IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA
CHARLESTON DIVISION

IN RE: DIGITEK PRODUCT LIABILITY LITIGATION **MDL NO. 1968**

THIS DOCUMENT RELATES ONLY TO:

Kathy McCornack, an individual; et al., MDL No. 2:09-CV-0671

Plaintiffs,

v.

Actavis Totowa, LLC, et al.,

Defendants.

MEMORANDUM IN SUPPORT OF DEFENDANTS' MOTION TO EXCLUDE DECEDENT'S POSTMORTEM BLOOD DIGOXIN CONCENTRATION, MOTION TO EXCLUDE THE EXPERT TESTIMONY OF RICHARD MASON, M.D. AND KEITH GIBSON, AND MOTION FOR SUMMARY JUDGMENT

I. INTRODUCTION

Defendants are entitled to summary judgment under Federal Rule of Civil Procedure 56 because Plaintiffs have no admissible evidence that Mr. Daniel McCornack received or ingested defective Digitek®, or that his death was caused by digoxin toxicity. Having tested six Digitek® tablets that were in Mr. McCornack's possession when he died and found no direct evidence of product defect, Plaintiffs base their entire theory of defect and causation on Mr. McCornack's postmortem blood digoxin level, which is not a reliable indicator of what his level was before death. Plaintiffs advance two experts on these issues – Keith Gibson on both defect and causation and Richard Mason on causation only. Pursuant to Rule 702 and *Daubert*, Defendants request that this Court exclude both experts on these issues. Mr. Gibson fails spectacularly on qualifications grounds alone. Defendants also move to exclude any expert testimony based on an

unreliable extrapolation from a postmortem to an antemortem blood digoxin level by Dr. Mason and Mr. Gibson. Plaintiffs' own highly qualified non-retained expert, E.J. Barbieri, testified that such extrapolation is not reliable, and would not have advised Dr. Mason to change the cause of death based upon it. Dr. Barbieri's view is consistent with the overwhelming consensus in scientific literature and opinion. Indeed, this Court has excluded an expert's opinions when they are based on extrapolation, unsupported by any methodology. *See e.g., Bourne ex rel. Bourne v. E.I. Dupont de Nemours and Co., Inc.*, 189 F.Supp.2d 482, 499 (S.D.W.Va. 2002). Because this evidence is unreliable and thus inadmissible, Plaintiffs have not placed in issue any material facts relating to causation or product defect. Indeed, because Plaintiff's only case-specific evidence of product defect is Mr. Gibson's opinion, and since Mr. Gibson lacks qualifications to offer it, the Court could grant summary judgment on that basis alone. Either way, summary judgment is appropriate.

II. RELEVANT FACTS

Mr. McCornack had a long-standing history of atrial fibrillation. Ex. 1 (Deposition of Dr. Lawrence Von Dollen (McCornack's cardiologist)), 42:15-18; Ex. 2 (Deposition of Dr. Gordon Lemm (McCornack's primary care physician)), 25:4-11. He was also overweight and hypertense. Ex. 1 at 44:5-6; 45:8-17; Ex. 2 at 25:1-13. He died unexpectedly on March 23, 2008 at 12:52 a.m. None of the Plaintiffs' doctors wrote expert reports. And none gave testimony to a reasonable degree of medical probability that Mr. McCornack exhibited any signs of digoxin toxicity temporal with his death. Ex. 2 at 75:17-76:7; Ex. 1 at 67:20-68:6; Ex. 3 (Deposition of Dr. Richard Mason), 63:18-21.

During Mr. McCornack's autopsy nearly seventy-nine hours after Mr. McCornack's death, Dr. Mason drew blood from a non-ligated axillary vein.[1] Ex. 3 at 20:24-21:5, 24:17-25:6; 26:24-27:1. He sent this postmortem blood specimen to NMS Laboratories ("NMS") for analysis. In the meantime, Dr. Mason determined that Mr. McCornack's cause of death was his underlying health condition. *See* Ex. 3 at Mason Exs. 2, 3 attached thereto; 15:18-16:10. Indeed, based on the autopsy findings, Mr. McCornack's heart disease is certainly a plausible explanation for his sudden natural death. Ex. 3 at 62:21-63:7.

On June 24, 2008, NMS reported Mr. McCornack's postmortem blood to contain two drugs – diltiazem and digoxin – at three times their therapeutic levels in living humans. *See* Ex. 3 at Mason Ex. 7 attached thereto.[2] Mr. McCornack's postmortem digoxin concentration was 3.6 nanograms per milliliter ("ng/mL"). *Id.* Toxicologists, including Dr. Barbieri, agree that a digoxin concentration of 0.5 to 2.0 ng/mL is therapeutic in a living human. *See* Ex. 4 (DiGregorio, G.J., and E.J. Barbieri, *Handbook of Commonly Prescribed Drugs (with Therapeutic, Toxic, and Lethal Levels)*, 276 (Medical Surveillance, Inc.)). Fifteen months after receiving the blood result and after being retained as an expert by Plaintiffs, Dr. Mason revised Mr. McCornack's death certificate based solely on the postmortem blood digoxin concentration. *See* Ex. 3 at Mason Exs. 4, 14 attached thereto; 33:11-34:2; 55:4-7; 67:9-13. Dr. Mason concluded that Mr. McCornack died as a result of ventricular arrhythmia, digoxin toxicity, and digoxin poisoning. Ex. 3 at 18:1-16; 72:14-21. If he had been asked, Dr. Barbieri, the NMS toxicologist who signed the lab report, would not have advised Dr. Mason to change the cause of death based on the blood result. Ex. 5 (Deposition of Edward Barbieri), 77:12-19.

[1] The axillary vein runs from the subclavian vein in the chest through the armpit into the arm.
[2] Diltiazem is for ventricular rate control and blood pressure and has some of the same risks as digoxin. Ex. 1 at 25:7-26:12; 27:5-14.

Plaintiffs had no less than six Digitek® tablets tested from those that were in Mr. McCornack's possession at the time of his death. Ex. 6 (Deposition of Matthew McMullin), 16:11-23; Ex. 5 at 42:9-14; 44:10-21. All of the Digitek® tablets tested were found to be within the FDA-approved specifications. Ex. 6 at 21:19-23:5; Ex. 5 at 42:16-43:18.

III. OPINIONS OF DR. MASON AND MR. GIBSON

Plaintiffs offer Dr. Mason to testify as an expert on causation. *See* Ex. 3 at Mason Ex. 14. Plaintiffs state that he will testify that: 1) Mr. McCornack's postmortem blood digoxin concentration is "elevated"; 2) it "cannot be fully explained by the potential effects of post mortem distribution"; 3) it is "sufficiently elevated to confirm a toxic digoxin level and digoxin poisoning regardless of distribution considerations"; and 4) "Mr. McCornack died of a cardiac arrest due to ventricular arrhythmia, digoxin toxicity, and digoxin poisoning." *See* Ex. 7 (Pls. Summary of Non-Retained Expert Opinions Pursuant to F.R.C.P. 26(a)(2)(c)) at 8. He believes that the 3.6 ng/mL level is a representative number for Mr. McCornack's digoxin level at or about the time of death. Ex. 3 at 64:1-8.

Plaintiffs offer Mr. Gibson as an expert on causation and product defect. *See* Ex. 8 (Expert Report of Keith Gibson) at 1. Like Dr. Mason, Mr. Gibson concludes that: 1) "Mr. McCornack had an elevated digoxin level at the time of his demise"; 2) "[t]he elevated digoxin level was probably the result of a change in the formulation of the Digitek tablet or a non-conforming tablet" (i.e., Mr. McCornack's Digitek® was defective); 3) the elevated levels of digoxin caused digoxin toxicity; and 4) "digoxin poisoning was the cause of his death." Ex. 8 at 9. Specifically, at least as stated in his report, Mr. Gibson believes that Mr. McCornack's digoxin level at the time of death was 2.5 ng/mL. Ex. 8 at 8. Mr. Gibson's opinion, based on the postmortem blood digoxin concentration, is the only case-specific evidence Plaintiffs offer to prove product defect. But Mr. Gibson's opinion on product defect is a conditional one, subject

to Plaintiff's counsel being able to prove defect. Ex. 9 (Deposition of Keith Gibson), 83:13-23. As is discussed hereafter, Mr. Gibson's opinions are without any reliable basis, and he is wholly unqualified to give them in any event.

IV. MR. GIBSON'S OPINIONS ON PRODUCT DEFECT MUST BE EXCLUDED.

To be admissible, expert testimony must meet the standards set by Federal Rule of Evidence 702 and the Supreme Court's decision in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). *See Hines v. Wyeth, d/b/a Wyeth, Inc.*, No. 2:04-cv-00690, 2011 WL 2680718, *1-2 (S.D.W.Va. Jul. 8, 2011) (slip. op.). First, to give expert testimony, "a witness [must be] qualified as an expert by knowledge, skill, experience, training, or education." Fed. R. Evid. 702. Second, expert testimony must pertain to "scientific, technical, or other specialized knowledge." Fed. R. Evid. 702; *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 260 (4th Cir. 1999) (citing *Daubert*, 509 U.S. at 592). This requires that: 1) the testimony is based upon sufficient facts or data; 2) the testimony is the product of reliable principles and methods; and 3) the witness has applied the principles and methods reliably to the facts of the case. Fed. R. Evid. 702. Third, expert testimony is admissible only if it "will assist the trier of fact to understand the evidence or to determine a fact in issue." *Id.* In other words, all scientific testimony or evidence admitted must be reliable and relevant. *See Daubert*, 509 U.S. at 589.

A court must exclude a witness who possesses no knowledge, skill, experience, training, or education in the field implicated by the relevant issues. *See, e.g., Cooper v. Laboratory Corp. of America,* 150 F.3d 376, 381-82 (4th Cir. 1998) (excluding witness proffered to testify about a urine alcohol test even though the witness had practical experience with breath alcohol testing). Establishing knowledge, skill, experience, training or education in one field does not necessarily qualify one as a witness in an analogous field. *See e.g., Wehling v. Sandoz. Pharm. Corp.,* No. 97-2212, 1998 WL 546097 (4th Cir., Aug. 20, 1998).

A. Mr. Gibson is unqualified to give an expert opinion about product defect.

Mr. Gibson is the only expert in this case designated to opine that Mr. McCornack's Digitek® was defective, but he is simply not qualified in any of the fields implicated by his product defect opinions. He has three jobs – he is a part-time public defender, a part-time administrative law judge, and a part-time pharmacist in a hospital. *See* Ex. 8 at Resume of Keith Gibson; Ex. 9 at 26:14-27:24. Mr. Gibson's experience as an attorney and judge does not qualify him to testify as an expert here – on the contrary, it qualifies him to understand why he should not.

Mr. Gibson should not be permitted to testify about product defect because he has no relevant experience. He has never worked as an employee or consultant to a pharmaceutical company, and he has no experience in manufacturing, quality assurance, pharmaceutical testing, or regulatory work. Ex. 9 at 32:18-33:1; 165:9-166-1. He has not seen any of the regulatory documents, batch records, quality assurance or manufacturing documents produced in this litigation. Ex.9 at 65:4-10; 72:5-23. Nor does he have any experience in postmortem redistribution, which is a fundamental part of his defect opinion. *See* Sec. VI. (A), *infra,* at 12. Mr. Gibson's lack of relevant experience in these issues renders him unqualified to testify as to product defect.

B. Mr. Gibson misapplies differential diagnosis to the facts of this case to reach an unreliable conclusion about product defect.

The basis for Mr. Gibson's layman conclusion on product defect is his speculation that "something was different that night." Ex. 8 at 9; Ex. 9 at 160:4-161:18. Unsurprisingly, he has no scientific opinions about how Mr. McCornack's tablets were defective. Ex. 9 at 83:13-84:8; 160:4-166-8; 174:15-175:22. Specifically, he testified that Mr. McCornack received and ingested Digitek® that was properly dosed with digoxin; it was not defective by excess or

insufficient dose. Ex. 9 at 153:22-156:12). He speculated about "bioavailability" (an excess of available drug) in his report, but admitted at deposition that he did not have an opinion about this. Ex. 9 at 83:13-23. He explained that he was only stating that if Plaintiff's counsel could prove formulation issues, product defect would be a possible cause of a toxic dose. *Id.* He cannot testify to the essential elements of dose and duration[3] because he has no opinion about how many "nonconforming" tablets Mr. McCornack ingested or when he ingested them. Ex. 9 at 174:15-175:22. Further, Mr. Gibson, who is not qualified to engage in diagnosis in any event, has misapplied differential diagnosis by using the "likelihood of toxicity" factors to rule out the various possible things that might have caused Mr. McCornack to experience digoxin toxicity. Mr. Gibson rules out several factors based on a lack of evidence or because they were "highly unlikely," without explaining why product defect did not make it into either of those categories.

Further, Mr. Gibson fails to explain why he singled out Digitek® versus diltiazem as a causal factor when both were found in Mr. McCornack's postmortem blood at three times the therapeutic levels for living humans. Indeed, he has not read the diltiazem label and does not know the degree of increased risk for arrhythmias at higher diltiazem levels (Mr. McCornack was on the maximum daily dose). Ex. 9 at 93:2-3; 95:1-18. He also fails to explain how Mr. McCornack could have been toxic without exhibiting any of the signs of toxicity. Dying suddenly from digoxin toxicity, without first experiencing more general symptoms, is an unusual or unlikely scenario, and patients on digoxin can experience arrhythmias, even if not toxic. Doc. 522, Ex. 3 (Ex. 3 to General Background Statement, Deposition of Dr. Marc Semigran), 65:15-66:4. So Dan McCormack's sudden death proves nothing about his Digitek® dose or whether his tablets were defective.

[3] For a full discussion of dose and duration, please see Memorandum In Support of Defendants' Motion for Summary Judgment, Doc. 524, at 16, fn. 8.

Because Mr. Gibson's opinion is the only proffered evidence of product defect, the Court could grant summary judgment based on Mr. Gibson's lack of qualifications alone and not even consider the *Daubert* issues about reliability.

V. THERE IS NO SCIENTIFICALLY RELIABLE WAY TO EXTRAPOLATE FROM A POSTMORTEM TO AN ANTEMORTEM BLOOD DIGOXIN CONCENTRATION.

The Plaintiffs' defect and causation opinions all depend upon the interpretation of Mr. McCornack's postmortem blood digoxin level. This is their bedrock, whether they calculate a number or just conclude it was a high or toxic level. While toxicological analysis of a postmortem blood specimen can sometimes assist in determining the cause of an individual's death, it cannot do so here because of a phenomenon called postmortem redistribution ("PMR"). PMR is the process by which a substance stored in body tissue at relatively high concentrations while a person is alive migrates after death to areas of the body, most notably blood, that contain lower concentrations of the substance. Ex. 10 (Richard E. Ferner, *Post-mortem Clinical Pharmacology*, 66(4) British Journal of Clinical Pharmacology 430, 435 (2008)); Ex. 9 at 104:14-107:5. PMR, consequently, can cause postmortem blood drug concentrations to be significantly higher than antemortem blood drug concentrations. Ex. 11 (William H. Anderson, *Postmortem Redistribution of Drugs Must Be Considered*, Clinical and Forensic Toxicology News, March 2004) at 4.[4] The effects of PMR increase with the amount of time that elapses between death and the blood draw. *Id.* And according to Dr. Barbieri, seventy eight hours is a very long time in terms of postmortem redistribution. Ex. 5 at 68:4-8. The extent to which PMR affects postmortem blood drug concentrations also depends on the method of collection, site sampled, ambient temperature, physical properties of the drug, preservative used, position of the

[4] Under Federal Rule of Evidence 803(18), Defendants' experts will testify to the reliability of the scientific references cited within this brief.

body, and the amount of unabsorbed drug present at the time of death. *See* Ex. 12 (Steven B. Karch, *Alternate Strategies for Postmortem Drug Testing*, 25 Journal of Analytical Toxicology 393, 393 (2001)). Postmortem redistribution occurs at all sites, but blood specimens drawn from or near the heart are more prone to PMR. *See, e.g.*, Ex. 13 (S.M. Fletcher et al., *Radioimmunoassay of Cardiac Glycosides in Haemolysed Blood: Derivation of Serum Levels*, 19 J. Forensic. Sci Soc. 183, 183 (1979)); Ex. 5 at 67:10-68:21. Mr. McMullin, who has published on PMR of other drugs, agrees. Ex. 6 at 45:5-16; 51:2-9. "[E]ven taking all these variables into account, there is still no assurance that drug concentrations measured after death bear any relationship at all to concentrations measured in life." Ex. 12 at 393.

It is well known within the toxicological community that digoxin undergoes PMR, but the extent to which PMR increases digoxin concentrations in postmortem blood is unknown and varies based on multiple factors. *See, e.g.*, Ex. 14 (Randall C. Baselt, Disposition of Toxic Drugs and Chemicals in Man 462 (Biomedical Publications 2008)). The magnitude that PMR and other factors can increase postmortem blood digoxin concentrations varies wildly and is still not understood by toxicologists. For instance, one article concluded that "post mortem levels may be 1.5 to 10 times higher than ante-mortem levels." Ex. 15 (Gideon Koren & Ruth Parker, *Interpretation of Excessive Serum Concentrations of Digoxin in Children*, 55 Am. J. of Cardiology 1210, 1213 (1985)). Consequently, the consensus in the toxicological community is that "one cannot readily use…post mortem data to predict ante-mortem concentrations." *Id.*

Despite decades of studying postmortem blood digoxin concentrations, toxicologists have not been able to reliably extrapolate antemortem blood digoxin concentrations from postmortem concentrations. In 2000, Drs. Cook and Braithwaite concluded that "[t]here is a lack of evidence that such an extrapolation is possible" and that such a calculation was "dangerous" and "not

recommended." Ex. 5 at 96:11-98:19; Ex. 16 (D.S. Cook et al., *Estimating antemortem drug concentrations from postmortem blood samples: the influence of postmortem redistribution*, 53 J. of Clinical Pathology 282, 284 (2000)). The study went on to say that "a high degree of error can arise from attempting to predict ante-mortem concentrations from post-mortem concentrations" and emphasized "the need for continued research into this area of pathology practice." *Id.*. Toxicologists came to the same conclusions in 2008, stating that "[t]here is no reliable or obvious connection between concentrations measured in life and subsequent to death. Consequently, concentrations measured after death cannot generally be interpreted to yield concentrations present before death." Ex. 5 at 88:19-90:8; Ex. 10 at 440.

Even plaintiffs' experts themselves have admitted that there is no reliable way to determine the extent to which PMR impacted Mr. McCornack's postmortem blood. Dr. Mason stated:

> Would I want to know how exactly [PMR] worked, how it was quantified? Yeah, if I could, but I can't. There is no way for me to know these things. ...It would be some esoteric fudge factor for me to say how much was redistributed. I don't know. I don't know that somebody could tell you well, maybe it occurs this much in somebody, and it may be this much in somebody else. I don't know.

Ex. 3 at 54:21-23; 55:18-22. Mr. Gibson stated, "I mean, I think Dig [digoxin] levels with postmortem redistribution do tell you stuff, but I don't think you can extrapolate backwards on one data point." Ex. 9 at 16:3-10. Mr. Gibson also testified that he could not "extrapolate from that [postmortem] level back to the time of [Mr. McCornack's] death with any accuracy." Ex. 9 at 118:2-3. When asked whether he had performed any independent research about the degree to which digoxin levels can increase, Mr. Gibson further stated:

> When I started looking at the literature, I didn't really think that you would be asking me questions to extrapolate backwards on this Dig (digoxin) level. The Dig (digoxin) level is what it is, and it's just a data

> point. It could be ten times greater. It could be half as much. I don't know. There's a lot of factors involved here.

Ex. 9 at 170:24-171:9. Dr. Barbieri of NMS, a pharmacologist by education and toxicologist by training, testified that the extrapolation of antemortem concentrations from postmortem concentrations is "fraught with all kind of perils." Ex. 5 at 73:8-12.

Thus, there is no scientifically reliable way to determine Mr. McCornack's antemortem blood digoxin concentration or his clinical diagnosis from his postmortem level.

VI. DR. MASON'S AND MR. GIBSON'S CAUSE OF DEATH OPINIONS MUST BE EXCLUDED.

A. Dr. Mason and Mr. Gibson lack the qualifications to render their proffered opinions.

Neither Dr. Mason nor Mr. Gibson should be permitted to testify about PMR, postmortem blood digoxin concentrations, and digoxin toxicity because they lack requisite education, training, knowledge, skill, and experience. *See, e.g., Cooper,* 150 F.3d at 381-82; *Wehling,* 1998 WL 546097. In *Wehling*, the Court excluded an expert's testimony that the interaction of two drugs in Plaintiff's body caused her injury because although the expert was a pharmacist and toxicologist and had assisted "medical examiners in determining causes of death", he was "neither a pharmacologist nor a medical doctor." *Id.* at *3-4. Furthermore, the expert had no education, training, or experience in the treatment of patients with the drug at issue. *Id.* at *4. An individual cannot cure this lack of education, training, and experience by reading literature for litigation. *See, e.g., United States v. Paul*, 175 F.3d 906, 912 (11th Cir. 1999) (an individual's "review of literature" in an area outside his field does "not make him any more qualified to testify as an expert ... than a lay person who read the same articles."); *Newton v. Roche Laboratories, Inc.*, 243 F.Supp.2d 672, 678 (W.D.Tex. 2002) (pharmacist was not qualified to testify that Accutane could cause schizophrenia, where he had no relevant expertise

or experience "outside of that which he has gleaned from a scant literature review for the purpose of consulting and testifying in this case").

Like the expert in *Wehling*, Dr. Mason and Mr. Gibson have expertise in one area, but are offering testimony in another. Mr. Gibson is a part-time pharmacist and Dr. Mason is a long-time coroner. Ex. 9 at 27:23-29:8; Ex. 3 at 5:4-9. Neither expert is a toxicologist or has education, training, skill, knowledge, and experience in digoxin, digoxin toxicity, and PMR. Ex. 3 at 7:23-8:15; Ex. 9 at 118:19-121:3. Neither has ever performed an experiment, published, or presented anything about digoxin toxicity or postmortem redistribution. Ex. 3 at 7:23-8:15; 42:18-20; 57:13-16; Ex. 9 at 44-45; 112:18-20. Mr. Gibson has never diagnosed digoxin toxicity and never read anything about postmortem blood analysis and postmortem redistribution before he was hired as an expert for this litigation. Ex. 9 at 48:6-49:3, 69:14-70:7, 124:21-125:3. Dr. Mason did no research about PMR, and cannot recall a single case in which digoxin was a potential or actual cause of death. Ex. 3 at 7:23-8:15; 42:18-20. Dr. Mason also admitted that he cannot testify as to the meaning of Mr. McCornack's postmortem blood digoxin concentration or whether it was affected by PMR. Ex. 3 at 42:6 13; 55:18-22. Neither Dr. Mason nor Mr. Gibson is qualified to opine as to Mr. McCornack's cause of death based on his postmortem blood digoxin concentration. Moreover, neither expert can cure this lack of experience by reading literature for this litigation.

Further, Mr. Gibson lacks the qualifications to offer the two medical diagnoses contained in his report – that Mr. McCornack had digoxin toxicity and that he died from it – because his legal and pharmacy expertise do not qualify him to diagnose anything. First, given his admission that he is neither a doctor, nor a toxicologist, Mr. Gibson clearly lacks the education and training to offer these opinions. Ex. 9 at 118:19-121:3. In fact, in California, where he lives and works,

he is prohibited from providing diagnoses. Cal. Bus. & Prof. Code § 2052(a) states that any person who diagnoses any disease, disorder, or other physical condition of any person without having a valid license from the Board of Medicine is guilty of a fourth degree felony. "Diagnose" and "diagnosis" include any undertaking by any method to ascertain whether a person is suffering from any disorder. Cal. Bus. & Prof. Code §2038. Determining a cause of death here is also a diagnosis. *See Manocchio v. Moran*, 919 F.2d 770, 780 (1st Cir. 1990) (a determination of the cause of death following autopsy is, in effect, a medical diagnosis prepared by the pathologist). California law thus requires that death certificates be signed by a physician, surgeon, or coroner. Cal. Health & Safety Code §102875(a)(7). Knowing that he cannot legally make a diagnosis, Mr. Gibson testified that his "role here is not necessarily to make that diagnosis so much as to support the diagnosis already made by the... physicians." Ex. 9 at 21:17-22:7. Mr. Gibson's report contains no such qualifier. Ex. 9 at 22:19-23:2.

Courts consistently bar experts who lack medical education or training from testifying as to a diagnosis or cause of death. *See, e.g., Newton v. Roche Laboratories, Inc.*, 243 F.Supp.2d 672, 678 (W.D.Tex. 2002) (pharmacist with no M.D. or Ph.D. degree was not qualified to testify on general causation); *Fulton v. Loucks*, No. 90-6552, 1991 WL 224107, at *1 (6th Cir. Oct. 31, 1991) (excluding cause of death in death certificate where coroner was not a doctor); *Fanning v. Sitton Motor Lines, Inc.,* No. 08-CV-2464 CM/DJW, 2010 WL 4261476, at *3 (D.Kan. Mar. 30, 2010) (slip op.) (finding toxicologist, who was not a medical practitioner, unqualified to opine as to whether the decedent suffered withdrawal symptoms after cessation of medication); *Conde v. Velsicol Chem. Corp.,* 804 F.Supp. 972, 989, 1001-02 (S.D.Ohio 1992), aff'd, 24 F.3d 809 (6th Cir.1994) (concluding that toxicologist could not make a clinical diagnosis because he was not a medical doctor).

Second, Mr. Gibson lacks the experience to diagnose digoxin toxicity in a living patient or to attest to cause of death in the deceased. He does not diagnose patients in general. Ex. 9 at 38:13-15. He has never helped a doctor diagnose a patient with digoxin toxicity. Ex. 9 at 49:1-3. He has never rendered any diagnosis in a written medical record. Ex. 9 at 41:21-23. He has never prescribed a drug. Ex. 9 at 49:4-7. And he has never signed a death certificate, performed an autopsy, or rendered an opinion on cause of death. Ex. 9 41:9-20; 48:14-25. Experts with far more experience than Mr. Gibson have been excluded because they had never done what they proposed to do in litigation. *See Cooper*, 150 F.3d at 381-82 (excluding witness' testimony on urine alcohol testing, even though witness had experience with breath alcohol testing); *Lareau v. Page*, 840 F.Supp. 920 (D.Mass.1993), affirmed 39 F.3d 384 (absent showing that doctor was neurosurgeon or had ever dealt with medical decisions facing neurosurgeon who utilized contrast injection on patient, doctor was not qualified to testify on adequacy of contrast injection warnings made to neurosurgeons about use of contrast injection in brain surgery).

Lastly, Mr. Gibson does not have any specialized skill or knowledge about Mr. McCornack's underlying illness. He is not an expert in sudden cardiac death nor does he know anything about the risk of arrhythmia in patients with Mr. McCornack's conditions. Ex. 9 at 42:2-4. In short, Mr. Gibson is no more than a layperson with respect to clinical medicine and toxicology. Both he and Dr. Mason lack the requisite expertise about postmortem redistribution and digoxin.

As set forth above, Dr. Mason and Mr. Gibson should be excluded from giving any evidence as to Mr. McCornack's antemortem digoxin level, alleged digoxin toxicity, or cause of death.

B. Dr. Mason's and Mr. Gibson's opinions are unreliable because they rely on Mr. McCornack's postmortem blood digoxin concentration without any methodology other than the *ipse dixit* of the expert.

The opinions of Dr. Mason and Mr. Gibson are based on unreliable methodology. Dr. Mason concludes that there was no postmortem redistribution, a conclusion at odds with all scientific literature. *See* Sec. V., *supra,* at 8. It is also directly at odds with Dr. Barbieri's testimony. Ex. 5 at 68:13-74:17. Mr. Gibson uses the postmortem blood level to extrapolate back to some conclusion about Mr. McCornack's antemortem serum level, or a diagnosis of toxicity, or both. There is no reliable scientific method for doing so, and those opinions should be excluded.

It is "essential to ensure that only scientifically reliable methods are used to generate the opinions offered to a jury." *Dunn v. Sandoz Pharm. Corp.*, 275 F.Supp.2d. 672, 676 (M.D.N.C. 2003). The party offering the evidence bears the burden of establishing admissibility by a preponderance of the evidence. *See e.g., Battle v. Gold Kist, Inc.*, No. 3:06-cv-782-J-32TEM, 2008 WL 4097717, *3 (M.D. Fla., Sept. 2, 2008) (citing *Hall v. United Ins. Co. of Am.*, 367 F. 3d 1255, 1261 (11th Cir. 2004)). "[C]arrying this burden requires more than 'the *ipse dixit* of the expert.'" *Id.* (citation and internal quotation marks omitted) (quoting *Cook ex rel. Estate of Tessler v. Sheriff of Monroe County, Fla.*, 402 F. 3d 1092, 1113 (11th Cir. 2005)). A court may consider four non-exclusive factors in determining whether an expert's testimony is reliable: 1) whether a theory or technique can be or has been tested; 2) whether it has been subjected to peer review and publication; 3) whether a technique has a high known or potential rate of error and whether there are standards controlling its operation; and 4) whether the theory or technique enjoys general acceptance within a relevant scientific community. *Daubert*, 509 U.S. at 592-94.

An expert's opinions are not built on indicia of reliability when they are based on extrapolation of an individual's exposure to an agent without any methodology. *See, e.g.,*

Bourne ex rel. Bourne v. E.I. Dupont de Nemours and Co., Inc., 189 F.Supp.2d 482, 499 (S.D.W.Va., 2002). In *Bourne*, the plaintiff's expert testified that the plaintiff's mother's exposure to a fungicide while she was pregnant caused the plaintiff's birth defects. *Id.* at 484. To come to this conclusion, the plaintiff's expert estimated the amount of fungicide to which the plaintiff's mother was exposed. *Id.* at 485. The plaintiff's expert conducted a "back-calculation" to estimate the plaintiff's mother exposure, based on the volume of the fungicide required to achieve the minimum fungicide concentration found by numerous studies to affect cells. *Id.* at 486. The court concluded that the expert's calculation of the plaintiff's exposure was "purely speculative" at best and "devised to ensure that a certain desired [exposure] was met" at worst. *Id.* at 499. Furthermore, the exposure rate was not "capable of being tested or reproduced." *Id.* The court ultimately rejected this extrapolation, as being "contrary to principles of sound scientific method," and ultimately excluded the plaintiff's expert. *Id.* at 499, 501.

Extrapolations of postmortem to antemortem blood drug concentrations cannot survive reliability challenges. *See, e.g.*, *Battle*, 2008 WL 4097717. In *Battle*, the plaintiff argued that her decedent was struck by a forklift negligently operated by one of the defendants' employees. *Id.* at *1-2. The defendant's expert relied on a postmortem femoral blood specimen reflecting marijuana use to opine that he was impaired. *Id.* at *2. The court excluded the expert's testimony, finding that he did not provide "an adequate basis for establishing the reliability of the [postmortem] femoral blood test." *Id.* at *7. The court pointed to the expert's own literature, including one piece that stated that extrapolations of antemortem blood concentrations from postmortem blood concentrations "are prone to considerable error and generally should be viewed as unreliable and not evidence based." *Id.* at *4-5 (citing Ex. 17, Olaf Drummer et al., 329 Forensic Science in the Dock, British Medical Journal, 636 (2004)).

Dr. Mason and Mr. Gibson have failed to establish the reliability of their respective views of Mr. McCornack's postmortem blood digoxin concentration or provide any scientific explanation of their extrapolation to either an antemortem concentration or his clinical condition. We can start with the Plaintiffs' expert pharmacologist's agreement that one cannot take an "isolated serum level and deduce toxicity . . ." Doc. 522, Ex. 4 (Ex. 4 to General Background Statement, Deposition of E. Don Nelson), 73:13-14. Dr. Von Dollen agreed. Ex. 1 at 33:2-34:11. Next, Drs. Mason, Lemm, and Dr. Von Dollen were unable to state to a reasonable degree of medical probability that Mr. McCornack had clinical signs or symptoms of digoxin toxicity before he died. Ex. 2 at 75:17-76:7; Ex. 1 at 67:20-68:6; Ex. 3 at 63:18-21. Yet Dr. Mason testified at his deposition that he "think[s] 3.6 [ng/mL] represents a toxic level." Ex. 3 at 56:5. Extrapolating toxicity from a level alone with no clinical signs is not a reliable method. Nor is merely accepting "the [postmortem] 3.6 as a representative number for his digoxin level at or about the time that [Mr. McCornack] died" a reliable method. *See* Ex. 3 at 64:1-11. Dr. Mason testified that he relied upon the postmortem concentration "Because it's what I've got. And that's the way I'm doing it." *Id.* That is impermissible *ipse dixit.* And in addition to Dr. Barbieri, even Mr. Gibson agrees that 3.6 ng/mL was not Mr. McCornack's digoxin level when he died. Ex. 9 at 168:14-17.

Dr. Mason testified that he did not concern himself with postmortem redistribution because Mr. McCornack's blood was drawn from a peripheral site. Ex. 3 at 48:18-24. But the literature says PMR occurs at all sites. Ex. 18 (Pelissier-Alicot, Mechanisms Underlying Postmortem Redistribution of Drugs: A Review, 27 Journal of Analytical Toxicology 533, 541 (Nov/Dec 2001) (specifically noting that while femoral veins are the best site from which to draw blood, they are still subject to postmortem redistribution.); *see* also, Ex. 13; Ex. 5 at 68:13-

21; Ex. 6 at 45:5-16; 51:2-9, 74:6-23. There is no literature support for the proposition that peripheral vessels experience no postmortem redistribution.

Furthermore, Dr. Mason's testimony regarding Mr. McCornack's cause of death is inconsistent with the opinions of forensic toxicologists, upon whom Dr. Mason testified he would rely. Dr. Mason chose NMS to analyze Mr. McCornack's blood because of their skill. Ex. 3 at 28:6-15. When he was asked if he would want to know what the forensic toxicologists at NMS thought about the meaning of the 3.6 ng/mL blood result, he testified that:

> Yeah, it would [be of interest to me to know what a forensic toxicologist said about the 3.6 ng/mL result] because the asshole didn't put anything in his report and I would hope he would convey something to me. That's what we pay them for. But they are very noncommittal, so I would be really anxious to hear what he had to say.

Ex. 3 at 42:6-13. Two NMS forensic toxicologists have testified in this case. Consistent with the scientific literature, Dr. Barbieri – the NMS toxicologist who signed the report – testified that "more likely than not" Mr. McCornack's antemortem blood digoxin concentration was "substantially lower" than 3.6 ng/mL. Ex. 5 at 74:6-15. Even if this postmortem concentration was representative of Mr. McCornack's antemortem concentration, Dr. Barbieri testified that it would not necessarily be fatal. Ex. 5 at 77:6 9. Thus, Dr. Barbieri testified that he "probably would not have" advised Dr. Mason to conclude that Mr. McCornack died as a result of digoxin toxicity. Ex. 5 at 77:12-19. One of Dr. Barbieri's colleagues at NMS, Mr. Matthew McMullin, agreed and testified in this case that, "standing alone," Mr. McCornack's postmortem blood digoxin concentration of 3.6 ng/mL is not necessarily fatal and does not provide any information regarding his cause of death. Ex. 6 at 86:18-21. It is not a reliable indicator of Mr. McCornack's antemortem level, and Dr. Mason cannot conclude it was toxic. Ex. 6 at 74:6-23; 87:10-88:5.

Mr. Gibson, on the other hand, attempts to iterate a methodology explaining his extrapolation in his report, but then testified that such an extrapolation was not reliable. *See* Ex.

8 at 9; *compare* Ex. 9 at 118:2-3; 16:3-1. At deposition, Mr. Gibson testified that, "I don't think you can extrapolate backwards on one data point" and that he could not "extrapolate from that [postmortem] level back to the time of [Mr. McCornack's] death with any accuracy." *See* Ex. 9 at 118:2-3; 16:3-10. Furthermore, Mr. Gibson's extrapolation is subject to enormous error and uncertainty, as he testified that Mr. McCornack's antemortem blood digoxin concentration "could be ten times greater. It could be half as much. I don't know. There's a lot of factors involved here." *See* Ex. 9 at 170:24-171:9. And Dr. Barbieri, Plaintiffs' non-retained expert, testified that, given all the variables in this case, there was no reliable way to back calculate an antemortem level from a postmortem one. Ex. 5 at 73:8-74:17.

Mr. Gibson nonetheless performed a calculation in his report, arriving at a range of antemortem digoxin levels for Mr. McCornack, based on data from one article. Ex. 8 at 8; *see*, Ex. 19 (Vorpahl T.E. and Coe J.L., Correlation of ante mortem and post-mortem digoxin levels, J. Forensic Sci. (1978) 23(2); 329-34). This article used data from three draw sites (heart, subclavian, and femoral), none of which are the draw site Dr. Mason chose in this case. Unsurprisingly, when that "theory" was tested at deposition, Mr. Gibson could not support his calculation to a scientific probability. First, the Vorpahl article, upon which Mr. Gibson relied to make his calculation, does not say one can use those formulas to reliably predict antemortem levels. It says the opposite:

> It is clear from this investigation that post-mortem digoxin levels taken from cardiac blood, venous blood or vitreous humor do not mirror the ante-mortem levels. Substantial increases in serum levels occur following death irrespective of the source of the sample.

Ex. 19 at 333; Ex. 9 at 131:1-133:18. And they drew their samples between 1 and 22.4 hours after death, a far cry from the 78 hours in this case. *Id.* The literature and Dr. Barbieri make clear that time matters. *See* Sec. V., *supra*, at 8.

Next, when asked if he had found a single piece of peer-reviewed literature supporting reliable predictions of antemortem levels from postmortem ones, Mr. Gibson said "no." Ex. 9 at 12:1-13:7. He further conceded that there were too many variables and "that's not real science." Ex. 9 at 13:15-14:3. And when asked whether the Vorpahl article's formulas were accurate, he said "whether that's reliable or not, I don't think it is." Ex. 9 at 16:3-10. In fact, he expressed that he was not sure who had been the "proponent of the idea extrapolating this Dig [digoxin] level back. I'm certainly not." Ex. 9 at 134:12-22. And of course Mr. Gibson only read this article for litigation and has never been called upon in his career to make such an assessment. *See* Sec. VI. (A), *supra,* at 12-13.

Not only do Dr. Mason and Mr. Gibson fail to provide any methodology, but they do not account for factors well-recognized in the literature to affect postmortem drug concentrations. *See* Sec. V., *supra*, at 10-11. Dr. Mason testified that, to incorporate the effects of PMR, he would have to apply an "esoteric fudge factor." Ex. 3 at 55:18-22. Mr. Gibson did not take into account the amount of time between Mr. McCornack's death and the blood draw, a factor known within the literature to affect PMR. Ex. 9 at 117:24-188:4. Mr. Gibson failed to consider whether Mr. McCornack's blood specimen, taken from an axillary vein, was a heart or peripheral sample. Ex. 9 at 77:24-78:3. The source of the postmortem blood specimen can have significant impact on the measured postmortem drug concentration. *See* Sec. V, *supra,* at 8-9.

Dr. Mason's and Mr. Gibson's conclusions are the very type of '*ipse dixit* of the expert' that Rule 702 seeks to exclude. Like the excluded expert in *Bourne,* Dr. Mason's and Mr. Gibson's conclusions that Mr. McCornack's antemortem concentration was toxic is "purely speculative" at best and "devised to ensure that a certain desired [exposure] was met" at worst. Expert testimony about antemortem drug concentrations, based on "esoteric fudge factor[s]"

because they "could be ten times greater" or "half as much," according to Plaintiffs' experts, is unreliable, misleading, and would lead to jury confusion. And sudden cardiac death alone is not enough to make causation in this case; there were admittedly plausible explanations for Mr. McCornack's death because of his heart disease. Dr. Mason's own consultant, NMS Labs, would not have advised him to change the cause of death. Without knowing the patient's serum digoxin level while alive, admitting there were no clinical signs or symptoms of toxicity, and having no electrocardiogram available, Plaintiffs' experts have no reliable scientific methodology to attribute the death to digoxin toxicity.

VII. MR. MCCORNACK'S POSTMORTEM BLOOD DIGOXIN CONCENTRATION SHOULD BE EXCLUDED BECAUSE IT IS UNRELIABLE AND LIKELY TO LEAD TO CONFUSION.

Evidence that is otherwise relevant may be excluded if "its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence." Fed. R. Evid. 403. In *Battle*, the court excluded Plaintiff's decedent's postmortem femoral blood drug test results, concluding that the scientific community cannot meaningfully extrapolate antemortem drug levels from postmortem drug results, even those based on the relatively more reliable femoral blood. *Battle* 2008 WL 4097717, at *4-5, 8. Mr. McCornack's postmortem blood digoxin concentration should likewise be excluded. His postmortem blood result is even less reliable than that in *Battle* because it was drawn from a source closer to the heart and is thus more prone to PMR. Failure to exclude this postmortem blood result would confuse the issues and mislead the jury.

VIII. SUMMARY JUDGMENT UNDER FEDERAL RULE CIVIL PROCEDURE 56 IS APPROPRIATE BECAUSE PLAINTIFFS HAVE PRESENTED NO ADMISSIBLE EVIDENCE TO SUPPORT THEIR CLAIM THAT MR. MCCORNACK DIED AS A RESULT OF DEFECTIVE DIGITEK®.

"Summary judgment is appropriate only when the court, viewing the record as a whole and in the light most favorable to the non-moving party, finds there is no genuine issue of material fact and that the moving party is entitled to a judgment as a matter of law." *Wehling*, 1998 WL 546097, at *5 (citing Fed. R. Civ. P. 56(c)). Expert testimony is required for a plaintiff to establish causation under a products liability claim. *See, e.g., Rohrbough v. Wyeth Laboratories, Inc.*, 916 F.2d 970, 972 (4th Cir. 1990). "Under California law, proof of causation in this context requires that [Plaintiffs] demonstrate to a 'reasonable medical probability based upon competent expert testimony that the defendant's conduct contributed to [their] injury.'" *In re Baycol Prods. Litig.,* 596 F.3d 884, 889 (8th Cir. 2010) (citing *Rutherford v. Owens-Illinois, Inc.*, 16 Cal.4th 953 (1997) (citation omitted); *see also Bockrath v. Aldrich Chem. Co., Inc.*, 21 Cal. 4th 71 (1999). Probability is a higher standard than possibility, requiring Plaintiffs to prove that, "in the absence of other reasonable causal explanations, it [is] more likely than not that the injury was a result of [Defendants'] action. This is the outer limit of inference upon which an issue may be submitted to the jury." *In re Baycol Prods. Litig.,* 596 F.3d at 889 (citing *Jones v. Ortho Pharm. Corp.*, 163 Cal.App.3d 396 (1985)). If such expert testimony is excluded, summary judgment is appropriate. *Cooper v. Smith & New, Inc.*, 259 F.3d 194, 203 (4th Cir. 2001) (granting summary judgment for defendant when plaintiff's expert witness, testifying that a medical device caused plaintiff's injury, was excluded).

Summary judgment is appropriate here because Plaintiffs have provided no admissible expert testimony establishing that the death of Mr. McCornack was due to defective Digitek®. Dr. Mason and Mr. Gibson have improperly relied on the postmortem digoxin blood

concentration because there is no other evidence to suggest that Mr. McCornack had an elevated digoxin level in his blood prior to his death. Because their testimony must be excluded for the reasons set forth above, Plaintiffs must prove causation and product defect by other means.

But Mr. McCornack's treating physicians offer no evidence to a medical probability establishing causation. Mr. McCornack's cardiologist, Dr. Lawrence Von Dollen, and his primary care physician, Dr. Gordon Lemm, both testified that they had no evidence indicating that Mr. McCornack experienced the signs or symptoms of digoxin toxicity on the day he died. Ex. 2 at 33:18-34:1; 75:17-76:7; Ex. 1 at 67:20-68:6. Dr. Von Dollen testified that an elevated serum digoxin level does not necessarily equate to toxicity, nor is it necessarily fatal, nor does it prove excess dose. Ex. 1 at 33:2-14; 52:22-53:1. While Dr. Von Dollen speculated that Mr. McCornack might have had digoxin toxicity, he noted that he "could easily be wrong," Ex. 1 at 82:20-84:4, and he quickly agreed that even if Mr. McCornack had had digoxin toxicity, he had no idea as to the cause. Ex. 1 at 85:8-10. His speculation included relying on the "elevated" level, which is unreliable, as set forth above, because it is based on extrapolations that are unsupported by any methodology.

Likewise, Dr. Lemm was "suspicious" of toxicity, but has no opinion to a reasonable probability that Mr. McCornack actually experienced digoxin toxicity. Ex. 2 at 23:1-10. Mr. McCornack had heart disease, which could account for his death. Ex. 2 at 25:1-13. An elevated serum level is not proof of excess dose. Ex. 2 at 39:19-24. Dr. Lemm had no means of calculating Mr. McCornack's antemortem blood digoxin concentration, Ex. 2 at 55:21 -56:2; said he was not an expert qualified to interpret the postmortem level, Ex. 2 at 70:25-71:7; 95:11-19; and had not seen anything indicating that Mr. McCornack had ingested excessive doses of

digoxin, Ex. 2 at 83:16-21. Thus, Plaintiffs have no expert to testify as to specific causation or product defect and no means of proving these essential elements of their claims.

IX. CONCLUSION

Defendants respectfully request that this Court grant summary judgment as Plaintiffs have failed to provide any admissible evidence that Mr. McCornack died as a result of ingesting defective Digitek®.

Respectfully submitted,

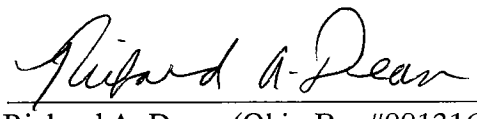
GUTHRIE & THOMAS, PLLC
Rebecca A. Betts, LIAISON COUNSEL
500 Lee Street East, Suite 800
Charleston, West Virginia 25301
Tel: (304) 345-7250
Fax: (304) 345-9941
E-mail: rabetts@agmtlaw.com

Attorney for Defendants

SHOOK, HARDY & BACON LLP
Harvey L. Kaplan, CO-LEAD COUNSEL
Madeleine M. McDonough, CO-LEAD COUNSEL
2555 Grand Blvd.
Kansas City, Missouri 64108-2613
Tel: (816) 559-2214
Fax: (816) 421-5547
E-mail: hkaplan@shb.com
E-mail: mmdonough@shb.com

Attorneys for Mylan Pharmaceuticals Inc., Mylan Bertek Pharmaceuticals Inc., and UDL Laboratories, Inc.

TUCKER ELLIS & WEST LLP

By: 

Richard A. Dean, (Ohio Bar #0013165)
CO-LEAD COUNSEL
Matthew P. Moriarty (WV Bar #4571)
925 Euclid Avenue, Suite 1150
Cleveland, OH 44115-1414
Tel: (216) 592-5000
Fax: (216) 592-5009
E-mail: richard.dean@tuckerellis.com
matthew.moriarty@tuckerellis.com

Attorneys for Defendants Actavis Inc., Actavis Elizabeth LLC, and Actavis Totowa LLC

CERTIFICATE OF SERVICE

I hereby certify that on August 3, 2011, a copy of the foregoing was filed electronically. Notice of this filing will be sent to all parties by operation of the Court's electronic system. Parties may access this filing through the Court's system.

Attorney for Plaintiffs

GUTHRIE & THOMAS, PLLC

Rebecca A. Betts, LIAISON COUNSEL
500 Lee Street East, Suite 800
Charleston, West Virginia 25301
Tel: (304) 345-7250
Fax: (304) 345-9941
E-mail:rabetts@agmtlaw.com

Attorney for Defendants

SHOOK, HARDY & BACON LLP

Harvey L. Kaplan, CO-LEAD COUNSEL
Madeleine M. McDonough, CO-LEAD COUNSEL
2555 Grand Blvd.
Kansas City, Missouri 64108-2613
Tel: (816) 559-2214
Fax: (816) 421-5547
E-mail:hkaplan@shb.com
E-mail:mmdonough@shb.com

Attorneys for Mylan Pharmaceuticals Inc., Mylan Bertek Pharmaceuticals Inc., and UDL Laboratories, Inc.

TUCKER ELLIS & WEST LLP

By: Richard Dean

Richard A. Dean, (Ohio Bar #0013165)
CO-LEAD COUNSEL
Matthew P. Moriarty (WV Bar #4571)
925 Euclid Avenue, Suite 1150
Cleveland, OH 44115-1414
Tel: (216) 592-5000
Fax: (216) 592-5009
E-mail:richard.dean@tuckerellis.com
matthew.moriarty@tuckerellis.com

Attorneys for Defendants Actavis Inc., Actavis Elizabeth LLC, and Actavis Totowa LLC