

**UNITED STATES DISTRICT COURT  
SOUTHERN DISTRICT OF GEORGIA  
SAVANNAH DIVISION**

KAYLA SOPER,	)	
	)	
Plaintiff,	)	
	)	
v.	)	CV420-324
	)	
CHIPOTLE MEXICAN GRILL	)	
OF COLORADO, LLC,	)	
	)	
Defendant.	)	

**ORDER**

Before the Court is Defendant Chipotle Mexican Grill of Colorado, LLC’s (“Chipotle”) Motion to Exclude Opinion Testimony of Plaintiff Kayla Soper’s Expert Witness Dr. Michael Freeman. Doc. 27. Soper responded in opposition, doc. 32, and Chipotle replied, doc. 37. For the following reasons, Chipotle’s motion is **GRANTED**. Doc. 27.

**BACKGROUND**

Soper’s Complaint alleges that she suffered vomiting, diarrhea, and an eventual skin infection after dining at a Chipotle restaurant with her son and mother. Doc. 1-3 at 6-7. She shared a meal with her son at the restaurant, and her mother had a different meal. *Id.* at 6. That evening, her son “became violently ill with diarrhea and vomiting.” Plaintiff

subsequently experienced the same symptoms. *Id.* She noticed “an abscess in her rectal area” five days after the meal, *id.*, and a surgeon diagnosed her with a necrotizing soft tissue infection.<sup>1</sup> *See* doc. 29-9 at 16-17.

Soper identified Freeman as a retained expert in forensic medicine and epidemiology. Doc. 27-4 at 3; *see also* Fed. R. Civ. P. 26(a)(2)(B). He testifies that the most likely cause of both Soper’s initial gastrointestinal symptoms, *i.e.*, the vomiting and diarrhea, and her subsequent skin infection, is her consumption E. coli-contaminated food at Chipotle. *See, e.g.*, doc. 29-2 at 222-23. Chipotle argues that the Court should exclude his testimony because he is not qualified to render medical causation opinions, his methodology is unreliable, and his opinions “will not assist the trier of fact[.]” Doc. 27-1 at 2.

### ANALYSIS

Federal Rule of Evidence 702 compels the Court to act as a “gatekeeper” for expert evidence. *United States v. Frazier*, 387 F.3d 1244, 1260 (11th Cir. 2004) (citing *Daubert v. Merrell Dow Pharms., Inc.*, 509

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<sup>1</sup> Freeman refers to the infection as necrotizing fasciitis in his report and deposition. *See generally* doc. 29-2. The Court will refer to the condition as the “skin infection.”

U.S. 579, 589 n. 7, 597 (1993)). In performing this task, the Court must consider whether the party offering the evidence has shown:

(1) the expert is qualified to testify competently regarding the matters he intends to address; (2) the methodology by which the expert reaches his conclusions is sufficiently reliable as determined by the sort of inquiry mandated in *Daubert*; and (3) the testimony assists the trier of fact, through the application of scientific, technical, or specialized expertise, to understand the evidence or to determine a fact in issue.

*Frazier*, 387 F.3d at 1260 (quoting *City of Tuscaloosa v. Harcros Chems., Inc.*, 158 F.3d 548, 562 (11th Cir.1998)). The proponent of the expert opinion bears the burden of establishing qualification, reliability, and helpfulness by a preponderance of the evidence. *Allison v. McGhan Med. Corp.*, 184 F.3d 1300, 1306 (11th Cir. 1999) (citing *Daubert*, 509 U.S. at 592 n.10).

Under the first prong, “experts may be qualified in various ways. While scientific training or education may provide possible means to qualify, experience in a field may offer another path to expert status.” *Frazier*, 387 F.3d at 1260–61; *see also* Fed. R. Evid. 702 (a witness may be qualified as an expert by “knowledge, skill, experience, training, or education[.]”). But, “[w]hen an expert witness relies mainly on experience to show he is qualified to testify, ‘the witness must explain

how that experience leads to the conclusion reached, why that experience is a sufficient basis for the opinion, and how that experience is reliably applied to the facts.’” *Payne v. C.R. Bard, Inc.*, 606 F. App'x 940, 942-43 (11th Cir. 2015.) (quoting *Frazier*, 387 F.3d at 1261).

As to the second prong, the reliability “criterion remains a discrete, independent, and important requirement for admissibility.” *Frazier*, 387 F.3d at 1261. “The Supreme Court in *Daubert* set out a list of ‘general observations’ for determining whether expert testimony is sufficiently reliable to be admitted under Rule 702.” *United States v. Brown*, 415 F.3d 1257, 1267 (11th Cir. 2005) (citation omitted). These factors, or observations, inquire into the expert's “theory or technique” and are: “(1) whether it can be (and has been) tested; (2) whether it has been subjected to peer review and publication; (3) what its known or potential rate of error is, and whether standards controlling its operation exist; and (4) whether it is generally accepted in the field.” *Id.* (citation omitted). “Sometimes the specific *Daubert* factors will aid in determining reliability; sometimes other questions may be more useful.” *Frazier*, 387 F.3d at 1262. “Indeed, the Committee Note to the 2000 Amendments of Rule 702 expressly says that, ‘[i]f the witness is relying solely or primarily

on experience, then the witness must explain *how* that experience leads to the conclusion reached, why that experience is a sufficient basis for the opinion, and how that experience is reliably applied to the facts.’” *Id.* at 1261.

Lastly, expert testimony must assist the trier of fact. *Frazier*, 387 F.3d at 1262. “By this requirement, expert testimony is admissible if it concerns matters that are beyond the understanding of the average lay person.” *Id.* (citation omitted). This inquiry is commonly called the “helpfulness” inquiry. *Prosper v. Martin*, 989 F.3d 1242, 1249 (11th Cir. 2021) (citing *Frazier*, 387 F.3d at 1260). “Expert testimony which does not relate to any issue in the case is not relevant and, ergo, non-helpful.” *Id.* (quoting *Daubert*, 509 U.S. at 591).

Dr. Freeman’s report explains that he applied a three-step “causation analysis”:

- 1) Whether the investigated exposure had the potential to cause the disease in question (general causation), and if known, the magnitude of that potential (risk);
- 2) The degree of temporal proximity between the exposure and the onset of the symptoms reasonably indicating the presence of the illness; and
- 3) Whether there is a more likely alternative explanation for the occurrence of the illness at the same point in time, versus the investigated exposure (also known as a differential etiology/ diagnosis). This alternative or

competing cause is quantified for the individual, given their predictive characteristics and the temporal relationship quantified in [S]tep 2.

Doc. 29-2 at 209. The “differential etiology/ diagnosis” described in the third step is “a scientific technique where the expert identifies the cause of a medical problem by ‘eliminating the likely causes until the most probable one is isolated.’” *Longoria v. Ethicon, Inc.*, 2020 WL 7238151, at \*2 (M.D. Fla. Dec. 9, 2020) (quoting *In re C.R. Bard, Inc.*, 948 F. Supp. 2d 589, 602 (S.D.W. Va. 2013)). “When properly conducted, a differential diagnosis can be a reliable methodology under *Daubert*.” *Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245, 1253 (11th Cir. 2010). It “need not rule out all possible alternative causes[.]” *Id.* “The alternative causes . . . affect the weight that the jury should give the expert's testimony and not the admissibility of that testimony[.]” *Katsiafas v. C. R. Bard, Inc.*, 2020 WL 1808895, at \*2 (M.D. Fla. Apr. 9, 2020) (internal quotations omitted). “However, a ‘differential diagnosis that fails to take serious account of other potential causes may be so lacking that it cannot provide a reliable basis for an opinion on causation.’” *Dotson v. Am. Med. Sys., Inc.*, 2020 WL 2844738, at \*3 (N.D. Ga. Mar. 11, 2020) (quoting *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265 (4th Cir. 1999)); *see also*

*Katsiafas*, 2020 WL 1808895, at \*2 (differential etiology is not reliable if “the expert is unable to offer any explanation for his or her causation opinion in light of the alternative causes offered by the opposing party.”).

As discussed above, Freeman opines that the most likely cause of both Soper’s GI symptoms and her subsequent skin infection was *E. coli* from a contaminated Chipotle meal. Doc. 29-2 at 222-23. Soper tersely argues that Freeman conducted a sufficient differential etiology by quoting almost the entirety of his report’s “third step” analysis:

Given the history I obtained from [Soper during telephonic discussion] as well as the medical history I have reviewed, there are no competing alternative explanations our for [Soper and her son’s] acute gastroenteritis aside from a foodborne illness resulting from consumption of contaminated and tainted food item at Chipotle. While there are other *plausible* sources of foodborne illness in their history prior to the onset of their illness, none are sufficiently likely to be considered as likely competing explanations for the illness occurring in [Soper] (both her GI and NF illnesses) and [her son’s] GI illness.

Doc. 32 at 20-21; *see also* doc. 29-2 at 223 (original report). Far from “tak[ing] serious account” of alternative GI-symptom and skin-infection causes, *Westberry*, 178 F.3d at 265, this conclusory section does not explain why other “plausible sources” are “[in]sufficiently likely.” Doc. 29-2 at 223; *Jones v. Novartis Pharms. Corp.*, 235 F. Supp. 3d 1244, 1276

(N.D. Ala. 2017), (“Expert reports must include both ‘how’ and ‘why’ the expert reached a certain result, not just conclusory opinions.”). Although Freeman need not “rule out *all* possible alternative causes”, *Guinn*, 602 F.3d at 1253 (emphasis added), “mere conclusory statements that [he] considered and ruled out alternative explanations [are] . . . insufficient.” *Greger v. C.R. Bard, Inc.*, 2021 WL 3855474, at \*7 (E.D. Tex. Aug. 30, 2021). Accordingly, Soper falls short of showing that Freeman’s causation opinions clear *Daubert*’s “reliability” hurdle.

Even if Soper’s briefing had more robustly discussed Freeman’s etiology, the remainder of his report and deposition testimony do not establish that he sufficiently considered alternative causes. The parties imprecisely frame Freeman’s testimony as rendering two opinions: (1) that *E. coli* from a Chipotle meal is the most likely cause of Soper’s GI symptoms, and (2) that *E. coli* from Chipotle is the most likely cause of her skin infection. *See, e.g.*, doc. 27-1 at 6-7; doc. 32 at 1-2. The first opinion alone consists of at least two distinct component opinions: (1) that *E. coli* is the most likely cause of her GI symptoms,<sup>2</sup> and (2) assuming *E.*

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<sup>2</sup> *See, e.g.*, doc. 29-2 at 223 (Freeman opines that “the fact that *E. coli* was cultured from [Soper’s skin infection] in close temporal proximity to her acute [GI symptoms] *essentially dictates* that this organism was the cause.” (emphasis added)).



coli is the most likely cause of her E. coli symptoms, that the most likely source of the E. coli was her contaminated meal at Chipotle.<sup>3</sup> As discussed below, this first sub-opinion is **EXCLUDED** because Freeman’s report and deposition testimony do not include a sufficient discussion of the likelihood of non-E. coli pathogens causing the GI symptoms. Further, the unreliability of this threshold opinion undermines his remaining causation opinions.

Freeman spends much of his report explaining that E. coli likely caused Soper’s GI symptoms because it was cultured from her skin infection wound.<sup>4</sup> The closest he comes to discussing an alternative to this conclusion is his recognition that E. coli are “normal inhabitants of the human gastrointestinal tract”, doc. 29-2 at 210, and that the culture taken from the wound “wasn’t cultured for pathotype. It was just cultured for positive growth for E. coli[.]” *Id.* at 59. This discussion could

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<sup>3</sup> *See, e.g.*, doc. 29-2 at 223 (“The most likely (and only apparent) cause of the outbreak of foodborne infection described in this report was Kayla and [her son’s] consumption of contaminated food prepared at the Chipotle Restaurant[.]”).

<sup>4</sup> *See, e.g.*, doc. 29-2 at 210 (“E. coli was subsequently cultured from the [skin infection] wound on [Soper’s] left buttock, suggesting that organism was a likely candidate cause of the foodborne illness outbreak[.]”); *id.* at 223 (“[T]he fact that E. coli was cultured from [Soper’s skin infection] in close temporal proximity to her acute [GI symptoms] essentially dictates that this organism was the cause.”); *id.* at 216 (noting that E. coli “was the only organism identified in the infected area”).

be construed as acknowledging an alternative non-E. coli-GI-symptom cause. Specifically, he notes the possibility that the E. coli cultured from the wound could have originated in Soper's gastrointestinal tract, and that some pathogen other than E. coli could have caused the GI symptoms. The Eleventh Circuit, however, has explained that "[a] district court is justified in excluding evidence if an expert utterly fails . . . to offer an explanation for why the proffered alternative cause was ruled out." *Hendrix ex rel. G.P. v. Evenflo Co.*, 609 F.3d 1183, 1197 (11th Cir. 2010) (quoting *Clausen v. M/V New Carissa*, 339 F.3d 1049, 1058 (9th Cir. 2003) (quotations and citations omitted)). The Court cannot conclude that this opinion is reliable because Freeman does not articulate *why* the cultured E. coli less likely originated in her gastrointestinal tract than from a Chipotle meal. *See Wilson v. Pinnacle Foods Inc.*, 2022 WL 419595, at \*9 (W.D. Ky. Feb. 10, 2022) (excluding opinions when expert acknowledged "[relevant] alternative causes", and failed to rule them out).<sup>5</sup>

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<sup>5</sup> Freeman also testified that "if [Soper] had that much pathogenic E. coli in her gut, it's likely related to a foodborne illness[.]" Doc. 29-2 at 43. A vague, conclusory statement that the presence of "that much" E. coli at the skin infection site is "likely related" to the GI symptoms does not constitute a sufficient discussion of alternative causes, especially in light of his recognition that E. coli are "normal inhabitants" of the gastrointestinal tract. *Id.* at 210.

Freeman referenced two non-E. coli pathogens at his deposition: Campylobacter and Norovirus. Even if he raised them to acknowledge non-E. coli alternatives,<sup>6</sup> Soper cannot show that they support the reliability of his differential etiology. He mentions Campylobacter as an example of a pathogen that “takes a while to incubate”, doc. 29-2 at 44,<sup>7</sup> and he briefly discusses Norovirus as a pathogen that he “might be more inclined to look at” if E. coli had not been cultured from the skin infection. *See id.* at 71-72.<sup>8</sup> This offhanded reference to two non-E.coli pathogens

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Further, at his deposition, Freeman had the chance to clarify the relative likelihood of non-E.coli pathogens when he was directly asked “[why he] believe[s] that [Soper] had E. coli?” Doc. 29-2 at 57. The most charitable construction of his response is that E. coli “invaded the tissue” to cause Soper’s skin infection, which suggests that the GI symptoms were caused by E coli. *Id.* at 57. As discussed, a statement that E. coli was cultured from the skin infection does not address any alternative causes, including that alternative cause that Freeman himself raises.

<sup>6</sup> Given Freeman’s casual reference to these pathogens, the Court is not convinced that the testimony is an attempt to “provide reasons for rejecting alternative hypotheses using scientific methods and procedures[.]” *Chapman v. Procter & Gamble Distrib., LLC*, 766 F.3d 1296, 1310 (11th Cir. 2014) (internal quotation marks and citation omitted).

<sup>7</sup> *See, e.g.*, doc. 29-2 at 43 (“So, now that we know it's E. coli, what's the range of E. coli incubation times and durations? . . . Because there's sometimes, for example, if we were talking about Campylobacter and someone starts throwing up about an hour after a meal, you'd say, no way, that's not Campylobacter.”); *id.* at 45-46 (“So, if, for example, [Soper and her son] had started to throw up an hour, and gotten sick, an hour after they had eaten at Chipotle, and they had both had chicken, we wouldn't point to Chipotle and say, ‘That must be the source of a Campylobacter.’ Let's say she has a Campylobacter infection.”).

<sup>8</sup> *See also* doc. 29-2 at 96-97 (The only other time Freeman referenced Norovirus at his deposition is when he was asked “What’s more prevalent, a virus in a young child

falls short of “using scientific methods and procedures” to “enumerate a comprehensive list of alternative causes and to eliminate those potential causes.” *Chapman*, 766 F.3d at 1310.<sup>9</sup>

Several portions of the report could be construed as attempts to rule out alternative causes as less likely by stating that *E. coli* from Chipotle is *so likely* that other explanations are *unlikely*. See, e.g., doc. 29-2 at 210 (“[d]iarrheagenic *E. coli* are among the most frequent bacterial causes of [GI symptoms] worldwide”); *id.* at 219 (“*E. coli*, a common cause of foodborne illness in restaurant settings, . . .”). Statements about how commonly *E. coli* causes GI symptoms do not constitute a reliable differential etiology absent a comparison to the likelihood of other pathogens that might have caused Soper’s GI symptoms.

The remaining portions of the report and deposition which could be construed as discussions of alternative causes *assume* that *E. coli* was the

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or *E. coli*?” He responded “A virus . . . A Norovirus is going to be – any of the Enteroviruses are going to be much more likely in a kid like this.”)

<sup>9</sup> Similarly, the Court cannot conclude that Freeman considered alternatives to his opinion that the GI symptoms were related to a *foodborne* pathogen. See generally doc. 29-2. The report, for example, does not evaluate non-foodborne alternative causes. Instead, it purports to opine on “[t]he most likely (and only apparent) cause of the outbreak of [Soper’s] *foodborne infection*.” Doc. 29-2 at 223 (emphasis added).

most likely GI-symptom cause. For example, the report includes a “diet history” which evaluates the meals Soper ate on the day before and day of her Chipotle meal. Doc. 29-2 at 205. Freeman uses this history to evaluate whether a non-Chipotle meal caused the GI symptoms. *See id.* at 205, 220-222. The report describes the incubation times and symptoms of various E. coli pathotypes, *id.* at 212, and concludes that enteropathogenic E. coli (“EPEC”) is the most likely cause because its incubation time is most consistent with the time between Soper’s Chipotle meal and her symptoms, and it is associated with symptoms similar to the ones she experienced, *id.* at 222.<sup>10</sup> This analysis does not constitute a consideration of non-E. coli pathogen causes because it assumes that the GI symptoms were caused by E. coli, and does not explain whether Freeman considered other pathogens less likely than E. coli. *See also id.* at 50 (Freeman did not consider meals prior to those

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<sup>10</sup> *See also* doc. 29-2 at 211 (“The vomiting and diarrhea experienced by [Soper and her son] after they ate at Chipotle are consistent with diarrheagenic E. coli pathotypes. Furthermore, the timing of their symptom onset relative to when they visited the restaurant are also consistent with certain pathotypes[.]”); *id.* at 47 (“So we have about 13 hours in between illness for these two people. So that's kind of in the range of, you know, how people get ill, particularly from E. coli.”).

captured by the diet history because “this is E. coli, [and] E. coli typically isn’t going to wait around two days to manifest.”).<sup>11</sup>

In multiple instances, Freeman discusses the likelihood of E. coli from the Chipotle meal causing the GI symptoms by noting that Soper and her son shared a meal at Chipotle, her mother had a different meal, and only Soper and her son experienced GI symptoms. *See, e.g.*, doc. 29-2 at 51, 222; *see also id.* at 220-21 (explaining that the time between their shared meal and symptoms are consistent with the incubation time of several E. coli pathotypes). First, the report and deposition do not include an analysis of whether non-E. coli pathogens have similar incubation times. Second, even if Freeman uses the shared meal to opine that *a pathogen* from Chipotle is the most likely cause of the GI symptoms, he does not analyze the possibility of *non-foodborne pathogens* as alternative

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<sup>11</sup> Further, at his deposition, when asked whether Dr. Freeman “prepared any other timelines or looked at any other timelines shifting the timing of the exposure”, he responded “[w]ell, when I’m doing the initial analysis, of course.” Doc. 29-2 at 61. To the extent this constitutes testimony that he considered pathogen causes other than E. coli, as discussed above, “mere conclusory statements that [he] considered and ruled out alternative explanations [are] . . . insufficient.” *Greger*, 2021 WL 3855474, at \*7.

causes.<sup>12</sup> Accordingly, this discussion does not support the reliability of his causation opinion.

Since Soper has failed to show that Freeman adequately considered alternatives to his opinion that the GI symptoms were most likely caused by *E. coli*, that opinion is **EXCLUDED**. This threshold opinion underpins his first broad causation opinion: assuming *E. coli* is the most likely cause of the GI symptoms, the *E. coli* most likely originated from Soper's Chipotle meal. Since Soper has not shown that the threshold opinion is reliable, the broader causation opinion must be **EXCLUDED**. Further, Freeman uses this first broad causation opinion as a premise of his second broad causation opinion: that *E. coli* from the Chipotle meal is the most likely cause of Soper's skin infection. *Id.* at 219 ("Given [Soper's] acute diarrhea after dining at Chipotle, she was at increased risk for a breach to her inner buttock skin. Exposure of the breached skin to a diarrheagenic *E. coli* pathotype that had also acquired genetic material encoding a necrotizing factor . . . is a highly plausible cause of [her skin

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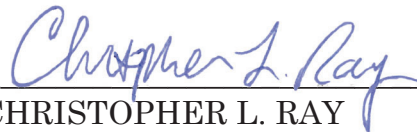
<sup>12</sup> In fact, much of Freeman's report definitively states that the GI symptoms' cause is foodborne without evaluating alternatives. *See generally* doc. 29-2. It purports to opine on "[t]he most likely (and only apparent) cause of the outbreak of [Soper's] *foodborne infection*." *Id.* at 223 (emphasis added).

infection].”). This opinion on the skin infection’s cause is **EXCLUDED**.<sup>13</sup>

### CONCLUSION

For the foregoing reasons, Chipotle’s Motion is **GRANTED**,<sup>14</sup> doc. 27, and his testimony is **EXCLUDED**.

**SO ORDERED**, this 12th day of July, 2022.



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CHRISTOPHER L. RAY  
UNITED STATES MAGISTRATE JUDGE  
SOUTHERN DISTRICT OF GEORGIA

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<sup>13</sup> Since Freeman’s testimony is excluded under *Daubert*’s “reliability” prong, the Court need not evaluate Chipotle’s arguments that he is “unqualified to opine as to the medical cause” of Soper’s GI symptoms and skin infection, doc. 27-1 at 16, and that those opinions will not “assist the trier of fact”, *id.* at 23.

<sup>14</sup> Since the exclusion of Freeman’s testimony is non-dispositive, the disposition does not require adoption by the District Judge. *See* 28 U.S.C. § 636; *Stewart v. Johnson*, 2021 WL 6752312, at \*1 (S.D. Ga. Aug. 10, 2021) (citing *Rodriguez v. GEICO Gen. Ins. Co.*, 2021 WL 1053156, at \*1 (M.D. Fla. Feb. 4, 2021) (“A *Daubert* motion is not a dispositive motion.”); *Villafana v. Auto-Owners Ins.*, 2007 WL 1810513, at \*1 (S.D. Ala. June 22, 2007) (“[T]he weight of authority holds that a magistrate judge’s order that excludes a plaintiff’s expert from testifying is not a dispositive ruling.”)).