Deepwater Horizon Belo Cases*, 119 F.4th 937 (11th Cir. 2024). Toxicology is

[36] Salem Dep. 25-280:1.
[37] Salem Dep. 184:3-22.
[38] Salem Dep. 167:19-25.
[39] *McClain* at 1242.

about dose; epidemiology is not. Epidemiological observational studies often cannot gauge dose, given that they are looking at real-world exposures that have already happened. So, while reliable toxicological evidence examines dose, reliable epidemiological evidence—the best kind of causation evidence in an environmental emissions case—does not always. *Id.* In this environmental emissions case, as in *DH Belo*, *Taylor* and *Williams*, the Court finds that epidemiological evidence may provide the strongest causation evidence since dose may not be "discoverable". However, as discussed below, Plaintiffs' experts fail to satisfy the standards established in those cases when relying on epidemiology as the principal method for establishing causation.

Types of epidemiological studies include:

1. observational cohorts, which compare the incidence of disease between exposed and unexposed groups;
2. case control studies, which compare the frequency of exposure between groups with the disease and those without; and
3. cross-sectional studies, which collect data from a certain group to see if each individual has the disease or not at a particular point in time.

See id. at 945. Experts relying on epidemiological studies to show general causation must identify an association between a particular substance and a particular disease, and whether that association shows a cause-effect relationship.

This second step typically involves considering the "Bradford Hill" factors:

1. temporal relationship;
2. strength of the association;
3. dose-response relationship;
4. replication of the findings;
5. biological plausibility;
6. consideration of alternative explanations;
7. cessation of exposure;

8. specificity of the association; and

9. consistency with other knowledge.

Id. No one factor is dispositive, and scientists may reasonably interpret the factors in different ways. *See Abilify*, 299 F. Supp. 3d at 1307.

The Court is cognizant that studying diseases in humans caused by potentially beneficial medicines or drugs is vastly different than those caused by toxic chemicals, heavy metals, or solvents not meant to be ingested by humans. So, it is natural that many of the seminal cases in this area, like *McClain, Chapman*, and *Abilify* which emphasize the importance of epidemiological studies and the dose-response relationship, are drug cases, not toxin cases. Because exposing humans to toxins for research raises ethical concerns, the Court recognizes that the studies and evidence typical in drug cases may not be available here. The Court considers this limitation when reviewing Plaintiffs' expert testimony. That said, this scientific reality does not lessen Plaintiffs' burden to produce reliable evidence on causation.

4.1. Epidemiological studies must be evaluated by Bradford Hill factors.

Drs. Stayner and Felsher's Bradford Hill analysis of the epidemiological studies was cursory. As such, their opinions are unreliable to show a true causal relationship to reach the conclusion that the association they purportedly identified reflected a true causal relationship. If an expert claims to use the Bradford Hill criteria, "the specific techniques by which the weight of the evidence/Bradford Hill methodology is conducted must themselves be reliable according to the principles articulated in *Daubert*," in that "all of the relevant evidence must be gathered, and the assessment or weighing of that evidence must not be arbitrary but must itself be based on methods of science." *DH Belo* at 941.

Dr. Stayner testified that when an epidemiologist weighs the evidence for a given exposure causing a specific type of cancer, "[t]here should be a strong association between the presence of an exposure and the occurrence of the

cancer[,]." [40] However, none of the studies Dr. Stayner cited involving EtO showed a statistically significant relative risk.[41] *See also, Allison v. McGhan Med. Corp.*, 184 F.3d 1300, 1315 n.16 (11th Cir. 1999) (stating "[t]he threshold for concluding that an agent more likely than not caused a disease is 2.0" and noting "we do not think the district court abused its discretion in finding a 1.24 risk minimal in terms of causation.")

When questioned regarding the application of the Bradford Hill criteria to dose-response, Dr. Stayner stated that these criteria are not used in analyses involving multiple studies, but are applied only when evidence is derived from a single study.[42] When asked if he applied the Bradford Hill criteria to his opinion in these cases, Dr. Stayner testified that he did not go through the checklist but considered them.[43]

Another Plaintiffs' expert, Dr. Felsher, testified that he applied the Bradford Hill criteria.[44] However, as Dr. Felsher's report indicates, if he applied the Bradford Hill criteria, he did so only to assert that EtO has generally been demonstrated to cause cancer.[45] Plaintiffs' expert Dr. Felsher states it this way. "Evidence from human epidemiolocal studies is strong but less than conclusive in associating specific cancers with EtO exposure."[46] When asked to identify the strongest association between EtO and cancer in any epidemiological study, Dr. Felsher characterized the question as a non sequitur.[47]

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[40] Stayner Dep. 72:14-73:13.
[41] *Id.*
[42] Stayner Dep. 71:19-25.
[43] *Id.*
[44] Felsher Dep. 40:8-14.
[45] Felsher Report p. 6-7.
[46] Felsher Report p. 25.
[47] Felsher Dep. 373:12-13.

Another instance of Dr. Felsher's conflicting testimony involves the Jinot paper[48] referenced in his deposition and cited by him in his report.[49] The Jinot paper is a review article authored by three EPA employees "as part of [their] official duties as an Employee of the United States," which discusses the "key finding and scientific issues addressed in EPA's studies."[50] The Jinot paper did not conclude that any level of EtO exposure above background levels causes cancer. Furthermore, the Jinot paper acknowledged that the EPA authors' Bradford Hill analysis is limited when determining the strength of association and dose-response relationship between EtO and cancer in occupational epidemiologic studies. Those studies only had **moderate** evidence of consistency for associations between EtO and lymphohematopoietic cancer and breast cancer. But Dr. Felsher stated the association between EtO and cancer was a **strong** association based on government and public health agencies' classification of EtO as a known carcinogen, rather than on epidemiological studies.[51]

Again, lack of the use of Bradford Hill criteria, internal inconsistencies in their reports and testimony, and the almost exclusive reliance on regulatory studies make Dr. Stayner's and Dr. Felsher's opinions suspect and therefore unreliable. They fall short of the requirements of O.C.G.A. § 24-7-702, *Daubert* and *McClain*.

4.2. Epidemiological studies are not well-suited to answer the question of low dose thresholds.

In fact, epidemiology is not helpful at low doses. In his deposition, Dr. Stayner states, "[O]ne of my key points is that you really can't because when you go down to lower and lower exposures, because the risk becomes smaller and smaller,

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[48] *Carcinogenicity of Ethylene Oxide: Key Findings and Scientific Issues*, Jennifer Jinot, Jason M. Fritz, Suryanarayana V. Vulimiri & Nagalakshmi Keshava (2018).
[49] Felsher Dep. 62:19-21, 63:24-25.
[50] Felsher Dep. 73:6-74:4.
[51] Felsher Report p. 10.

you reach certainly a point at which the epidemiology is uninformative because it's unable to detect that level of risk." [52] Dr. Stayner opines that even one day is a risk but epidemiology could not detect it.

> Q: Let's ---is it your opinion that a day—one day of exposure to ethylene oxide in the environment could be enough to cause cancer?
> A: I think in principle yes, we say that there's no threshold for saying that any exposure increases risk. Now, one day may be a trivial risk, certainly a risk probably that epidemiology would not be able to detect. But under the assumption that there is a threshold relationship between this genotoxic chemical and cancer, then a risk of one day—there should be some finite risk, albeit very small."[53]

Without data, epidemiological studies cannot prove causation in this case. "[W]hether the epidemiological data is sufficient or not on its own to say if there's a causal association, I'd say it's debatable because IARC reviews come back and said no, the data was limited." [54] This quote proves EtO is not a Category 1 toxin. As such, Plaintiffs' experts cannot prove general causation through epidemiological studies.

4.3. Dr. Felsher argues that epidemiological studies support the conclusion that no safe level of exposure to EtO exists. However, his deposition testimony contradicts this point.

Dr. Felsher writes in his report, "In conclusion, epidemiologic studies have shown there is an association between EtO and cancer that when EtO is used as a continuous variable there is no level that is not associated with increased risk..."[55] This is in contradiction to his deposition testimony where he repeatedly states there

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[52] Stayner Dep. 34:7-12.
[53] Stayner Dep. 56:24-57:11.
[54] Stayner Dep. 274:7-11.
[55] Felsher Report p. 9.

is a risk "above background." This topic was extensively analyzed in the Court's November 22, 2024 Order.[56] As such, his epidemiological opinion is excluded.

DH Belo affirms that general causation experts who rely on epidemiology must determine the specific exposure levels at which a chemical becomes harmful and must also meet the stringent requirements of valid epidemiological analysis. *DH Belo* at 941. Drs. Stayner and Felsher do not meet these essential criteria. This case therefore provides a roadmap— indeed a cautionary tale—for litigants and experts in toxic exposure litigation, underscoring the necessity of methodological rigor and precision throughout the process.

Experts may rely on other, non-epidemiological evidence to prove causation; but where epidemiology is lacking, "the nature of the other evidence ... becomes that much more important, and [a] court's consideration of such evidence and the methodologies used must be that much more searching." *See*, *Kilpatrick v. Breg, Inc.*, 613 F.3d 1329, 1337 n.9 (11th Cir. 2010). With that instruction in mind, this Court now addresses another methodology, referred to as background risk of disease.

5. Background Risk of Disease

5.1. Occupational studies cannot establish causation under *McClain* because they depend on high-dose exposure, and the studies do not consider the background risk of disease.

It should be noted that the only studies which showed a correlation between exposure to EtO and cancer in humans are the occupational studies.[57] And occupational studies cannot prove general causation in this case. First, because they analyze the effect of high doses of EtO. Epidemiological studies on worker exposure consist of "cohorts of workers who were exposed to very high, episodic levels of EtO for very short times, as opposed to the continual exposure facing individuals living

[56] *See* pgs. 13-18.

[57] Stayner Dep. 275:4-9.

near an emissions source."[58] Conducting occupational studies inherently distorts data. "And Mikoczy and Stayner and Steenland particularly point out that it's important to do internal comparison analyses, because all the worker studies, the epidemiological studies, have been compromised of their various distortions of data that occur as a consequence of doing a worker study."[59] Dr. Felsher again describes the limitation of using high doses in occupational studies. "[B]ecause all the worker studies tend to use cumulative dose, and it's very biased for the people exposed to extremely high doses in short periods of time, which is very different effect in a human, an organism, than even a significantly lower dose but for a much longer period of time."[60] In other words, comparing the incidence of cancer in worker studies to the incidence of cancer in the areas surrounding the Sterigenics plant is like comparing apples to oranges.

And, second, the occupational studies do not take into consideration the background risk of disease. *McClain* sets out the background risk of disease as an important factor in determining general causation in a Category 2 toxic tort case. "[T]he likelihood that the chemical caused the disease or illness in an individual should be considered in the context of other known causes." A reliable methodology should take into account the background risk. The background risk is not the risk posed by the chemical or drug at issue in the case. It is the risk the general public has of suffering the disease or injury that plaintiff alleges <u>without</u> exposure to the drug or chemical in question. The background risks include all those causes of a disease, whether known or unknown, excluding the drug or chemical in question. [61] Lacking a background risk of disease level is fatal to establishing general causation in a toxic

[58] Felsher Report p. 28.
[59] Felsher Dep. 68:5-12.
[60] Felsher Dep. 252:19-25.
[61] *McClain* at 1243.

tort case. "[W]ithout background risk as a baseline, determining whether an association is anything more than a coincident becomes difficult, if not impossible."[62]

Assessing background risk for disease is especially problematic for EtO as it is not only exogenous, but endogenous.[63] "The human body also produces low levels of EtO that has been ascribed to being 'endogenous' EtO."[64] At least one study, by Vernon Walker, states that a control group is difficult because chromosomal aberrations can occur in "control populations of humans and experimental animals without known exposure" to EtO.[65] Dr. Salem is unqualified to opine on his own whether the background risk for disease methodology proves causation in this case because he was unaware of the background risk for disease for breast cancer, leukemia, lymphoma, or birth defects.[66] Furthermore, Dr. Salem acknowledges occupational studies relied upon by Plaintiffs' experts are not good for background risk of disease analysis.[67] They do not compare workers to general population.[68] Stayner agrees. "[T]here's great differences between the general population and your study population."[69] The Mikoczy study has the same limitations. It did not compare workers to general population.[70] As such, the background risk of disease methodology does not survive *Daubert* analysis to establish general causation in this case.

Also, these occupational exposure studies involved workers who were exposed for more concentrated levels of EtO for much longer periods of time than

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[62] *In Re: Deepwater Horizon Belo Cases*, 119 F.4th 937, 942 (2024).
[63] Salem Dep. 102:9-19.
[64] Felsher Report p. 29.
[65] Salem Dep. 101:18-102-19.
[66] Salem Dep. 269:22-270:22.
[67] Salem Dep. 253:20-25.
[68] Salem Dep. 284:16-285:5.
[69] Stayner Dep. 241 :1-2.
[70] Salem Dep. 282:9-283:3.

Plaintiffs. Plaintiffs' experts failed to explain how studies showing an association between cancer and multiple years of occupational EtO exposure could be used to conclude there is an association between Plaintiffs' injuries: persons whose alleged exposure is/was not concentrated, is/was not measurable, and is/was confounded by numerous other exposures. *See* this Court's Phase 2 opinion.

6. Other Flaws Undermine Plaintiffs' Experts' Reliability.

6.1. Unsubstantiated analogies to other toxic substances do not establish an "any exposure standard" in a Category 2 toxic tort case.

Although nothing prevents a plaintiff from alleging their exposure to a mixture of chemicals caused their injury, using a study which identifies an association between one mixture of chemicals and a certain injury to infer that exposure to a different mixture of chemicals can also cause that injury does not reflect a sound methodology, absent some rational explanation for why such an inference is permissible. *See Hendrix ex rel. G.P. v. Evenflo Co., Inc.*, 609 F.3d 1183, 1196-97 (11th Cir. 2010) (noting courts will admit expert opinions on general causation that are supported by epidemiological studies, "provided the expert explains how the findings of those studies may be reliably connected to the facts of the particular case.") (citation omitted).

Dr. Stayner's testimony as to "any exposure increases risk" is akin to Dr. O'Donnell's testimony in *McClain*. They both make unsubstantiated analogies to other substances. In *McClain*, O'Donnell likens the combination of caffeine and ephedrine to another class of drugs, sympathomimetics. The 11th Circuit found Dr. O'Donnell's testimony lacking in this regard. At least in *McClain*, Dr. O'Donnell provided a detailed analysis of the similarities between sympathomimetics and Metabolife. Here, Dr. Stayner likens the any-exposure-to-EtO-increases-risk to risks

associated with radiation.[71] However, Dr. Stayner provides no support for his statement that the "mechanism of action is similar to radiation, which most scientific organizations (National Research Council 2006) assume has no safe level of exposure (i.e., threshold)."[72] The Court finds this testimony *ipse dixit* and that Plaintiffs' experts, including Dr. Stayner, have not established a dose-response threshold for the harm alleged by Plaintiffs in accordance with the *McClain* decision.

6.2. Plaintiffs' expert Dr. Felsher's reliance on ecological studies is insufficient and unreliable.

During his deposition, Dr. Felsher acknowledged that ecological studies are primarily "hypothesis-generating" and can only suggest, rather than establish, a causal association.[73] He also admitted that the ecological study he referenced "[a]bsolutely" had "significant limitations" and agreed that "any observed increase in and of itself is insufficient to draw conclusions regarding the potential impact of ethylene oxide exposure."[74] Furthermore, several other ecological studies that Dr. Felsher neither considered nor addressed did not find increased incidences of cancer near EtO emission sources.[75]

Similarly, Dr. Salem in his report cites ecological studies. But in his deposition he admittedly only relied on two ecological studies.[76] Also in his deposition, he admits, as Dr. Felsher did, that ecological studies are unreliable. "Well, I would say what happened is the studies are – there's a mix... And that's what you'd expect from an underpowered methodology that can't take into account other parameters."[77]

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[71] Stayner Report p. 1.
[72] *Id.*
[73] Felsher Dep. 335:2-17; 336:15-23.
[74] *Id.* at 336:24-337:12.
[75] *Id.* at 339:14-346:13, 349:17-354:10.
[76] Salem Dep. 265:6-271:15.
[77] Salem Dep. 338:16-22.

Consequently, the studies supporting Drs. Felsher and Salem's opinions that low-level environmental exposure to EtO from Defendants' facility can cause cancer does not provide a reliable basis for that conclusion. Accordingly, Drs. Felsher and Salem's opinions should be excluded under O.C.G.A. § 24-7-702 and *Daubert*.

6.3. Plaintiffs' experts fail to prove causation in this Category 2 toxic substance case through alternative methodologies suggested by the Court of Appeals.

Experts may use secondary methodologies as part of their overall analysis to support a primary methodology, but the failure to use at least one primary methodology is fatal. *See Chapman*, 766 F.3d at 1308; *see also Abilify*, 299 F. Supp. 3d at 1306. Aside from primary methodologies, secondary methodologies used to support a general causation opinion, include:

1. biologically plausible explanations for the substance's mechanism of action,
2. anecdotal case studies or reports such as adverse event reports and de-challenge/re-challenge tests,
3. hypotheses,
4. animal studies, and
5. extrapolations from analogous drugs.

See Chapman, 766 F.3d at 1308; *McClain*, 401 F.3d at 1245–47, 1250, 1253–55; *Abilify*, 299 F. Supp. 3d at 1306. Experts may also combine various methodologies in weighing the entire body of scientific evidence to form an overall general causation opinion, which is known as the "weight of the evidence" approach. *See Abilify*, 299 F. Supp. 3d at 1311–12; *cf. Waite*, 194 F. Supp. 3d at 1313–16. But even under this approach, experts may not combine multiple unreliable methodologies to add up to a reliable one. On the contrary, each step in the weighing process must be reliable. *See Abilify*, 299 F. Supp. 3d at 1311–12.

In this case, Plaintiffs' experts attempt to combine several unreliable secondary methodologies in an effort to meet the standards established in *Daubert* and *McClain;* however, these approaches are not supported by any of the three primary methodologies. Courts have consistently determined that establishing general causation requires only one of the three primary methodologies, rather than reliance on multiple secondary methodologies. Although secondary methodologies may be considered, they must be supported by at least one primary methodology to withstand scrutiny under O.C.G.A. 702, *Daubert,* and *McClain*.

5.6. Equivocal statements regarding the mechanics of disease progression were found to be below the *Daubert* standard in *McClain* and are also found to be below that standard here.

Plaintiffs' experts opine that they know the mechanism that establishes EtO's link to cancer. But their testimony falls short of the *Daubert* standard, and the standard articulated in *McClain*. Like Dr. O'Donnell's *McClain* testimony, Dr. Stayner's testimony and is filled with equivocations and leaps in logic. When Dr. O'Donnell testified about ephedrine's family or drug class connection and effects,

> ...he left a trail of equivocation by making the following statements at various points in his testimony: Sympathomimetics can constrict blood vessels. And when you constrict blood vessels, you may raise blood pressure. Sympathomimetics stimulate the heart and increase the pulse, increase the heart rate. If you stimulate the heart, you may cause an abnormal heart rate or an abnormal heart rhythm. If you constrict blood vessels, if it happens in a cerebral vessel in the brain, it may cause vasospasm which may lead to a stroke. If you stimulate or cause a constriction in the coronary blood vessel that can cause vasospasm and it may lead to chest pain, angina, arrhythmia, or myocardial infarction. He also testified that "aggravation of blood pressure is something that the ephedrine and caffeine in Metabolife or any product containing those drugs can do." He further explained that the ephedrine/caffeine combination "can elevate blood pressure and stimulate the

heart, and it has been reported to be associated with strokes and heart attacks." [78]

Or as O'Donnell stated: "this may be dangerous for some patients."[79]

Dr. Stayner testified similarly.

> So first of all, I think most scientists I know who are genetic or who study genetics and mutagenicity would agree that, that there's—for genotoxic and mutagenic, a compound that's direct acting that there would be no threshold in linear low doses. Part of it comes from something call multistage theory that you need two changes in the DNA to get an increase in cancer. And the first could be any molecule in theory could cause the first change, and then a second exposure to something else could cause a second or maybe spontaneously there could be a mutation. But , anyway, based on theory, it's been, I think—long been contention of many scientists who do work in the field of mutagenicity that the mechanism of action of ethylene oxide, which by the way is similar to things like radiation, that there's no—that there's a general belief, let's say, that it's unlikely there's a safe level of exposure or threshold.[80]

Dr. Salem also uses equivocating language when opining about mechanistic theories. "When we look at a molecular mechanism of action, we can say, you know, that a DNA adduct may have formed. And that DNA adduct can lead to gene mutations, and those gene mutations can lead to cancer or other diseases, but that's a separate statement from the risk of it happening because if you have a DNA adduct form, there is the potential it can be repaired."[81] He continues. "So I think the accurate statement is 'can cause,' DNA adducts can cause mutations."[82] Dr. Salem's testimony as to breast cancer is similarly equivocal. "[T]here is a correlation between ethylene oxide exposure and breast cancer that is worthy of further exploration." [83]

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[78] *McClain* at 1240. (Emphasis in original.)
[79] *Id.*
[80] Stayner Dep. 33:7-34:2. (Emphasis added.)
[81] Salem Dep. 71:24-72:7. (Emphasis added.)
[82] Salem Dep. 73:17-19. (Emphasis added.)
[83] Salem Dep. 279:7-10. (Emphasis added.)

This quote alone proves EtO is not generally accepted by the medical community as causing Plaintiffs' alleged harms. As the 11th Circuit found in *McClain*, this Court must find that this testimony does not meet the *Daubert* standard.

6. Conclusion

If an expert fails at any step, Daubert requires exclusion. "The requirement that the expert testify to scientific knowledge -- conclusions supported by good grounds for each step in the analysis -- means that any step that renders the analysis unreliable under Daubert renders the expert's testimony inadmissible." *McClain* at p. 1245, citing *Amorgianos v. AMTRAK*, 303 F.3d 256, 267 (2002) (quoting *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 745 (3rd Cir. 1994)).

Cognizant of the directives of the Court of Appeals and after an exhaustive de novo review of *McClain* and its progeny, the parties' briefing and oral argument, deposition testimony, reports and exhibits of Plaintiffs' three general causation witnesses, Drs. Stayner, Salem and Felsher, the Court finds as follows:

- EtO is a Category 2 substance.
- Plaintiffs' experts fail to establish a threshold dose level where harm to humans can occur that satisfies the *McClain* standard.
- Plaintiffs' expert opinions are not reliably based on other methodologies to pass muster under a O.C.G.A. § 24-7-702 and *Daubert* analysis and *McClain*.
- Plaintiffs' expert opinions are unreliable, and they fail to meet the helpfulness standard therefore their exclusion is mandated.

This Court finds Plaintiffs' experts' opinions are inadmissible and summary judgment for Defendants is mandated.

SO ORDERED this 30th March 2026.

Jane P. Manning, Judge
State Court of Cobb County