IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
CHARLESTON DIVISION

IN RE: LIPITOR (ATORVASTATIN CALCIUM) MARKETING, SALES PRACTICES AND PRODUCTS LIABILITY LITIGATION	**MDL No. 2:14-mn-02502-RMG** **CASE MANAGEMENT ORDER NO. 55** **This Order relates to:** *Hempstead v. Pfizer, Inc.*, 2:14-cv-1879

In this MDL, Plaintiffs allege that Lipitor caused them to develop Type 2 diabetes. To carry their burden, Plaintiffs must prove both general and specific causation. *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 263 (4th Cir. 1999). The Court will address Plaintiffs' general causation testimony by separate order. The Court makes no finding here as to whether the testimony of Plaintiffs' general causation experts is admissible or whether Plaintiffs can survive summary judgment on the issue of general causation in this particular case. For the purposes of this motion, the Court assumes that general causation can be established in this case and turns to Plaintiffs' specific causation expert testimony.

Plaintiff has disclosed two case-specific expert witnesses in this bellwether case, Dr. Daniel Handshoe and Dr. Elizabeth Murphy. Both experts opine that Lipitor caused, or was a substantial contributing factor of, Ms. Hempstead's Type 2 diabetes. Pfizer has moved to exclude the testimony of both experts. (Dkt. Nos. 1004, 1006). Here, the Court addresses Pfizer's motion to exclude the testimony of Dr. Elizabeth Murphy. (Dkt. No. 1006).

I. Legal Standard

Under Rule 104(a) and 702, "the trial judge must ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable." *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 589 (1993). Thus, the trial court must ensure that (1) "the testimony is the product of reliable principles and methods," that (2) "the expert has reliably applied the principles and methods to the facts of the case," and (3) that the "testimony is based on sufficient facts or data." Fed. R. Evid. 702(b), (c), (d). "This entails a preliminary assessment of whether the reasoning or methodology underlying the testimony is scientifically valid," *Daubert,* 509 U.S. at 592-93, and whether the expert has "faithfully appl[ied] the methodology to facts." *Roche v. Lincoln Prop. Co.*, 175 F. App'x 597, 602 (4th Cir. 2006)

Factors to be considered include "whether a theory or technique . . . can be (and has been) tested," "whether the theory or technique has been subjected to peer review and publication," the "known or potential rate of error," the "existence and maintenance of standards controlling the technique's operation," and whether the theory or technique has garnered "general acceptance." *Daubert,* 509 U.S. at 593-94; *accord United States v. Hassan*, 742 F.3d 104, 130 (4th Cir. 2014). However, these factors are neither definitive nor exhaustive, *United States v. Fultz*, 591 F. App'x 226, 227 (4th Cir. 2015), *cert. denied*, 135 S. Ct. 2370 (2015), and "merely illustrate[] the types of factors that will bear on the inquiry." *Hassan*, 742 F.3d at 130. Courts have also considered whether the "expert developed his opinions expressly for the purposes of testifying," *Wehling v. Sandoz Pharm. Corp.*, 162 F.3d 1158 (4th Cir. 1998), or through "research they have conducted independent of the litigation," *Daubert v. Merrell Dow Pharm., Inc.*, 43 F.3d 1311, 1317 (9th Cir. 1995) (on remand), and whether experts have "failed to meaningfully account for . . .

literature at odds with their testimony." *McEwen v. Baltimore Washington Med. Ctr. Inc.*, 404 F. App'x 789, 791-92 (4th Cir. 2010).

Rule 702 also requires courts "to verify that expert testimony is 'based on sufficient facts or data.'" *E.E.O.C. v. Freeman*, 778 F.3d 463, 472 (4th Cir. 2015) (quoting Fed. R. Evid. 702(b)). Thus, "trial judges may evaluate the data offered to support an expert's bottom-line opinions to determine if that data provides adequate support to mark the expert's testimony as reliable." *Id.* The court may exclude an opinion if "there is simply too great an analytical gap between the data and the opinion offered." *Id.* "The proponent of the [expert] testimony must establish its admissibility by a preponderance of proof." *Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 199 (4th Cir. 2001).

The Court is mindful that the *Daubert* inquiry involves "two guiding, and sometimes competing, principles." *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 261 (4th Cir. 1999). "On the one hand, . . . Rule 702 was intended to liberalize the introduction of relevant expert evidence," *id.*, and "the trial court's role as a gatekeeper is not intended to serve as a replacement for the adversary system." *United States v. Stanley*, 533 F. App'x 325, 327 (4th Cir. 2013) *cert. denied*, 134 S. Ct. 1002 (2014). On the other, "[b]ecause expert witnesses have the potential to be both powerful and quite misleading, it is crucial that the district court conduct a careful analysis into the reliability of the expert's proposed opinion." *United States v. Fultz*, 591 F. App'x 226, 227 (4th Cir.) *cert. denied*, 135 S. Ct. 2370 (2015); *accord Westberry*, 178 F.3d at 261.

II. Ms. Hempstead's Medical History and Diagnosis of Diabetes

Juanita Hempstead began seeing primary care physician Dr. Lou Sabih in January of 1998. (Dkt. No. 1275-1 at 9). In March of 1998, Dr. Sabih ordered a lipid panel, which showed

that Ms. Hempstead had total cholesterol of 243 mg/dL, LDL of 151 mg/dL, HDL of 41 mg/dL, and triglycerides of 255 mg/dL.[1] (*Id.*). In response to these labs, Dr. Sabih prescribed 20 mg of Lipitor daily. (*Id.*). Because of apparent concerns about possible liver toxicity, Ms. Hempstead did not start taking Lipitor at that time. (*Id.*).

Over a year later in June of 1999 (and after adjustments to her blood pressure medication), Ms. Hempstead had another lipid panel, which showed Ms. Hempstead had a total cholesterol of 250 mg/dL, LDL of 175 mg/dL, HDL of 46 mg/dL, and triglycerides of 142 mg/dL. (*Id.* at 10). Ms. Hempstead weighed 176 lbs at the time and had a body mass index (BMI) of 26.37.[2] (*Id.*). Her glucose measurement at the time was 97 mg/dL.[3] (*Id.*). Ms. Hempstead was again prescribed 20 mg of Lipitor daily. (*Id.*). Ms. Hempstead took Lipitor inconsistently over the next year. (*Id.*). Pharmacy records indicate that she did not refill the prescription at all between January and July of 2000. (*Id.*).

In July of 2000, Ms. Hempstead began taking Lipitor regularly. (*Id.* at 11). In the fall of 2000, Dr. Michael Ausmus became Ms. Hempstead's primary care physician. (*Id.* at 10-11). Two years later, in September of 2002, a lipid panel showed Ms. Hempstead had total cholesterol

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[1] Normal levels for these lipid measurements are less than 200 md/dL for total cholesterol; less than 130 mg/dL for LDL; between 35 and 150 md/dL for HDL; and less than 200 mg/dL for triglycerides. (Dkt. No. 1064-7 at 29).

[2] BMI is a measurement based on height and weight that is used as an indicator of body fat and is method of screening for weight category, such as underweight, normal or healthy weight, overweight and obesity. Centers for Disease Control and Prevention, http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html (last visited Dec. 10, 2015). A BMI of 25.0-29.9 is considered overweight and a BMI of 30.0 or above is considered obese. *Id.; accord* U.S. Department of Health &and Human Services, National Heart, Lung, and Blood Institute, http://www.nhlbi.nih.gov/health/educational/lose_wt/BMI/bmicalc.htm (last visited Dec. 10, 2015).

[3] The parties generally agree that fasting blood glucose of $\leq$ 100 mg/dL is normal, multiple fasting blood glucose levels between 100 mg/dL and 125 md/dL is diagnostic for pre-diabetes, and multiple fasting blood glucose levels > 125 mg/dL is diagnostic for diabetes. (*See* Dkt. No. 972 at 12, Dkt. No. 1047 at 10, 12).

of 179 md/dL, LDL of 111 mg/dL, HDL of 40 mg/dL, and triglycerides of 139 mg/dL. (*Id.* at 10). She had an abnormal glucose reading of 114 mg/dL. (*Id.*). She had gained a total of 13 pounds since starting Lipitor in 1999, resulting in a total weight of 184 and a BMI of 27.57. (*Id.*).

In the fall of 2003, Ms. Hempstead stopped taking Lipitor for three weeks due to abdominal pain. (*Id.* at 11). A lipid panel taken after being off Lipitor for approximately three weeks showed Ms. Hempstead's blood lipid levels had risen. She had total cholesterol of 258 mg/dL, LDL of 173 mg/dL, HDL of 46 mg/dL, and triglycerides of 194 mg/dL. (*Id.* at 10). Her glucose reading after three weeks off Lipitor was 122 mg/dL. (*Id.*). Three months later, after resuming Lipitor, her lipid levels came back down, with total cholesterol of 176 mg/dL, LDL of 103 mg/dL, HDL of 37 mg/dL, and triglycerides of 179 mg/dL. (*Id.* at 10, 11).

In February of 2004, Ms. Hempstead was seen in the emergency room and diagnosed with colitis. (*Id.* at 11). She had a random blood glucose reading of 214 mg/dL, but there was no diagnosis of diabetes. (*Id.*). On May 14, 2004, Ms. Hempstead was admitted to the hospital with a blood glucose level of 613 mg/dL and diagnosed with new-onset diabetes. (*Id.*). She was two weeks shy of her sixtieth birthday. (Dkt. No. 1094 at 8). Ms. Hempstead's weight was not properly recorded during her hospital stay. However, her weight a couple of weeks prior to the hospital stay was 191 pounds, with a BMI of 28.6, and her weight at her first follow-up appointment after the hospital stay was 180 pounds, with a BMI of 27. (Dkt. No. 1275-1 at 10, 11).

III. The Role of Relative Risk in General and Specific Causation

Before addressing Dr. Murphy's opinion, it is helpful to review the difference between general and specific causation and how each is proven. "[I]n order to carry the burden of

proving a plaintiff's injury was caused by exposure to a specified substance," a plaintiff must demonstrate general and specific causation. *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 263 (4th Cir.1999); *Zellers v. NexTech Ne., LLC*, 533 F. App'x 192, 196 n.6 (4th Cir. 2013) *cert. denied*, 134 S. Ct. 911 (2014). "General causation is whether a substance is capable of causing a particular injury or condition in the general population and specific causation is whether a substance caused a particular individual's injury." *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005). "Plaintiff must first demonstrate general causation because without general causation, there can be no specific causation." *Id.*; *accord Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 351-52 (5th Cir. 2007).

The parties agree that epidemiologists use a two-step process for establishing general causation. (Dkt. No. 972 at 27-28; Dkt. No. 1053 at 13); *see also Ambrosini v. Labarraque*, 101 F.3d 129, 136 (D.C. Cir. 1996). First, studies must establish an association or correlation between two variables, here, Lipitor and diabetes. If two variables correlate, the incidence of one variable (diabetes) changes with the incidence of another (Lipitor). In other words, one variable (Lipitor) increases the risk of the other (diabetes). Once an association is established, epidemiologists apply the "Hill factors" to evaluate whether an association is causal. Reference Manual on Scientific Evidence 600 (3d ed. 2011); *In re Zoloft (Sertraline Hydrochloride) Products Liab. Litig.*, 26 F. Supp. 3d 449, 454-55 (E.D. Pa. 2014), *recon. denied*, 2015 WL 314149 (E.D. Pa. Jan. 23, 2015).

However, even if Plaintiffs establish that there is an association between Lipitor and diabetes (i.e., that Lipitor increases the risk of diabetes) and that Lipitor is capable of causing diabetes, it does not necessarily follow the Lipitor caused the development of diabetes in a particular plaintiff. For specific causation, the plaintiff must "demonstrate[] that the substance

actually caused injury in her particular case." *Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245, 1249 n.1 (11th Cir. 2010). Under a preponderance-of-evidence burden, a plaintiff must show, more likely than not, the substance caused, or was a substantial contributing factor to, her particular injury.

Some courts have held that one way that a plaintiff can meet her burden of production on specific causation is to have an expert witness testify to specific causation based on epidemiologic studies that find a relative risk of injury of 2.0 or higher and what is referred to as "the logic of the effect of doubling of the risk." Reference Manual on Scientific Evidence 612 (3d ed. 2011); *see also Marder v. G.D. Searle & Co.*, 630 F. Supp. 1087, 1092 (D. Md. 1986) ("In epidemiological terms, a two-fold increased risk is an important showing for plaintiffs to make because it is the equivalent of the required legal burden of proof–a showing of causation by the preponderance of the evidence or, in other words, a probability of greater than 50%.").

The relative risk ratio is the risk of disease or injury among people exposed to an allegedly harmful substance divided by the risk of the disease among those not exposed to the substance. Reference Manual on Scientific Evidence at 627. For instance, if the risk of developing diabetes while on Lipitor is 6% and the risk of developing diabetes not on Lipitor (i.e., in a placebo group) is 4%, then the relative risk of developing diabetes for Lipitor is 6/4 or 1.5.[4] A relative risk of 1.0 indicates no difference between the two groups in risk. The risk in the two groups is the same (e.g., 5% divided by 5% or 20% divided by 20%).

Assuming no confounding factors, bias, etc., a statistically significant relative risk ratio between 1.0 and 2.0 can be used, in conjunction with the "Hill factors," to establish general

[4] Absolute risk is the risk in a particular population without relation to another group. Here, the absolute risk in the Lipitor group is 6%, the absolute risk in the placebo group is 4%, and the difference in the absolute risk is 2%.

causation but cannot be used, by itself, to establish specific causation. A relative risk ratio in this range suggests that while some people exposed to the substance developed the disease due to exposure (i.e., there is the potential for the substance to cause the disease), most would have developed the disease anyway. Take the example above with a relative risk ratio of 1.5,[5] where 4% of the placebo group and 6% of the Lipitor group develop diabetes. In a group of 100 people all taking Lipitor, six of them will develop diabetes. Four of these people (4% of 100) would have developed diabetes regardless, as in the placebo group. The other two diabetics (2% of 100) would not have developed diabetes but for Lipitor. In other words, two-thirds of the people who take Lipitor and develop diabetes would have done so regardless, and one-third of people who take Lipitor and develop diabetes would not have developed diabetes but for the Lipitor. For this minority (the one-third), Lipitor is a substantial contributing factor to their diabetes. Thus, to establish specific causation with a relative risk between 1.0 and 2.0, an expert—utilizing a valid methodology, applying it reasonably and relying on sufficient data to support her opinions—must opine to a reasonable degree of medical certainty that the particular plaintiff is in the minority of those that developed the disease due to exposure to a particular drug or substance rather than in the majority that would have developed the disease regardless.

A relative risk ratio greater than 2.0, however, suggests that of the people exposed to a substance who developed the disease, most of them developed the disease *due* to the exposure. For example, if the relative risk of diabetes due to Lipitor were 3.0,[6] then two-thirds of those on Lipitor who develop diabetes would not have developed diabetes but for the drug. An example

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[5] Other ways to state this relative risk of 1.5 are (a) someone on Lipitor is 1.5 times as likely to develop diabetes as someone not on Lipitor, (b) Lipitor increases the risk of diabetes by 50%, or (c) Lipitor increases the risk of diabetes by half.

[6] Other ways of stating this relative risk of 3.0 are (a) there is a threefold increase in risk or (b) a 300% increase in risk.

of this would be if, in a randomized study, 6% of the Lipitor group developed diabetes and 2% of the placebo group developed diabetes. In this example, six out of a hundred people on Lipitor would develop diabetes. Of these six, two would have done so regardless and four would develop diabetes only because they took Lipitor.

Thus, for a relative risk ratio above 2.0, there is a "probability of greater than 50%" that the substance at issue caused the disease or injury. *Marder*, 630 F. Supp. at 1092. In other words, it is more likely than not that the substance caused the injury or disease. This logic does have its limitations. It relies on studies "identifying a genuine causal relationship and a reasonably reliable measure of the increased risk." Reference Manual on Scientific Evidence at 612. If confounding factors, bias or random error is the source of the association, rather than a true causal relationship, the logic falls apart. *Id.* at 612-13. Thus, a finding of statistical significance is very important, and observational studies with the potential for confounding and bias may not be sufficient for this theory of specific causation. Randomized controlled trials may be required to show an actual doubling of the risk exists (i.e., to show a valid relative risk ratio). Even with randomized controlled trials, this logic is based on the assumption that the plaintiff at issue is similar to the study subjects and has the same amount of exposure to the substance as the study subjects. *Id.* at 613.

Turning to the actual case at hand, Dr. Murphy determined that "the most reliable data suggests the [relative] risk ratio is somewhere around 1.6."[7] (Dkt. No. 1006-3 at 49). Thus,

[7] The 2011 Waters study of the SPARCL data found a slightly lower relative risk ratio of 1.37. (Dkt. No. 972-29 at 2). In that study, diabetes developed in 8.71% of the Lipitor group versus 6.06% of the placebo group. (*Id.*). The study authors found a hazard ratio of 1.37 with a 95% confidence interval of 1.08 to 1.75. (*Id.*). This data suggests that out of 100 people on Lipitor, six would develop diabetes regardless and an additional 2-3 people would develop diabetes due to Lipitor. The key to proving specific causation is being able to show that a particular plaintiff

using her estimate of relative risk, most of the people who develop diabetes while on Lipitor would have done so anyway, and she cannot use the logic of "doubling the risk" to provide a specific causation opinion. Using her estimate, 63% of the people who take Lipitor and develop diabetes would have done so *in the absence of Lipitor*, whereas 37% of the people who take Lipitor and develop diabetes did so only because they took Lipitor.[8] The question then becomes how does Dr. Murphy conclude that Ms. Hempstead is in the 37% that develop diabetes due to Lipitor, rather than the 63% that would have done so regardless.

IV. Dr. Murphy's Opinion

Dr. Murphy is a Professor of Clinical Medicine at the University of California, San Francisco and Chief of the Division of Endocrinology and Metabolism at San Francisco General Hospital. (Dkt. No. 1006-1 at 3). She received her M.D. from Harvard Medical School and a Ph.D. in Biochemistry from Oxford University. (*Id.*). As a practicing endocrinologist, she has a

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was one of the additional 2-3 people who developed diabetes due to Lipitor, rather than one of the six that would have developed it regardless.

[8] At oral argument, counsel made the point that this relative risk ratio of 1.6 adjusts for certain risk factors like BMI. (Dkt. No. 1277 at 40, 44, 54-55). This statement is an argument that the relative risk ratio is a valid one and not confounded by other factors. It does not solve the specific causation problem presented by a relative risk ratio between 1.0 and 2.0. Assuming that this relative risk ratio adjusted for ***all*** of Ms. Hempstead's identified risk factors, a relative risk of 1.6 suggests that in a pool of women who (1) have Ms. Hempstead's risk factors, (2) take Lipitor and (3) develop diabetes, most of them—63%—would have developed diabetes *regardless*. It shows that Lipitor increases the risk of diabetes in that population and suggests that 37% of the women who developed diabetes in that population would have not done so but for Lipitor. However, it says nothing about *which* women would be in that 37%.

Dr. Murphy and counsel would like the Court to conclude that because Lipitor increases the risk of diabetes, it must be the cause of Ms. Hempstead's diabetes. This argument confuses general and specific causation because it conflates overall risk of an event with the question of whether an event happened in a particular instance. *See Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245, 1255 (11th Cir. 2010) ("The fact that exposure to [a substance] may be a risk factor for [a disease] does not make it an actual cause simply because [the disease] developed."). It argues that if general causation is established, then specific causation is necessarily established in all cases where the relative risk ratio of 1.6 is valid. As explained above this is simply not true.

wealth of experience treating patients with diabetes. (*Id.*). Pfizer does not dispute Dr. Murphy's impressive credentials, only her methodology in this instance. (*See* generally, Dkt. No. 1006).

A. Dr. Murphy's Reliance on *Post Hoc Ergo Propter Hoc*

Dr. Murphy opines that "Lipitor was a substantial contributing factor in Ms. Hempstead developing diabetes in May 2004. In other words, but for her use of Lipitor, Ms. Hempstead would not have been diagnosed with diabetes at that time." (Dkt. No. 1006-1 at 4). While Dr. Murphy purports to use a five-part test, her opinion that Lipitor substantially contributed to Ms. Hempstead's diabetes is based only on (1) the fact that Lipitor increases the risk of diabetes (general causation) and (2) that Ms. Hempstead developed diabetes after taking Lipitor.

Ms. Hempstead states that her assessment was based on a five-part test, which she incorrectly numbered as a four-part test:

1) Are there reports in the scientific literature and/or reliable expert data analyses showing the occurrence of new onset diabetes with Lipitor?

2) Is it biologically plausible that Lipitor can cause new onset diabetes?

2) Did new onset diabetes appear after the Lipitor was given?

3) Were there other possible causes for the development of new onset diabetes?

4) How likely is it that Lipitor caused new onset diabetes in this individual at this time?

(*Id.* at 5). Dr. Murphy claims that this methodology is "commonly used" in her report. (*Id.* at 4). However, when pressed in her deposition she could not identify any organizations or peer-reviewed texts that contain this methodology. (Dkt. No. 1006-3 at 167). She testified that she had only seen this type of methodology used in expert reports in this case, including Defendant's experts, and from that "made the assumption" that these methods were "commonly used in litigation." (*Id.* at 168). She did not know of any colleagues in the medical profession that used

her methodology to determine the cause of Type 2 diabetes. (*Id.* at 171). She has never used this methodology to diagnose the cause of her own patients' diabetes and, in fact, has never diagnosed a patient with statin-induced diabetes. (*Id.* at 34-35). She cannot say whether she has even ever treated a patient with statin-induced diabetes. (*Id.*).

With regard to the first question in Dr. Murphy's methodology, she discusses various studies and concludes that evidence exists that Lipitor increases the risk of diabetes. (Dkt. No. 1006-1 at 7-8). She determined that "the most reliable data suggests the [relative] risk ratio is somewhere around 1.6." (Dkt. No. 1006-3 at 49). She opined that the relative risk was "perhaps in the range of 1.5 to 1.7" and with the addition of the 2015 Cedarburg article showing a relative risk of 1.46, the relative risk is "somewhere in that general realm." (*Id.*). With regard to the second question in her methodology, Dr. Murphy determined that "[t]here is evidence for several mechanisms by which Lipitor could lead to the development of new onset diabetes." (Dkt. No. 1006-1 at 9; *see also* Dkt. No. 1275-2 at 178-80). Turing to Step 3, Dr. Murphy determines that Ms. Hempstead began taking Lipitor before she was diagnosed with diabetes by reviewing her medical files. (*See* Dkt. No. 1275-1 at 9-12; Dkt. No. 1275-2 at 179).

While Dr. Murphy goes on to discuss Ms. Hempstead's other risk factors for diabetes in her report, her causation analysis essentially stops at Step 3. As she testifies in her deposition, "[i]f the patient was taking the Lipitor and they developed diabetes while on it, . . . I would think that it would be a contributing factor." (Dkt. No. 1006-3 at 125). The only exceptions to this general rule that Dr. Murphy could name were examples where this temporal relationship[9] was

[9] Although there is a temporal relationship between Lipitor and diabetes in that one preceded the other, the relationship is not a "proximate" one. Hempstead did not develop diabetes until almost five years after starting Lipitor. (Dkt. No. 1275-1 at 10).

absent: patients with pre-existing diabetes and patients who did not take the drug regularly.[10] (*Id.* at 125-26). Dr. Murphy verified her exclusive reliance on a temporal relationship when she was asked what evidence she had that Lipitor contributed to Ms. Hempstead's development of diabetes. She responded, "The temporal relationship and the increased glucose and the fact that she was taking it."[11] (Dkt. No. 1275-2 at 189). The only other basis for her opinion that she could provide is the population-level evidence of an increased risk of diabetes (Step 1 of her methodology). (*See, e.g.*, Dkt. No. 1275-2 at 233 ("When you . . . control for risk factors in the studies as we've talked about, Lipitor has an increased risk, and that is why my conclusion is that it was a contributing cause.").

Plaintiff claims that "Dr. Murphy's insistence on the need to review actual records precludes the possibility that she formed her opinion about Juanita Hempstead based solely on the temporal relationship between her use of Lipitor and the onset of her diabetes." (Dkt. No. 1094 at 19-20). However, Dr. Murphy was unable to point to a single piece of evidence that she found in Ms. Hempstead's medical files, or a single piece of evidence that she might find in another patient's files (other than pre-existing diabetes and failure to regularly take the drug), that would affect her assessment of whether Lipitor caused the patient's diabetes. (Dkt. No. 1006-3 at 125-26, Dkt. No. 1275-2 at 189). At oral argument, the Court repeatedly asked counsel to identify what evidence served as the basis of Dr. Murphy's opinion that Lipitor

[10] Although this testimony suggests that, in Dr. Murphy's opinion, a patient must take Lipitor regularly for it to cause diabetes, Dr. Murphy later testified that even a single 10-milligram Lipitor pill "could be a contributing cause to diabetes." (Dkt. No. 1006-3 at 266).

[11] Plaintiff attempts to make much out of Dr. Murphy's statement that "I can't speak to other cases that I haven't reviewed to know if there was something or things that I haven't thought of that would[] change my mind." (Dkt. No. 1006-3 at 125, Dkt. No. 1094 at 19). However, the caveat that there could possibly be something she has not thought of that might change her mind does not change Dr. Murphy's current opinion that if a patient were taking Lipitor at the time of her diagnosis of Type 2 diabetes, then "I would think that it would be a contributing factor, yes." (Dkt. No. 1006-3 at 125).

contributed to Ms. Hempstead's diabetes. Counsel could only identify studies indicating risk (general causation) and a temporal relationship (Ms. Hempstead's glucose increased after taking Lipitor). (Dkt. No. 1277 at 33, 35-36, 38, 44, 52-53, 54-55).

In sum, Dr. Murphy's conclusion that Lipitor was a contributing factor in Ms. Hempstead's development of diabetes rests on (1) the fact that population studies show Lipitor increases the risk of diabetes (Step 1, or general causation) and (2) that Ms. Hempstead took Lipitor before developing diabetes (Step 3, or a temporal relationship). As she explains,

> I would try and establish that there's background evidence that there is an increased risk of diabetes with Lipitor in larger studies. And then in that individual, did the diabetes appear after the Lipitor was given?
>
> And—and I then look at the risk and whether or not the Lipitor was given and whether or not the patient took the Lipitor, and then with the background risk, I'm able to attribute that as a causative factor in their development of diabetes.

(Dkt. No. 1006-3 at 124). In other words, according to Dr. Murphy, if general causation exists (i.e., Lipitor has the *potential* to cause diabetes), then specific causation exists for every patient that took Lipitor and thereafter developed diabetes. Again, this logic is flawed. "The fact that exposure to [a substance] may be a risk factor for [a disease] does not make it an actual cause simply because [the disease] developed." *Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245, 1255 (11th Cir. 2010).

Dr. Murphy concedes that clinical studies show that some patients taking a placebo also develop diabetes, "presumably developing their diabetes from other reasons," meaning that some of those who took Lipitor and developed diabetes would have done so in the absence of Lipitor. (Dkt. No. 1006-3 at 220; Dkt. No. 1006-4 at 356-57, Dkt. No. 1006-5 at 86-87). As explained above, taking Dr. Murphy's 1.6 estimate of relative risk, ***most*** (63%) of those who took Lipitor and developed diabetes would have developed diabetes anyway.

Dr. Murphy also admits that, of the patients on Lipitor who developed diabetes in a clinical trial, she had no way to determine which of these patients developed diabetes due to Lipitor versus due to other causes. (Dkt. No. 1006-3 at 153-54). Just as with "identify[ing] which patient Lipitor has prevented a [heart attack] in. You can't—you can't do that." (*Id.* at 153). There are no biological markers or "any kind of fingerprint" that would allow a clinician to "distinguish the effects of [Lipitor] from any of the other well-recognized risk factors for Type 2 diabetes." (*Id.* at 152-53, 157; *see also* Dkt. No. 1275-2 at 191-92). Thus, unable to identify particular patients whose diabetes diagnoses were caused by Lipitor, Dr. Murphy is left with the overly broad conclusion that "[i]f the patient was taking the Lipitor and they developed diabetes while on it, . . . I would think that it would be a contributing factor." (Dkt. No. 1006-3 at 125).

B. Dr. Murphy's Discussion of Risk Factors

Dr. Murphy does identify other risk factors for diabetes in her report in accordance with Step 4 of her methodology. However, she never answers or even asks the fifth question, which is the crux of specific causation: How likely is it that Lipitor caused new-onset diabetes in this individual at this time?

Dr. Murphy identifies the following risk factors for diabetes: family history and race/ethnicity, weight, age, hypertension, gestational diabetes, inactivity, diuretic use, ciprofloxacin use, stress, smoking, hypothyroidism, and Lipitor. (Dkt. No. 1275-1 at 12-16). In Ms. Hempstead's case, Dr. Murphy ruled out gestational diabetes, hypothyroidism, and inactivity as potential risk factors. (*See id.*). She determined that Ms. Hempstead's occasional smoking in the past was "not significant, or recent enough to constitute a significant risk factor for the development of diabetes," that there was "insufficient scientific evidence at this point to conclude stress played a role in the development of T2DM in Ms. Hempstead," and that "the

literature does not support a significant effect of ciprofloxacin on increasing glucose levels in non-diabetic individuals."[12] (*Id.* at 15-16). Because Ms. Hempstead was on the lowest dose of HCTZ (a diuretic) and because her potassium did not lower significantly on HCTZ, Dr. Murphy concluded that Ms. Hempstead's risk of diabetes attributable to HCTZ was "minimal" and "would be very small in comparison to the other risks to be discussed in her case." (*Id.* at 15).

1. BMI

However, there are other risk factors that Dr. Murphy "can't rule out." (Dkt. No. 1275-2 at 209). Dr. Murphy testifies that "BMI is the strongest predictor of diabetes risk." (Dkt. No. 1006-3 at 244). Before taking Lipitor, Ms. Hempstead was overweight with a BMI of 26.37, and close in time to her diagnosis, Ms. Hempstead was overweight with a BMI of 28.6. (Dkt. No. 1275-1 at 10, 13). Dr. Murphy observed in her report that because Ms. Hempstead was overweight but not obese, her risk of developing diabetes was less than if she had been obese. (*Id.* at 13). While this is certainly true, Dr. Murphy did not state in her report the extent of Ms. Hempstead's increased risk of diabetes associated with her BMI of 26.37 compared to a patient with a normal BMI.[13] However, under questioning in her deposition, Dr. Murphy agreed that in an article published in the American Journal of Epidemiology, the Nurses' Health Study found that women with a body mass index of 25 to 26.9 had more than a fivefold increased risk of

[12] In the language of differential etiology, Dr. Murphy concluded that the literature did not support "ruling in" Ciprofloxacin or stress as potential causes of Type 2 diabetes.

[13] She did state than an African American woman who is overweight will have an absolute increase in lifetime risk of diabetes of 8.8%. (Dkt. No. 1275-1 at 13). However, she does not give the absolute risk for a normal weight African American woman or the relative risk ratio and does not discuss how this risk of 8% is increased over the risk of diabetes for women of normal weight.

diabetes compared to women with a body mass index below 22.[14] (Dkt. No. 1275-2 at 242; Dkt. No. 1006-17 at 5). Dr. Murphy further acknowledged in her deposition that Ms. Hempstead's BMI was a substantial contributing factor in her development of diabetes. (Dkt. No. 1275-2 at 185, 186).

2. Adult Weight Gain

Dr. Murphy also acknowledged that weight gain, independent of BMI, increases a person's risk of diabetes. (Dkt. No. 1006-3 at 245). Between September of 1994 and April of 2004, Ms. Hempstead gained 24 pounds. (Dkt. No. 1275-2 at 246-47). Dr. Murphy agrees that the Nurses' Health Study found a weight gain in this range, adjusted for BMI, was associated a twofold increase in the risk of diabetes. (*Id.* at 247). With Ms. Hempstead's BMI, this amount of weight gain was associated with a more than fourfold increased risk of diabetes. (Dkt. No. 1006-17 at 8). Dr. Murphy acknowledged in her deposition that Ms. Hempstead's 24-pound weight gain in the ten years preceding her diagnosis of diabetes was a substantial contributing factor in Ms. Hempstead's development of diabetes but failed to mention this weight gain as a factor in her report. (Dkt. No. 1275-2 at 247).

The record also demonstrates that Ms. Hempstead gained a total of approximately 60 pounds from her thirties until diagnosis of diabetes at age 59. (*See* Dkt. No. 1004-37 at 187). This total adult weigh gain, according to the Nurses' Health Study, is associated with a twelvefold increase in the risk for developing diabetes, after adjusting for age and BMI. (Dkt. No. 1275-2 at 250; Dkt. No. 1006-17 at 7). However, Dr. Murphy admitted in her deposition she

[14] In her report, Dr. Murphy states that "the most validated data on risk assessment comes from studies such as the Women's Health Initiative and the Nurses' Health Study that enrolled primarily Caucasian women." (Dkt. No. 1006-1 at 12).

did not consider the increased risk associated with total adult weight gain in her analysis and did not mention it in her report. (Dkt. No. 1275-2 at 249; *see also* Dkt. No. 1275-1).

3. Family History

According to Dr. Murphy, "[f]amily history is a significant contributor in general to diabetes." (Dkt. No. 1275-2 at 183; *see also* Dkt. No. 1006-3 at 160 ("A positive family history of Type 2 diabetes increases your risk of Type 2 diabetes.")). Because Ms. Hempstead's father had diabetes, studies that attempt to stratify family risk "would stratify Ms. Hempstead as moderate risk for diabetes" with a relative risk of 2.3. (Dkt. No. 1275-1 at 13). However, "given her father's advanced age at the time of diagnosis, and the fact that she has four siblings over the age of 70 without diabetes," Dr. Murphy concludes that her risk from family history "would actually be lower, somewhere between average and moderate risk." (*Id.*). Dr. Murphy agrees that Ms. Hempstead's family history was significant in the development of her diabetes. (Dkt. No. 1275-2 at 186).

4. Age

"There is a steady increase in risk [of diabetes] with increasing age." (Dkt. No. 1006 at 13). According to Dr. Murphy, "Ms. Hempstead's age of 55 at the time she . . . started on Lipitor would infer an increased risk for the subsequent development of diabetes." (Dkt. No. 1275-1 at 13). Dr. Murphy agrees that age was significant in the development of Ms. Hempstead's diabetes. (Dkt. No. 1275-2 at 186).

5. Hypertension

Dr. Murphy also states that "[h]ypertension is an established risk factor for the development of [Type 2 diabetes]." (Dkt. No. 1275-1 at 14). She notes that a study from the Women's Health Initiative found that the adjusted hazard ratio for developing diabetes in an

overweight woman with diagnosed hypertension was 1.76. (*Id.*). She agrees that hypertension was significant in the development of Ms. Hempstead's diabetes. (Dkt. No. 1275-2 at 186).

6. Metabolic Syndrome

Finally, Dr. Murphy did not consider metabolic syndrome because she does not find consideration of it helpful in clinical practice. (Dkt. No. 1275-2 at 252). However, she conceded that it "does confer an increased risk" of diabetes and that in 1998 Ms. Hempstead "would . . . perhaps, meet the definition for metabolic syndrome." (*Id.* at 252, 254).

7. Comparison of Risk Factors

In the final step of Dr. Murphy's methodology, she is to consider, given these other risk factors, "[h]ow likely is it that Lipitor caused new onset diabetes in this individual at this time?" (Dkt. No. 1006-1 at 5). However, Dr. Murphy ***never*** does any analysis that speaks to this question. To the extent that she answers it at all, she states in a conclusory, *ipse dixit* fashion that Lipitor was a substantial contributing factor to the development of Ms. Hempstead's diabetes. (Dkt. No. 1006-1 at 17; *see also* Dkt. No. 1277 at 51-52). She never determines the likelihood that Lipitor caused Ms. Hempstead's diabetes and does not compare the magnitude of the risk from Lipitor to the magnitude of the risks from other factors. She testifies that she does not know how she could rank or quantify the relative contributions of Ms. Hempstead's risk factors. (Dkt. No. 1275-2 at 186-87, 209-10, 230-31, 248). However, she has quantified the relative risk due to Lipitor as 1.6, and has admitted that studies have shown the relative risk of other factors were much higher. For example, Ms. Hempstead's BMI gave her a relative risk over 5.0, her total adult weight gain gave her a relative risk of 12.0, and her hypertension gave her a relative risk of 1.76. (Dkt. No. 1275-2 at 242, 247, 250; Dkt. No. 1275-1 at 14). Similarly, Dr. Murphy testifies that with regard to the fluctuations in Ms. Hempstead's glucose levels before her

diagnosis of diabetes, "I'm not sure that . . . . I can explain . . . what was going [on]. There's [sic] many potential causative factors." (Dkt. No. 1275-2 at 201).

Dr. Murphy admits that she has seen patients with risk factors such as age, family history, hypertension, and BMI, without any evidence of statin use, develop diabetes, and concedes that Ms. Hempstead may have developed diabetes in the future without Lipitor.[15] (*Id.* at 234, 164). But because Lipitor also increases the risk of developing diabetes, she concludes that it also must be a "contributing cause" in Ms. Hempstead's case. (*Id.* at 233). In other words, in Dr. Murphy's opinion, anything that increases the risk of diabetes must be a substantial contributing factor in development of diabetes for every patient with that risk factor. However, as explained above, this conclusion conflates general and specific causation and is contrary to Dr. Murphy's own assessment of the relative risk of Lipitor of 1.6, which recognizes that nearly two-thirds of the patients (63%) who develop diabetes while taking Lipitor would have developed diabetes regardless.

V. Discussion

"[S]imply because a person takes drugs and then suffers an injury does not show causation. Drawing such a conclusion from temporal relationships leads to the blunder of the *post hoc ergo propter hoc* fallacy." *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1243 (11th Cir. 2005); *see also Roche v. Lincoln Prop. Co.*, 278 F. Supp. 2d 744, 752 (E.D. Va. 2003) ("Dr. Bernstein's reliance on temporal causation as the determinative factor in his analysis is suspect

[15] Dr. Murphy suggests that "it would be ten years or more before [Ms. Hempstead] would have [developed diabetes]" absent Lipitor. (Dkt. No. 1275-2 at 165). However, this was based on average lifetime risk of developing diabetes "as an African American woman with a given BMI." (*Id.*). She admits that "there isn't data available for each of her risk factors" and that this estimate does not account for the other risk factors at play. (*Id.*). She also does not consider the average lifetime risk of diabetes due to Lipitor and assumes, without explanation, that the development of diabetes before this 10-year average must be due to Lipitor rather than other risk factors. (*Id.*).

because it is well settled that a causation opinion based solely on a temporal relationship is not derived from the scientific method and is therefore insufficient to satisfy the requirements of Rule 702.") (internal quotes omitted).

Plaintiffs note that in *Westberry*, the Fourth Circuit held that "depending on the circumstances, a temporal relationship between exposure to a substance and the onset of a disease or a worsening of symptoms can provide compelling evidence of causation." *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265 (4th Cir. 1999). However, *Westberry* also states that "[o]f course, the mere fact that two events correspond in time does not mean that the two necessarily are related in any causative fashion" *Id.* There are circumstances where temporal proximity is particularly compelling. *See, e.g., Cavallo v. Star Enter.*, 892 F. Supp. 756, 774 (E.D.Va. 1995) (explaining that "there may be instances where the temporal connection between exposure to a given chemical and subsequent injury is so compelling as to dispense with the need for reliance on standard methods of toxicology," for example, if one were exposed to a substantial amount of "chemical X and immediately thereafter developed symptom Y").

However, this is not always the case. *See Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278 (5th Cir. 1998) ("In the absence of an established scientific connection between exposure and illness, or compelling circumstances such as those discussed in *Cavallo*, the temporal connection between exposure to chemicals and an onset of symptoms, standing alone, is entitled to little weight in determining causation."). In *Westberry*, it was "undisputed that inhalation of high levels of talc irritate[d] mucous membranes" and that the plaintiff's sinus disease began shortly after he started as a gasket cutter where he "worked in clouds of talc . . . that covered him and his clothes." 178 F.3d at 265. Every time the plaintiff stayed out of work, his sinuses improved. *Id.* Every time he returned, they worsened. The Fourth Circuit found these particular

circumstances compelling, but they are not remotely analogous to facts here. In this matter, there was no immediate response to the Lipitor. Dr. Murphy admits that two years after Ms. Hempstead began taking Lipitor, it "did not appear to have . . . an effect on increasing her glucose level." (Dkt. No. 1275-2 at 205). There was no "clinically significant difference" in her glucose levels at that time. (*Id.*). Ms. Hempstead did not develop diabetes until almost five years after starting Lipitor. (Dkt. No. 1275-1 at 10). This is a far cry from the temporal proximity and factual situations in *Westberry* and *Cavallo*.

Two Seroquel cases are instructive. In *Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245 (11th Cir. 2010), the plaintiff claimed that Seroquel caused her to develop diabetes. The district court excluded the case-specific expert testimony and granted summary judgment and the Eleventh Circuit upheld both decisions. In *Guinn*, the expert admitted that "other risk factors alone were sufficient to explain the onset of [plaintiff's] diabetes" and that "she knew of no methodology for ruling out alternative causes and thus had not attempted to do so." *Id.* at 1249-50. Recognizing the problem with this opinion, the expert submitted a supplemental declaration stating that "she believed other potential causes were not solely responsible because [plaintiff] had gained weight and developed diabetes soon after taking Seroquel when [plaintiff's] other risk factors remained constant." *Id.* at 1254. However, even assuming the plaintiff's risk factors for diabetes remained constant,[16] the Eleventh Circuit rejected this argument as a reliable methodology: "Dr. Marks' reliance on the temporal proximity of the introduction of an additional risk factor, however, does not satisfy the requirement that a differential diagnosis consider possible alternative causes on the facts of this case." *Id.* at 1254.

[16] As the Eleventh Circuit noted in *Guinn*, "one important risk factor—[plaintiff's] age—did not remain constant." *Id.* at 1254. Here, Ms. Hempstead's weight also did not remain constant.

First, "[t]emporal proximity is generally not a reliable indicator of a causal relationship. . . . [and] several factors make it especially unreliable in this case." *Id.* Second, and more importantly, the expert "does not explain why having a stable risk profile makes it unlikely that preexisting factors caused [plaintiff's] diabetes when such factors put her at an extremely high risk for diabetes." *Id.* Plaintiffs "numerous other risk factors for diabetes make it hard to draw any inferences from the temporal proximity." *Id.* The *Guinn* decision is in accord with *Westberry*'s statement that temporal proximity can be compelling in particular circumstances; those circumstances are just not present here:

> After considering the nature of the temporal relationship at issue in this case, we conclude Dr. Marks did not adequately consider possible alternative causes simply by noting the temporal proximity between Guinn's ingestion of Seroquel and subsequent development of diabetes. *We do not hold that a temporal relationship can never be used to consider alternative causes of a plaintiff's injury; instead, we merely find that temporal proximity is not sufficient on the facts of this case.*

Id. at 1255 (emphasis added).

Next, the expert in *Guinn* provided a second explanation of how she considered alternative causes–by testifying that all the risk factors work together. This is Dr. Murphy's explanation. (*See* Dkt. No. 1275-2 at 230-34). The Eleventh Circuit also rejected this reasoning as a valid methodology, holding that "[a]n expert, however, cannot merely conclude that all risk factors for a disease are substantial contributing factors in its development." *Id.* at 1255.

> The fact that exposure to [a substance] may be a risk factor for [a disease] does not make it an actual cause simply because [the disease] developed." *Cano v. Everest Minerals Corp.*, 362 F.Supp.2d 814, 846 (W.D.Tex.2005). "[A]lthough the differential diagnosis technique is well accepted ... [, a finding] that all possible causes are causes does not appear to have gained general acceptance in the medical and scientific communities." *Id.* While multiple factors can work together to cause diabetes, Dr. Marks was still required to provide some analysis of why she concluded that, more likely than not, Seroquel substantially contributed to Guinn's weight gain and such weight gain was among the factors that substantially contributed to her diabetes.

Id.

As in *Guinn*, Dr. Murphy cannot simply opine that all present risk factors are "substantial contributing factors." Risk factors are *potential* causes of diabetes. Identifying potential causes is the work of general causation and, without more, does not suffice for a specific causation opinion. *See Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005) ("General causation is whether a substance is capable of causing a particular injury or condition in the general population and specific causation is whether a substance caused a particular individual's injury."); *see also Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 424 (5th Cir. 1987) (If a patient's "symptoms could have numerous causes," the expert cannot "simply pick[] the cause that is most advantageous to [the plaintiff's] claim"). "While multiple factors can work together to cause diabetes, [Dr. Murphy is] still required is still required to provide some analysis of why she concluded that, more likely than not, [Lipitor] substantially contributed to [Ms. Hempstead's] . . . diabetes." *Guinn*, 602 F.3d at 1255.

Haller v. AstraZeneca Pharm. LP, 598 F. Supp. 2d 1271 (M.D. Fla. 2009) is another Seroquel case where the plaintiff claimed the drug caused her weight gain and diabetes. The court excluded both case-specific causation experts. *Id.* at 1276. The endocrinologist expert in *Haller* "agreed that the complications from diabetes that he described in his report could have been solely caused by pre-existing obesity, years of smoking, metabolic syndrome, and uncontrolled hypertension that preceded [plaintiff's] ingestion of Seroquel" and "made crystal clear at his deposition that the sole basis for his opinion that Seroquel caused the development of diabetes in [plaintiff] was the temporal relationship between [plaintiff's] taking Seroquel and his subsequent weight gain, and the weight gain's temporal relationship with the development of diabetes." *Id.* at 1278, 1278-79. The expert "made equally clear that [plaintiff] would have

developed diabetes at some point even if he had never taken Seroquel. His opinion was that Seroquel somehow accelerated the development of diabetes," but he did not attempt to quantify the acceleration. *Id.* at 1279. Even though the plaintiff's risk factors in *Haller* are more extreme than Ms. Hempstead's, the court's conclusions are still instructive:

> Standing alone, the first factor–temporal connection–is legally insufficient. *McClain*, 401 F.3d at 1243. The second listed ground–that other risk factors had not yet caused diabetes–is also unpersuasive. This observation is largely temporal proximity in disguise. It posits that arguably the last additive factor–Seroquel–is necessarily the one that caused or substantially contributed to causing the disease. This overlooks Dr. Tulloch's own testimony regarding the slow progression of diabetes and the additive nature of the factors that can cause it. Moreover, it is equally plausible that the additive effects of, or an incremental increase in, one or more of the other risk factors was the actual tipping point. The third and fourth factors–biologically plausible mechanism and literature demonstrating a connection between Seroquel and diabetes–relate to *general* causation and carry little if any relevance to the question of whether Seroquel caused diabetes in Haller's *specific* case.

Id. at 1297-98 (emphasis in original). As in *Haller*, Dr. Murphy relies solely on temporal proximity and general causation evidence, without any other explanation for her conclusion that Lipitor caused diabetes *in this instance*.

Plaintiff argues that because Dr. Murphy utilized the differential diagnosis methodology and that methodology has been recognized as scientifically valid, the Court must accept Dr. Murphy's opinions as sufficient to meet *Daubert* standards. Under the traditional differential diagnosis approach, a physician, after clinically evaluating the patient, identifies "the cause of a medical problem by eliminating the likely causes until the most probable one is isolated." *Westbury*, 178 F.3d at 262. Courts have recognized that the differential diagnosis approach, sometimes referred to also as "differential etiology,"[17] may be permissible even where the

[17] "Technically, differential diagnosis refers to a method of determining which of two diseases a patient suffers from, whereas differential etiology is a term used to describe the process by which

physician has not examined the patient or where all possible causes of the patient's medical problems have not been ruled out. *See Kannankeril v. Terminix Int'l, Inc.*, 128 F.3d 802, 807 (3d Cir. 1997), *as amended* (Dec. 12, 1997) ("Depending on the medical condition at issue and on the clinical information already available, a physician may reach a reliable differential diagnosis without himself performing a physical examination, particularly if there are other examination results available."); *Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 202 (4th Cir. 2001) ("A medical expert's opinion based upon differential diagnosis normally should not be excluded because the expert has failed to rule out every possible alternative cause of a plaintiff's illness."). But courts have made clear that regardless of what the methodology may be called, it must meet the standards of Rule 702—an expert must use a valid methodology, the methodology must be reliably applied, and her opinions must be supported by sufficient facts and data. *Cooper,* 259 F.3d at 201; *see also McClain* at 401 F.3d at 1253 ("[A]n expert does not establish the reliability of his techniques or the validity of his conclusions simply by claiming that he performed a differential diagnosis on a patient.").

Under Fourth Circuit law, an expert need not rule out every possible alternative cause of a disease in a differential diagnosis. *Westberry*, 178 F.3d at 265. However, she must offer an explanation as to why these other recognized causes, alone, are not responsible for the disease in a particular plaintiff. *Cooper*, 259 F.3d at 202; *see also Westberry*, 178 F.3d at 265.

In this case, Dr. Murphy recognized that Plaintiff had a number of statistically significant risk factors for diabetes beyond ingestion of Lipitor, including her BMI, recent weight gain, total adult weight gain, age, family history, hypertension and possibly metabolic syndrome. Many of these risk factors greatly exceed the risk of developing diabetes associated with Lipitor. Dr.

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the cause of an injury is determined." *Guinn*, 602 F.3d at 1253. However, the trend among federal courts is to "use the term differential diagnosis to refer to both concepts." *Id.*

Murphy also acknowledged that she had no method to identify the minority of Lipitor users whose development of diabetes while on the medication was caused by Lipitor, as opposed to other risk factors. Confronted with this factual scenario, Dr. Murphy simply concludes that it is her opinion to a reasonable degree of medical certainty that "but for" the ingestion of Lipitor Plaintiff would not have developed diabetes. She offers no data or facts to make the leap from a possibility to a probability that Lipitor was a substantial contributing factor, and Plaintiff argues that so long as her methodology is labeled "differential diagnosis" that is good enough.

The Court's many difficulties with Dr. Murphy's testimony arise from the fact that she does not employ a reliable methodology to determine that Lipitor was a substantial contributing factor in Plaintiff's development of diabetes and does not have sufficient basis in fact or data to support that opinion. To the contrary, Dr. Murphy concludes that so long as the patient took Lipitor and developed diabetes, then Lipitor was a substantial contributing factor. Since it is well established by Dr. Murphy's estimation of risk that a majority of patients who develop diabetes while on Lipitor would develop the condition regardless of taking Lipitor, her method is invalid on its face. The powerful evidence that Plaintiff's many other risk factors can independently cause diabetes and cannot be ruled out further undermine Dr. Murphy's testimony. The gap between the available scientific evidence and Dr. Murphy's opinions are too great to survive a Rule 702 review. *See Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 424 (5th Cir. 1987) (If a patient's "symptoms could have numerous causes," the expert cannot "simply pick[] the cause that is most advantageous to [the plaintiff's] claim"); *In re Prempro Products Liab. Litig.*, No. 3:05CV00078-WRW, 2010 WL 8357351, at *2 (E.D. Ark. Sept. 26, 2010) ("Mentioning some risk factors and moving on to a conclusion, without a specific explanation, is not a proper differential diagnosis. . . . [it] is simply a recitation of facts—this does not help the jury.").

VI. Conclusion

The Court finds that Dr. Murphy's opinion is based only on (1) her conclusion that Lipitor increases the risk of diabetes and (2) that the fact that Ms. Hempstead was diagnosed with diabetes after taking Lipitor. She failed to point to any evidence, other than a (belated) temporal relationship, that Lipitor contributed to the development of diabetes in Ms. Hempstead's case. She failed to offer any explanation as to why Ms. Hempstead's other risk factors for diabetes (BMI, adult weight gain, age, hypertension, family history, and metabolic syndrome), alone or in combination, are not solely responsible for Ms. Hempstead's diabetes. In short, Dr. Murphy's opinion is not based on "sufficient facts or data," and to the extent that she purports to be applying a differential diagnoses methodology, she has not reliably applied this methodology. Therefore, her opinion is excluded under Rule 702 and *Daubert*, and Pfizer's motion, (Dkt. No. 1006), is **GRANTED**.

AND IT IS SO ORDERED.

Richard Mark Gergel
United States District Court Judge

December 11, 2015
Charleston, South Carolina