

**IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF MISSISSIPPI
SOUTHERN DIVISION**

MATTHEW WILLIAMS

PLAINTIFF

v.

CAUSE NO. 1:22CV278-LG-BWR

**BP EXPLORATION &
PRODUCTION INC.
and BP AMERICA
PRODUCTION COMPANY**

DEFENDANTS

**MEMORANDUM OPINION AND ORDER GRANTING DEFENDANTS'
MOTION FOR SUMMARY JUDGMENT, GRANTING DEFENDANTS'
MOTIONS TO EXCLUDE THE OPINIONS OF DR. JAMES J.J.
CLARK AND DR. MICHAEL FREEMAN, AND FINDING
AS MOOT ALL REMAINING PENDING MOTIONS**

BEFORE THE COURT are the [83] Motion for Summary Judgment, the [69], [71], [73], [75], [77], [79], [81], [85], [87] Motions to Exclude Expert Testimony, and the [105], [107], [109] Motions in Limine filed by Defendants BP Exploration & Production Inc. and BP America Production Company (hereafter collectively referred to as "BP") in this lawsuit that arose out of the April 20, 2010, Deepwater Horizon Oil Spill. Also before the Court is Plaintiff Matthew Williams' [111] Motion for Leave to File Tardy Motion in Limine. After reviewing the submissions of the parties, the record in this matter, and the applicable law, the Court finds that BP's Motions to Exclude Dr. Michael Freeman and Dr. James J.J. Clark should be granted. Because Plaintiff Matthew Williams cannot demonstrate causation, BP's Motion for Summary Judgment is also granted. All other pending Motions will be denied as moot.

BACKGROUND

After the BP oil spill Plaintiff, Matthew Williams performed oil spill clean-up work in Mississippi during the summer of 2010. (Compl. at 1, ECF No. 1). On September 24, 2020, Plaintiff was diagnosed with chronic pansinusitis.¹ On June 10, 2022, he filed this Back-End Litigation Option (“BELO”) lawsuit alleging that exposure to oil, dispersants, and other chemicals while performing oil spill response work caused his pansinusitis condition.

BP has filed numerous motions seeking to exclude Williams’ proposed expert witnesses, who have offered testimony and opinions on causation. BP seeks summary judgment arguing that that Williams cannot demonstrate causation as required by Mississippi tort law.

DISCUSSION

I. BP’S [83] MOTION FOR SUMMARY JUDGMENT

A motion for summary judgment may be filed by any party asserting that there is no genuine issue of material fact, and that the movant is entitled to prevail as a matter of law on any claim. Fed. R. Civ. P. 56. The movant bears the initial burden of identifying those portions of the pleadings and discovery on file, together with any affidavits, which it believes demonstrate the absence of a genuine issue of material fact. *Celotex Corp. v. Catrett*, 477 U.S. 317, 325 (1986). Once the movant

¹ “By definition, sinusitis implies an inflammation of the sinus mucosa lining the sinus cavity.” Overview—Generally, 9 Attorneys Medical Advisor § 107:5. “If many or all sinuses are infected, the terms “polysinusitis” and “pansinusitis” are used, respectively.” *Id.*

carries its burden, the burden shifts to the non-movant to show that summary judgment should not be granted. *Id.* at 324-25. The non-movant may not rest upon mere allegations or denials in its pleadings but must set forth specific facts showing the existence of a genuine issue for trial. *Anderson v. Liberty Lobby, Inc.*, 477 U.S. 242, 256-57 (1986). Factual controversies are resolved in favor of the non-moving party, but only when there is an actual controversy, that is, when both parties have submitted evidence of contradictory facts. *Little v. Liquid Air Corp.*, 37 F.3d 1069, 1075 (5th Cir. 1994).

In an unpublished opinion, the Fifth Circuit applied the toxic tort standard for causation to a case concerning an illness allegedly caused by the BP oil spill.² *Prest v. BP Expl. & Prod., Inc.*, No. 22-30779, 2023 WL 6518116, at *2 (5th Cir. Oct. 5, 2023). In toxic tort cases, “[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs’ burden in a toxic tort case.” *Allen v. Pa. Eng’g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996). These two requirements are referred to as “general causation” and “specific causation,” respectively. *See Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 351 (5th Cir. 2007).

² The Eleventh Circuit and numerous district courts, including the Southern District of Mississippi, have also held that the toxic tort standard applies. *In re Deepwater Horizon BELO Cases*, No. 20-14544, 2022 WL 104243, at *2 (11th Cir. Jan. 11, 2022); *Curbelo v. BP Expl. & Prod., Inc.*, No. CV 17-3690, 2023 WL 2742136, at *3 (E.D. La. Mar. 31, 2023); *Salmons v. BP Expl. & Prod. Inc.*, No. 1:20-CV-38-LG-RPM, 2021 WL 2149206, at *4 (S.D. Miss. May 26, 2021).

To establish general causation, the plaintiff must show that “a substance is capable of causing a particular injury or condition in the general population.” *Id.* A plaintiff demonstrates specific causation with evidence that “a substance caused [that] particular [plaintiff’s] injury.” *Id.* If the court finds that the plaintiff has not demonstrated general causation, there is no need to consider specific causation. *Id.* Thus, plaintiffs in BP oil spill cases “must prove, at a minimum, that exposure to a certain level of a certain substance for a certain period of time can cause a particular condition in the general population.” *Williams v. BP Expl. & Prod.*, No. 18-9753, 2019 WL 6615504, at *8 (E.D. La. Dec. 5, 2019) (citing *Knight*, 482 F.3d at 351). Such proof must be established through expert testimony. *Wells v. SmithKline Beecham Corp.*, 601 F.3d 375, 381 (5th Cir. 2010); *Prest*, 2023 WL 6518116, at *3.

Williams’ claim hinges on whether he can demonstrate that exposure to a substance is capable of causing a particular injury or condition in the general population and that his exposure to that particular substance caused his pansinusitis. Those causation elements are dependent upon the testimony of Williams’ proposed experts— Dr. Freeman and Dr. Clark. BP argues that absent admissible expert testimony regarding general and specific causation they are entitled to judgement as a matter of law.

II. BP’S MOTIONS TO EXCLUDE WILLIAMS’ EXPERT WITNESSES

The party offering the proposed expert has the burden of proving by a preponderance of the evidence that the expert’s proffered testimony satisfies Rule

702 of the Federal Rules of Evidence. *Mathis v. Exxon Corp.*, 302 F.3d 448, 459-60 (5th Cir. 2002). Rule 702 provides that an expert witness “who is qualified as an expert by knowledge, skill, experience, training, or education” may testify 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.

Fed. R. Evid. 702. Therefore, “expert testimony is admissible only if it is both relevant and reliable.” *Pipitone v. Biomatrix, Inc.*, 288 F.3d 239, 243 (5th Cir. 2002). “Reliability is determined by assessing whether the reasoning or methodology underlying the testimony is scientifically valid. Relevance depends upon whether that reasoning or methodology properly can be applied to the facts in issue.” *Knight*, 482 F.3d at 352 (quotation marks, citations, and brackets omitted). The Court may consider the following factors in determining reliability: (1) whether the technique has been tested, (2) whether the technique has been subjected to peer review and publication, (3) the technique’s potential error rate, (4) the existence and maintenance of standards controlling the technique’s operation, and (5) whether the technique is generally accepted in the relevant scientific community. *Burleson v. Tex. Dep’t of Crim. Just.*, 393 F.3d 577, 584 (5th Cir. 2004). These factors “do not constitute a ‘definitive checklist or test.’” *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 150 (1999) (quoting *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 593 (1993)). Rather, courts “have considerable leeway in deciding in a particular case

how to go about determining whether particular expert testimony is reliable.” *Id.* at 152. “Although there are ‘no certainties in science,’ the expert must present conclusions ‘ground[ed] in the methods and procedures of science.’ In short, the expert must ‘employ[] in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field.’” *Wells*, 601 F.3d at 378.

A. BP’S [75] MOTION TO EXCLUDE DR. FREEMAN

In his report, Dr. Freeman offers opinions pertaining to the fields of forensic medicine and forensic epidemiology. (Def.’s Mot., Ex. B at 2, ECF No. 75-2). The Fifth Circuit has acknowledged that “the most useful and conclusive type of evidence in a case such as this is epidemiological studies.” *Brock v. Merrill–Dow Pharms., Inc.*, 874 F.2d 307, 311 (5th Cir. 1989), *modified on reh’g*, 884 F.2d 166 (5th Cir. 1989), *cert. denied*, 494 U.S. 1046 (1990).

Epidemiology attempts to define a relationship between a disease and a factor suspected of causing it To define that relationship, the epidemiologist examines the general population, comparing the incidence of the disease among those people exposed to the factor in question to those not exposed. The epidemiologist then uses statistical methods and reasoning to allow her to draw a biological inference between the factor being studied and the disease’s etiology.

Id. at 311. Since epidemiology considers whether an agent is capable of causing a disease, it pertains to general causation, not specific causation. Michael D. Green et al., Reference Guide on Epidemiology, *Reference Manual on Scientific Evidence* 549, 2011 WL 7724261, at *2 (Fed. Judicial Ctr., 3d ed. 2011)).

“[T]he first question an epidemiologist addresses is whether an association exists between exposure to the agent and disease. An association between exposure to an agent and disease exists when they occur together more frequently than one would expect by chance.” *Id.* at *10. Importantly, “an association is not equivalent to causation.” *Id.* at *2.

Assessing whether an association is causal requires an understanding of the strengths and weaknesses of the study’s design and implementation, as well as a judgment about how the study findings fit with other scientific knowledge. It is important to emphasize that all studies have “flaws” in the sense of limitations that add uncertainty about the proper interpretation of the results. Some flaws are inevitable given the limits of technology, resources, the ability, and willingness of persons to participate in a study, and ethical constraints. In evaluating epidemiologic evidence, the key questions, then, are the extent to which a study’s limitations compromise its findings and permit inferences about causation.

Id. Courts are permitted to reject epidemiological studies that do not show a statistically significant increase in disease. *LeBlanc ex rel. Est. of LeBlanc v. Chevron USA, Inc.*, 396 F. App’x 94, 99 (5th Cir. 2010) (citing *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 145 (1997)).

If an association has been found between exposure to a substance and development of a disease, researchers next look for alternative explanations of the association, such as bias or confounding factors.³ Only after that process has been

³ In this context, “bias” refers “to anything that results in a systematic (nonrandom) error in a study result and thereby compromises its validity.” *Id.* at *19. For example, “[r]esearch has shown that individuals with disease (cases) tend to recall past exposures more readily than individuals with no disease (controls); this creates a potential for bias called recall bias.” *Id.* Meanwhile, “[c]onfounding occurs when another causal factor (the confounder) confuses the relationship between the agent of interest and outcome of interest.” *Id.* at *24. One possible confounding factor in

completed, “researchers consider how guidelines for inferring causation from an association apply to the available evidence.” Reference Guide on Epidemiology, 2011 WL 7724261, at *28. Sir Austin Bradford-Hill’s factors are generally used by epidemiologists to make this determination. *Id.*; see also *Yarbrough v. Hunt S. Grp., LLC*, No. 1:18CV51-LG-RHW, 2019 WL 4392519, at *4 (S.D. Miss. Sept. 12, 2019) (explaining that the Bradford-Hill factors are “widely used in the scientific community to assess general causation”). These factors are: 1. the strength of the association; 2. the consistency of the association; 3. the specificity of the association; 4. the temporal relationship of the association; 5. whether there is a dose-response relationship⁴; 6. whether causation is biologically plausible; 7. the coherence of the association, 8. the presence of experimental evidence, and 9. evidence by analogy. *Yarbrough*, 2019 WL 4392519, at *2.

1. DR. FREEMAN’S OPINIONS CONCERNING GENERAL CAUSATION

In his Report, Dr. Freeman cites several epidemiological studies in an attempt to establish an association between oil spill response work and sinusitis: (1) the health hazard evaluations of off-shore and on-shore oil spill response workers by the National Institute for Occupational Safety and Health (NIOSH); (2) the D’Andrea and Reddy study of the acute effects of BP oil spill response exposures; (3)

some studies is smoking. *Id.* at *25. “To evaluate whether smoking is a confounding factor, the researcher would stratify each of the exposed and control groups into smoking and nonsmoking subgroups to examine whether subjects’ smoking status affects the study results.” *Id.*

⁴ Dr. Freeman calls this factor “biological gradient.”

the GuLFSTUDY (Gulf Long-Term Follow-up Study); (4) the Rusiecki Deepwater Horizon Oil Spill Coast Guard Cohort Study and 5-year follow-up study; and (5) the Lawrence study; and (6) the study concerning the *Prestige* oil spill off the coast of Spain in November 2002. After discussing each study, Dr. Freeman applies the Bradford-Hill factors. He claims that the strength of association factor is satisfied because the “NIOSH data revealed that 18.0% of shoreline workers that reported any exposure to dripping oil, tar balls, dispersant, or dust also reported sinus problems while only 4.7% of those that indicated no exposure to any of the 4 reported sinus problems.” (Def.’s Mot., Ex. B at 29, ECF No. 75-2). He also notes that the seven-year follow-up of the D’Andrea and Reddy study “showed that 91% of the oil spill exposed subjects [sic] had developed chronic rhinosinusitis compared with none at baseline.” (*Id.*) He claims that the five-year follow-up results of the Rusiecki Deepwater Horizon Oil Spill Coast Guard Cohort Study “demonstrated responders reporting exposure to crude oil inhalation had elevated risks for all sinusitis . . . and unspecified chronic sinusitis . . .” (*Id.*)

Dr. Freeman also opines that these three studies “demonstrate consistent associations between exposure to products of the . . . oil spill and sinusitis or chronic sinusitis.” (*Id.*) Concerning specificity, Dr. Freeman explains, “While not exclusive to environmental exposures, chronic upper respiratory disorders are closely related to the inhalation of environmental toxins . . .” (*Id.*) He next states, “The temporal relationship described in the literature is appropriate in sequence and proximity. The chain of causation between the acute and chronic symptoms in the populations

studied over time is clearly established, and contiguous.” (*Id.*) As for biological gradient or dose-response relationship, Dr. Freeman contends that his analysis of the NIOSH survey data “revealed significant dose-response relationships between the duration and frequency of exposure and self-reported acute sinus problems.” (*Id.*)

Dr. Freeman also notes that the risk of having persistent respiratory symptoms increased for responders to the *Prestige* oil spill increased with the degree of exposure. (*Id.* at 30). He opines that there is a biologically plausible link between exposure to chemicals during oil spill response and both acute and chronic inflammatory conditions of the upper respiratory system. (*Id.*) He then asserts that the coherence factor is satisfied because “[i]t certainly ‘makes sense’ that exposure to inhaled irritants can cause acute and chronic sinusitis.” (*Id.*) He relies on an experiment performed on Nordic rats⁵ “exposed by inhalation of fuel oil-derived volatile organic compounds produced to mimic an oil spill” to support his claim that the experiment factor is satisfied. (*Id.*) Finally, he opines that the data obtained from *Prestige* oil spill study satisfies the analogy factor. (*Id.*)

BP argues that Dr. Freeman’s opinions should be excluded because his opinions on general causation are not reliable. BP cites two BELO opinions in which Dr. Freeman’s opinions were previously rejected — *Dufour v. BP Exploration & Production Inc.*, No. 1:19-CV-591-HSO-BWR, 2023 WL 3807923 (S.D. Miss. June

⁵ The Fifth Circuit has noted that “the results of animal studies are inconclusive at best.” *Allen*, 102 F.3d at 195.

2, 2023), and *In re Deepwater Horizon BELO Cases*, No. 3:19CV963-MCR-HTC, 2022 WL 17721595, at *19 (N.D. Fla. Dec. 15, 2022), *report and recommendation adopted In re Deepwater Horizon BELO Cases*, No. 3:19CV963, 2023 WL 2711573 (N.D. Fla. Mar. 30, 2023).

In *Dufour*, Dr. Freeman offered opinions concerning both general and specific causation, asserting, “In the case of Richard Allen Dufour, Jr., it is my opinion, to a reasonable degree of medical and scientific certainty, that exposures to oil spill-associated irritants during his response work were a substantial factor in causing his asthma to worsen during his employment and to persist afterward.” 2023 WL 3807923, at *8. The court determined that Dr. Freeman’s opinions were unreliable for numerous reasons, including his failure to identify the substances to which the plaintiff was actually exposed, his failure to provide “any meaningful analysis” of the epidemiological studies he cited, and his improper use of the Bradford-Hill criteria in an attempt to establish specific causation. *Id.* at *9. Williams correctly notes that the *Dufour* decision is distinguishable to the extent that Dufour claimed that his asthma was aggravated by oil spill clean-up work while Williams claims he developed pansinusitis. Dr. Freeman admitted that he “did not find any studies that directly evaluated aggravation of preexisting asthma in BPDWH oil spill clean-up workers.” *Id.*

In the *Deepwater Horizon BELO Cases*, Dr. Freeman stated that “[t]he results of [his] critical review of the relevant scientific and epidemiologic evidence support a general causal relationship between occupational exposure to chemicals

associated [with] the BP Deepwater Horizon oil spill” and subsequent chronic rhinosinusitis.⁶ 2022 WL 17721595, at *19. He explained, “Based upon the adjusted hazard ratio for chronic sinusitis associated with crude oil inhalation, there is a minimum additional 55% risk attributable to the exposure, among those who have been exposed and have the condition.” *Id.* The Magistrate Judge recommended that the district court find Dr. Freeman’s opinions to be unreliable because: (1) in his initial report, he “fail[ed] to identify a particular chemical or mixture of chemicals” capable of causing the plaintiff’s chronic rhinosinusitis; (2) he did not “describe a threshold dose of a chemical” that was capable of causing the plaintiff’s illness; (3) his use of the NIOSH data was “flawed” because, *inter alia*, it was a cross-sectional study with the goal of describing acute health effects that was based on workers’ self-reported exposures and symptoms; (4) his reliance of the Rusiecki 2022 study was “problematic” because he failed to address inconsistencies within the study and he “cherry picked a favorable analysis from the Rusiecki study while disregarding the analyses which undermined his ultimate opinion on general causation”; and (5) he failed to address the many limitations of the D’Andrea study. *Id.* at 20-22.

Ultimately, the Magistrate Judge found that “Dr. Freeman failed to meet the first step for relying on epidemiological data — that is, he failed to identify a reliable statistical association between exposure and chronic sinusitis . . . in the

⁶ Dr. Freeman also offered opinions concerning chronic conjunctivitis and dry eye, which are not at issue in the present case. *Id.* at *18.

epidemiological literature.” *Id.* at 23. She also determined that Dr. Freeman’s analysis of the Bradford-Hill factors was “cursory and superficial.” *Id.* She determined that opinions included in a supplemental report provided by Dr. Freeman should be stricken as both untimely and unreliable. *Id.* at 25-26.

BP notes that Dr. Freeman utilized “substantially the same methodology” when arriving at his opinions in the present case as he did in *Dufour* and the *Deepwater Horizon BELO Cases*. During his deposition, he testified that he combined his original and supplemental opinions in *Dufour* when writing his report in the present case. (Def.’s Mot., Ex. E at 17-18, ECF No. 75-5). However, he explained, “I tried to understand, at least from Mr. Dufour’s case, what the issues that [sic] were raised by the Court and try to make sure there wasn’t ambivalence in my report about any of those issues.” (*Id.* at 17). Williams argues that the orders striking Dr. Freeman’s opinions in the *Dufour* and *Deepwater Horizon BELO* cases are not relevant here because:

[T]his is the first Deepwater Horizon case in which Dr. Freeman submitted one rigorously complete report (without an accompanying supplemental report) that encompasses novel studies which show association, a thorough Bradford-Hill analysis of said studies, a discussion of the dose-response relationship, an identification of a level of exposure to oil-derived chemicals harmful to humans generally, and an exposure assessment of the Plaintiff followed by a differential etiology evaluation.

(Pl.’s Resp. at 6, ECF No. 98).

In the present case, the first question raised by BP is whether Dr. Freeman’s failure to identify a harmful dose or specific chemical renders his general causation opinion unreliable. General causation requires a finding that “a substance is

capable of causing a particular injury or condition in the general population.” *Knight*, 482 F.3d at 351. Therefore, a plaintiff must demonstrate “[s]cientific knowledge of the harmful level of exposure to a chemical.” *Allen*, 102 F.3d at 199.

Dr. Freeman admitted that he has not identified which chemicals “were within the toxins that the workers were exposed to,” and he will “leave that up to the toxicologists and environmental folks.” (Def.’s Mot., Ex. G at 18-19, ECF No. 75-7). As the Magistrate Judge explained in the Northern District of Florida BELO test cases, Dr. Freeman’s failure to identify a particular chemical or mixture of chemicals.

is problematic, as not all workers had the same types of exposures and the available epidemiological studies do not all address the same exposure scenario. Furthermore, the crude oil emitted from the wellhead underwent weathering as it traveled to the shore, which changed its chemical composition. Dr. Freeman acknowledged as much in his report, as he noted volatile organic compounds found in crude oil evaporate within hours of reaching the water’s surface.

In re Deepwater Horizon BELO Cases, 2022 WL 17721595, at *20.

Dr. Freeman claims that the harmful dose of these unidentified chemicals is included in Table 2 on page 20 of his report concerning Williams. (*Id.* at 13; Def.’s Mot., Ex. B at 20, ECF No. 75-2). Utilizing data from the NIOSH study, Dr. Freeman prepared this table in order to compare the number of workers who reported sinus problems with the number of days they performed oil spill response work. (*Id.*) This data caused Dr. Freeman to reach the conclusion that the longer a person performed response work, the more likely they were to report sinus problems. (Def.’s Mot., Ex. G at 15-21, ECF No. 75-2). He determined that, “for

every 30-day increase, there's a 43 percent increased odds [sic] that you're going to have sinus problem exposure." (*Id.* at 19).

Dr. Freeman's alleged dose-response opinion is unreliable because it is based on data from the NIOSH study, which concerns self-reported, acute sinus symptoms, not medical diagnoses of chronic sinus conditions like the one claimed by Williams. *See Dufour*, 2023 WL 3807923, at *12; *In re Deepwater Horizon BELO Cases*, 2022 WL 17721595, at *20-21. The study specifically states that its assessments were not intended to describe or investigate potential long-term or chronic health effects" like those claimed by Williams. (Def.'s Mot., Ex. E at 27-28, ECF No. 75-5). Furthermore, the NIOSH study is a cross-sectional study,⁷ which is "rarely useful in identifying toxic agents." *See in re Deepwater Horizon BELO Cases*, 2022 WL 17721595, at 20 (citing Reference Guide on Epidemiology, 2011 WL 7724261, at *4). Dr. Freeman agrees that a cross-sectional study is insufficient to establish causation on its own, but he believes that the effect of the substance measured by the cross-sectional study can be supported by the Bradford-Hill criteria in order to show causation. (Def.'s Mot., Ex. E at 41-43, ECF No. 75-5). Dr. Freeman's argument is circular because his analysis of the Bradford-Hill factors is largely based on the NIOSH data. (*See* Def.'s Mot., Ex. B at 29, ECF No. 75-2).

⁷ In a cross-sectional study, "individuals are interviewed or examined, and the presence of both the exposure of interest and the disease of interest is determined in each individual at a single point in time. . . ." Reference Guide on Epidemiology, 2011 WL 7724261, at *7. "[B]ecause both exposure and disease are determined in an individual at the same point in time, it is not possible to establish the temporal relation between exposure and disease--that is, that the exposure preceded the disease, which would be necessary for drawing any causal inference." *Id.*

Furthermore, the use of the Bradford-Hill factors “in the absence of any epidemiologic studies finding an association” between exposure and disease “does not reflect accepted epidemiologic methodology.” Reference Guide on Epidemiology, 2011 WL 7724261, at 28, n.141. As a result, the Bradford-Hill factors cannot shore up the sufficiency of the NIOSH study for establishing an association between oil spill clean-up work and sinusitis.

The other studies that Dr. Freeman cites also do not support Dr. Freeman’s findings of an association between oil spill response exposure and chronic pansinusitis. First, the D’Andrea and Reddy study pertained to workers who worked in oil spill clean-up for over three months, while Williams performed response work for only two months, between June 23, 2010, and August 27, 2010. (*Id.* at 5, 23). Dr. Freeman does not address this discrepancy. Like the NIOSH study, the D’Andrea and Reddy study was a cross-sectional study that is insufficient to establish causation on its own.

D’Andrea and Reddy also investigated acute effects, while Williams claims chronic effects. (*Id.*) The study did not review the workers’ medical records from before the oil spill to see whether symptoms preexisted the spill, and the study’s control group had different demographic characteristics from the response workers. (*Id.* at 23). While Dr. Freeman emphasizes that 91% of the 44 who returned for a seven-year follow up study complained of chronic sinus problems, he brushes over “[t]he fact that only 38% of workers returned for reassessment.” (*Id.* at 24). The follow-up study did not quantify the number of unexposed workers who reported

chronic sinusitis during the seven-year follow up or compare that figure with the number of returning exposed workers who reported sinusitis. *See in re Deepwater Horizon BELO Cases*, 2022 WL 17721595, at *22. Therefore, the relative risk of exposure could not be calculated. Finally, and perhaps most importantly, the D'Andrea and Reddy study did not perform adjustments to the data to account for workers who smoked. (*Id.*)

Dr. Freeman's discussion of the GuLF STUDY is likewise problematic. This study concluded that "[p]otential exposure to Corexit™ EC 9527A or EC9500A was associated with a range of health symptoms at the time of the [oil spill response and cleanup], as well as at the time of study enrollment, 1-3 [years] after the spill." (Def.'s Mot., Ex. I at 21, ECF No. 75-9). As with other studies cited by Dr. Freeman, the emphasis was on self-reported symptoms rather than diagnoses. Furthermore, the study did not mention sinusitis but only generally discussed coughing, wheezing, tightness in the chest, shortness of breath, burning in the nose, throat, and lungs, burning eyes, itchy eyes, and skin irritation. Therefore, the GuLF STUDY does not support an association between oil spill response exposure and pansinusitis or any other form of sinusitis.⁸

⁸ In his report, Dr. Freeman also discussed the Lawrence study, which used the GuLF STUDY data to evaluate the risk of response workers developing asthma. (Def.'s Mot., Ex. B, at 25-26, ECF No. 75-2). However, Williams has not claimed that his exposure caused him to develop asthma, and Dr. Freeman has not adequately explained the relevance of the evaluation of asthma risk to Williams' case.

Similarly, the five-year follow-up study of results from the Rusiecki Deepwater Horizon Oil Spill Coast Guard Cohort Study cited by Dr. Freeman was based on self-reported exposure information. (*Id.* at 27).

Calculated in terms of an adjusted hazard ratio a/k/a relative risk ratio, the study found no increased risk of chronic sinusitis (1) between Coast Guard responders versus non-responders (the largest group); (2) between Coast Guard responders reporting exposure to oil versus those who reported no exposure; (3) between Coast Guard responders reporting exposure to oil and dispersants versus those who did not report any exposure; and (4) Coast Guard responders in the vicinity of in-situ burns versus those who were not there. Interestingly, the study also found a reduced risk for sinusitis for those USCG responders exposed to both crude oil and dispersants and a reduced risk for chronic rhinitis for those who were exposed to oil versus those who were not.

Although the study found no statistically significant association between (1) chronic sinusitis and exposure to crude oil between responders and non-responders, (2) those responders exposed to crude oil versus those who had never been exposed; or (3) those who were exposed to in situ burns versus those who had never been exposed; the study found an increased risk in chronic sinusitis, at a hazard ratio of 1.48 for chronic sinusitis and 1.55 for unspecified chronic sinusitis, for Coast Guard responders who reported inhalation exposure to oil. However, when the confounders for smokers were removed (i.e., the study was restricted to “never” smokers), the results were statistically insignificant.

In re Deepwater Horizon BELO Cases, 2022 WL 17721595, at *10–11 (internal citations omitted). Dr. Freeman does not take these findings into account, which underscores his report’s unreliability.

Since Dr. Freeman has not identified any reliable statistically significant association between oil spill response work and chronic sinusitis, his opinions are unreliable and inadmissible. Even if he had identified an association, his analysis of the Bradford-Hill factors is similarly lacking.

The first factor is strength of association. As explained above, Dr. Freeman has not identified a statistically significant association using the studies he cited. For the second factor, consistency, Dr. Freeman claims the NIOSH, D'Andrea/Reddy 7-year follow-up study, and the Rusiecki 5-year follow-up study demonstrate “consistent associations.” (Def.’s Mot., Ex. B at 29, ECF No. 75-2).

[T]hose three studies did not examine the same exposure-disease relationship. *See* Ref. Man. at 604 (“Different studies that examine the same exposure-disease relationship generally should yield similar results.”). As discussed above, the NIOSH data involved acute symptoms while the other two studies involved chronic sinusitis. Similarly, Dr. Freeman relied on the Rusiecki study for its findings based on crude oil inhalation, while the other two studies included additional exposure scenarios. And within the Rusiecki study itself, the analyses of different types of exposure did not show consistent associations. Thus, Dr. Freeman's explanation of the consistency factor is misplaced and indicates he did not reliably apply the Bradford Hill criteria.

In re Deepwater Horizon BELO Cases, 2022 WL 17721595, at *23.

Dr. Freeman cites the NIOSH study and data from the *Prestige* oil spill to support his claim that the biological gradient or dose-response relationship factor is satisfied. Once again, Dr. Freeman has not identified which chemicals Williams was exposed to, so a determination of dose-response relationship has not been made. Furthermore, the NIOSH study did not concern chronic conditions, so it cannot support a finding of dose-response relationship between oil spill exposure and chronic sinusitis. He cites a study concerning rats for the experiment factor, and the *Prestige* oil spill findings for the analogy factor. He does not address the limitations of these studies, such as the potential for recall bias in the *Prestige* study, where workers answered questions in a telephonic interview six years after

the spill. He provides vague, conclusory statements regarding the factors of specificity, temporality, plausibility, and coherence.

For expert witnesses, “[t]he existence of sufficient facts and a reliable methodology is in all instances mandatory.” *Hathaway v. Bazany*, 507 F.3d 312, 318 (5th Cir. 2007). “[W]ithout more than credentials and a subjective opinion, an expert’s testimony that ‘it is so’ is not admissible.” *Id.* (quoting *Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 424 (5th Cir. 1987)). Dr. Freeman’s opinions on general causation to not satisfy the standards of Fed. R. Evid. 702.

2. DR. FREEMAN’S OPINION CONCERNING SPECIFIC CAUSATION

Out of an abundance of caution, the Court will also consider whether Dr. Freeman’s specific causation opinion is reliable. To determine specific causation, Dr. Freeman used the differential etiology approach, which “evaluates whether there are alternative plausible causes for a specific plaintiff and to reach a conclusion as to which cause is most likely.” (*Id.* at 13). He opines, without further explanation or analysis:

There are 6 broad theories on CRS etiology and pathogenesis: (1) the “fungal hypothesis,” (2) the superantigen hypothesis,” (3) the “biofilm hypothesis,” and (4) the “microbiome hypothesis,” all of which emphasize key environmental factors, and (5) “the eicosanoid hypothesis” and the “immune barrier hypothesis.” Because Mr. Williams did not have any significant medical problems prior to his employment as part of the BPDWH oil spill response, the likelihood of any of these possible competing explanations is exceedingly small.

(Def.’s Mot., Ex B at 30, ECF No. 75-2).

BP correctly notes that Dr. Freeman did not describe any attempts to evaluate whether any of the potential alternate causes he lists could have caused Williams' pansinusitis, and there is no discussion of Williams' subsequent work experience or home environment in Dr. Freeman's report.

BP also argues that Dr. Freeman's specific causation opinion fails to consider Williams' medical records, which do not mention any sinus symptoms until eight years after he performed the oil spill clean-up work. (Def.'s Mot., Ex. K, ECF No. 75-11). Dr. Freeman conceded at his deposition that he had never seen "anything that indicates that somebody who has no complaints at all in their airway for five years after an exposure would suddenly develop a condition that could be attributed to that exposure." (Def.'s Mot., Ex. E at 90, ECF No. 75-5).

"A court may rightfully exclude expert testimony where a court finds that an expert has extrapolated data, and there is 'too great an analytical gap between the data and the opinion proffered.'" *Burleson v. Texas Dep't of Crim. Just.*, 393 F.3d 577, 587 (5th Cir. 2004) (quoting *Joiner*, 522 U.S. at 146). An opinion that oil spill response work caused sinusitis for which no medical treatment was sought until eight years after exposure must be supported by more facts and analysis than that provided by Dr. Freeman. Since Dr. Freeman's general and specific causation opinions are both unreliable and inadmissible, it is not necessary for the Court to consider BP's argument that he is not qualified to give a specific medical causation opinion.

B. BP'S [69] MOTION TO EXCLUDE DR. CLARK

The parties dispute whether Dr. Clark has offered causation opinions. BP claims that Dr. Clark “attempts to backdoor inadmissible causation opinions in the form of his unreliable risk estimates.” (Def.’s Mem. at 5, ECF No. 84). Williams counters that Dr. Clark “was disclosed to offer testimony on [c]ausation and to conduct an exposure assessment, in which Dr. Clark offers quantitative doses of exposure to particular chemicals that Plaintiff Matthew Williams was exposed to during his approximate four (4) months of clean-up work during the Deepwater Horizon Oil Spill.”⁹ (Pl’s Resp. at 2, ECF No. 91). BP has filed a separate Motion to Exclude Dr. Clark’s opinions. As a result, the Court must determine whether Dr. Clark offers admissible causation opinions and testimony that would foreclose summary judgment.

Dr. Clark used the Environmental Protection Agency’s Risk Assessment Guidance for Superfund (“RAGS”) to evaluate Williams’ exposures. (Def.’s Mot., Ex. C at 8, ECF No. 69-3). The EPA “uses risk assessment to characterize the nature and magnitude of health risks to humans and ecological receptors from chemical contaminants and other stressors that may be present in the environment.” United States Environmental Protection Agency, “Risk Assessment,” <https://www.epa.gov/risk>.

⁹ While Williams claims he performed oil spill clean-up work for approximately four months, both Dr. Freeman and Dr. Clark state that Williams worked between approximately June 24, 2010, to August 31, 2010, or sixty-eight days. The Court has not located Williams’ deposition transcript or employment records in the record.

The EPA has explained:

The final step of risk assessment is risk characterization. This involves combining the exposure quantities . . . and the toxicity benchmarks . . . to calculate the excess lifetime cancer risks (risk) and noncancer hazards (hazard) for each of the pathways and receptors identified in Chapter 4. . . .

Risk from exposure to combustor emissions is the probability that a human receptor will develop cancer, based on a unique set of exposure, model, and toxicity assumptions. . . . In contrast, **hazard** is the potential for developing noncancer health effects as a result of exposure to COPCS. A hazard is not a probability but, rather, a comparison (calculated as a ratio) of a receptor's potential exposure relative to a standard exposure level (RfD or RfC). The standard exposure level is calculated over a similar exposure period and is estimated to pose no appreciable likelihood of adverse health effects to potential receptors, including special populations

(Def.'s Mot., Ex. B at 7-2, ECF No. 69-9). The RfD, or "reference dose," is a "daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a 70-year lifetime." (*Id.* at 7-6). An RfC, or "reference concentration" is "an estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations." (*Id.*). "The comparisons of oral and inhalation exposure estimates to RfD and RfC values . . . are known as hazard quotients (HQ)" (*Id.*). The HQ is calculated by either dividing the average daily dose by the RfD or dividing the exposure air concentration by the RfC. (*Id.*). The EPA Superfund has determined that no adverse health effects are expected at a hazard index of 1. (*Id.*).

However, because RfDs and RfCs do not have equal accuracy or precision, and are not based on the same severity of effect, the level of concern does not increase linearly as an HQ approaches and exceeds 1.

In addition, noncancer estimates only identify the exposure level below which adverse effects are unlikely; and RfD or RfC does not say anything about incremental risk for higher exposures.

(*Id.*).

In his report, Dr. Clark opines as follows:

1. Mr. Williams' written and oral testimony place him at locations which contained significant quantities of crude oil and the dispersants used by BP to facilitate the cleanup.
2. Clean up workers and others exposed involved in the BP Deepwater Horizon oil spill were exposed to crude oil and dispersants that contained complex mixture of chemicals of concern (COCs) including odorous compounds, volatile organic chemicals (VOCs), short and long chain aliphatic hydrocarbons (e.g., methane, ethane, butane, propane, pentane, heptane, octane, nonane), polyaromatic hydrocarbons (e.g., by increasing ring size naphthalene (Nap), anthracene (Ant), phenanthrene (Phe), pyrene (Pyr) and benzo(a)pyrene (BaP)), metals, and when burned or physically agitated particulate matter (PM).
3. Exposure to the COCs identified in this report will lead to the development of adverse health outcomes, including but not limited to respiratory distress, headaches, sinusitis, and rhinosinusitis.
4. Using a quantitative risk analysis approach from U.S. EPA, it is clear that Mr. Williams' calculated doses of petroleum hydrocarbons from dermal contact and his inhalation petroleum hydrocarbon vapors during his time as a deckhand, boat operator/rigger, and a crane operator in Mississippi Sound near Petit Bois Island increased his risk for developing adverse health outcomes, specifically his development of sinusitis.
5. Given Mr. Williams' work history, family history, exposure to the COC's released by the BP Deepwater Horizon Oil Spill, the doses of COCs that were calculated in this report, his symptoms at the time of his work for BP and his later work history, it is clear that his exposure to the COCs released by the BP Oil Spill were a substantial contributor to his development of sinusitis.

(Def.'s Mot., Ex. B at 6-7, ECF No. 69-2). During his deposition, he testified that this last opinion, which is based on the "totality of Williams's exposures and his

medical history,” “can be viewed as a specific causation [opinion].” (Def.’s Mot., Ex. C at 88-89, ECF No. 69-3). He explained:

I think what I’ve done with my opinion is provide a general overview, general causation, that he was exposed to a number of compounds during his time as a worker, and that in the literature I see relationships between exposure to VOCs and the development of headaches and an inflammatory response that goes along the way. And buttoning this up at the end, that looks pretty specific.

(*Id.* at 89). He agreed that Williams’s total non-cancer risk, which includes other conditions in addition to sinusitis, was 0.0101. (*Id.* at 90). He also conceded that this figure is “100 times lower than what the EPA deems acceptable for non-cancer risk.” (*Id.*) When asked how he could “possibly say” that a risk 100 times lower than the hazard index at which adverse health effects are expected is a “substantial contributor” to Williams’ development of sinusitis, he responded, “That’s my opinion that, given the totality of his exposure and his response, this is what I’m led to.” (*Id.* at 91-92). He further explained that the opinion is based on his thirty years of experience as well as the Rusiecki study. (*Id.* at 92-93). He claimed that this study showed that, as a whole, oil spill workers “ended up with more sinusitis than others five years after the fact.” (*Id.* at 84). He later acknowledged that the Rusiecki study found no significant associations between inhalation exposure to the oil spill clean-up and sinusitis when only non-smokers, like Williams, were considered. (*Id.* at 114-15).

Dr. Clark also cited the Lawrence study in his report. He admitted during his deposition that this study concerned asthma, not sinusitis, that 87% of the study participants who claimed they experienced wheezing had not been diagnosed with

asthma, and that the study authors found no statistically significant association between oil spill exposure and asthma when analysis was limited to participants who had been diagnosed with asthma by a physician. (*Id.* at 105-08). He claims that this study is relevant nevertheless because “[a]sthma is typically mediated by an inflammatory response . . . so it is relevant to the discussion about respiratory impacts.” (*Id.* at 109).

Dr. Clark’s testimony about other studies on which he relied was similarly concerning, due to study limitations that he failed to address as well as his reliance on studies that did not address sinusitis. He also admitted that the 0.0101 total non-cancer risk estimate that he calculated for Williams was “an overestimate based on [his] summing of hazard quotients and the assumption of additivity.” (*Id.* at 129-30).

Finally, it appears that Dr. Clark’s report was originally prepared for a different case because, at times, he mentions someone with the last name of Vincent instead of Williams. For example, Dr. Clark states, “Duration, frequency, and other input parameters were selected based upon reasonable estimates of exposure from the testimony of Mr. Vincent.” (Def.’s Mot., Ex. B at 48, EF No. 69-2). While typographical errors are understandable, one must beware of “cut and paste.” Dr. Clark’s research may be applicable to multiple cases, but the references to Vincent cause some doubt as to whether “cut and paste” findings in his report are correct since Vincent’s exposure levels and work experience may have been different from that of Williams.

The Court finds that Dr. Clark's purported general and specific causation opinions are unreliable and inadmissible. While he claims that his opinions are based on the EPA RAGS, his conclusions contradict RAGS. His attempts to explain this contradiction constitute impermissible ipse dixit, and he admitted that his report contained errors.

CONCLUSION

As the Fifth Circuit has stated, “[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain a plaintiff’s burden in a toxic tort case.” *Allen*, 102 F.3d at 199 (emphasis added). Since a plaintiff in a toxic tort case cannot expect lay factfinders to understand medical causation, expert testimony is required to establish causation. *Seaman v. Seacor Marine L.L.C.*, 326 F. App’x 721, 723 (5th Cir. 2009). Since the opinions of both of Williams’ proposed causation experts have been excluded as unreliable, Plaintiff cannot demonstrate the essential elements of causation. BP is therefore entitled to judgment as a matter of law. BP’s Motions to Exclude the opinions of Williams’ other proposed experts,¹⁰ BP’s Motions in Limine, and Williams’s Motion for Leave to File Tardy Motion in Limine will be denied as moot.

IT IS THEREFORE ORDERED AND ADJUDGED that the [69] Motion to Exclude Expert Testimony of Dr. James J.J. Clark and the [75] Motion to Exclude

¹⁰ The parties agree that Williams’ other proposed experts have not offered opinions concerning causation, so it is not necessary for the Court to address BP’s Motions to Exclude those experts.

Expert Testimony of Dr. Michael Freeman filed by Defendants BP Exploration & Production Inc. and BP America Production Company are **GRANTED**.

IT IS FURTHER ORDERED AND ADJUDGED that the [71], [73], [77], [79], [81], [85], [87] Motions to Exclude Expert Testimony, and the [105], [107], [109] Motions in Limine filed by Defendants BP Exploration & Production Inc. and BP America Production Company are **DENIED AS MOOT**.

IT IS FURTHER ORDERED AND ADJUDGED that the [111] Motion for Leave to File Tardy Motion in Limine filed by Plaintiff Matthew Williams is **DENIED AS MOOT**.

IT IS FURTHER ORDERED AND ADJUDGED that the [83] Motion for Summary Judgment filed by Defendants BP Exploration & Production Inc. and BP America Production Company is **GRANTED**. This lawsuit is **DISMISSED WITH PREJUDICE**. The Court will enter a separate judgment pursuant to Fed. R. Civ. P. 58(a).

SO ORDERED AND ADJUDGED this the 30th day of January, 2024.

sl Louis Guirola, Jr.

LOUIS GUIROLA, JR.
UNITED STATES DISTRICT JUDGE