UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

IN RE: JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES AND PRODUCTS LIABILITY LITIGATION	MDL Docket No. 2738
This Document Relates To All Cases	

DEFENDANTS JOHNSON & JOHNSON AND JOHNSON & JOHNSON CONSUMER INC.'S POST-*DAUBERT* HEARING BRIEF

DRINKER BIDDLE & REATH LLP
A Delaware Limited Liability Partnership
600 Campus Drive
Florham Park, New Jersey 07932
Telephone: (973) 549-7000

SKADDEN, ARPS, SLATE,
MEAGHER & FLOM LLP
1440 New York Avenue, N.W.
Washington, D.C. 20005
Telephone: (202) 371-7000

Attorneys for Defendants
Johnson & Johnson and Johnson and
Johnson Consumer, Inc.

TABLE OF CONTENTS

Page

TABLE OF AUTHORITIES

Page(s)

CASES

OTHER AUTHORITIES

INTRODUCTION

The five experts plaintiffs chose to testify at the Court's *Daubert* hearing were presumably the ones they believed would provide the strongest evidence in support of plaintiffs' general causation theories. Instead, the testimony of these experts highlighted all the reasons why their opinions are unreliable, including, most notably, that they are advancing outlier opinions that contradict the consensus of the scientific and medical communities; that they analyzed the epidemiological evidence in a results-oriented manner that is contrary to well-established epidemiological practices; that their biological plausibility opinions are based, at best, on speculative hypotheses; and that they abandoned fundamental scientific principles and methods in the service of advancing plaintiffs' litigation positions. The hearing thus reinforced defendants' *Daubert* briefing, confirming that all of plaintiffs' experts' general causation opinions should be excluded by the Court.

First, the hearing demonstrated that the theory that perineal talc use causes ovarian cancer is not generally accepted. As Dr. Clarke-Pearson acknowledged, no professional organization or regulator – not even Health Canada, on which plaintiffs place such great weight – has concluded that the scientific evidence demonstrates causation. Indeed, the very sources on which plaintiffs' experts rely disclaim a causal finding and/or indicate that further study is required.

Second, the hearing also highlighted that the "methods" plaintiffs' experts

adopted to defy this consensus were unscientific and unreliable. With respect to epidemiology, for example, plaintiffs' experts purported to apply the Bradford Hill criteria but did so in a highly unscientific manner, as the hearing further revealed. Although they described the objectively weak association between perineal talc use and ovarian cancer as "strong" in their reports, the experts essentially abandoned that position at the hearing. Dr. Carson expressly acknowledged that such an association would generally be regarded as "weak" or "modest," while other experts tried to avoid dwelling on strength because they could not credibly say that the association is strong. As to consistency, two of plaintiffs' experts (Drs. Clarke-Pearson and Carson) admitted that they dismissed an entire body of studies in reaching their opinions, while a third (Dr. McTiernan) endorsed a radical change in how epidemiologists assess studies in order to justify her consistency opinion. Plaintiffs' experts' opinions regarding dose response are similarly unscientific; indeed, they had to admit at the hearing that the very studies on which they rely did not conclude that a dose response has been identified.

Third, the hearing also highlighted that plaintiffs' experts' biological plausibility theories are speculative and unsupported. Dr. Longo's testimony, offered by plaintiffs to support their theory that the presence of asbestos demonstrates biological plausibility, confirmed that his methodology for supposedly detecting asbestos is unfixed, results-oriented and non-replicable. Dr.

Longo also admitted that he did not undertake any exposure analysis, a gaping hole in his analysis given defense toxicology expert Dr. Moore's showing that exposure to concentrations of asbestos that Dr. Longo claimed to find in talc would be exponentially lower than OSHA's permissible exposure limit ("PEL").

Plaintiffs' other biological plausibility theories did not fare any better. Dr. Carson's testimony regarding heavy metals made clear that plaintiffs have nothing to support general causation beyond the speculative theory that, at some unspecified dose, some metals might cause ***other*** cancers. Dr. Saed's testimony, intended to support the theory that talc itself is genotoxic, confirmed that his study did not support his opinions, that he was at best neglectful in conducting his experiment and, at worst, that he made up his results in an attempt to fulfill plaintiffs' litigation needs. And the hearing testimony also exposed numerous holes in plaintiffs' inflammation theory, including the lack of evidence that talc reaches a woman's ovaries, that it causes chronic inflammation there or even that chronic inflammation causes ovarian cancer.

Fourth, the hearing also confirmed that plaintiffs' experts have taken positions in the courtroom that they have not taken in the laboratory or clinic. For example, Dr. Clarke-Pearson admitted that he does not tell his patients about the supposed talc-ovarian cancer link, and Dr. McTiernan (who testified in Congress, flanked by plaintiffs' lawyers) conceded that her views about the hierarchy of

evidence expressed in this litigation are different from those she took as a panelist at the World Cancer Research Fund ("WCRF").

In short, the hearing confirmed that plaintiffs' experts have not approached the issues in this litigation with the rigor that behooves disinterested scientists.

ARGUMENT

I. THE HEARING CONFIRMED THAT PLAINTIFFS' EXPERTS' OPINIONS LACK GENERAL ACCEPTANCE IN THE SCIENTIFIC COMMUNITY.

It is widely recognized that expert opinions that conflict with conclusions reached by public health authorities should be viewed with particular skepticism. *See, e.g.*, *McMunn v. Babcock & Wilcox Power Generation Grp., Inc.*, No. 10-143 et al., 2013 WL 3487560, at *22 (W.D. Pa. July 12, 2013) (excluding opinion of general causation expert that was contrary to conclusions reached by public health organizations); *Miller v. Pfizer, Inc.*, 196 F. Supp. 2d 1062, 1067, 1085 (D. Kan. 2002) (excluding expert whose opinion that Zoloft could cause suicide conflicted with the scientific consensus, including the American College of Neuropsychopharmacology, the U.S. Food and Drug Administration ("FDA"), and the Medicines Control Agency).[1] Here, the testimony adduced at the *Daubert*

[1] (*See also* Defs.' GC Mot. at 108-09 (ECF No. 9736)); *In re Mirena IUS Levonorgestrel-Related Prods. Liab. Litig. (No. II)*, 387 F. Supp. 3d 323 (S.D.N.Y. 2019) (granting summary judgment and noting that experts had been excluded where they sought to advance a general causation opinion that no research outside litigation had reached).

hearing demonstrated that plaintiffs' experts' opinions contradict the scientific consensus that there is "inadequate," "limited" or inconclusive evidence of a causal relationship between perineal talc use and ovarian cancer. As Dr. Clarke-Pearson conceded, ***none*** of the relevant professional organizations in his field – including the Society of Gynecologic Oncology ("SGO"), American College of Obstetricians and Gynecologists ("ACOG"), Centers for Disease Control and Prevention ("CDC"), National Cancer Institute ("NCI") and the FDA – has concluded that perineal talc use causes ovarian cancer.[2] And Dr. Clarke-Pearson also conceded that the medical community has not reached any consensus on whether perineal talc use causes ovarian cancer.[3]

Plaintiffs' experts also admitted that their opinions contradict IARC's conclusions on a number of critical points. Most notably, plaintiffs' experts conceded at the hearing that IARC has concluded that there is only "***limited evidence*** in humans for the carcinogenicity of perineal use of talc based body powder."[4] Further, while plaintiffs' experts base their epidemiology opinions

---

[2] (*See, e.g.*, 7/30/19 Hr'g Tr. 1586:23-1589:22, 1604:1-1601:11 (discussing SGO); *id.* 1599:4-1602:5 (discussing ACOG); *id.* 1607:5-10 (discussing CDC); *id.* 1610:3-1611:6 (discussing NCI); *id.* 1620:3-8 (discussing FDA).)

[3] (*Id.* 1691:20-23 ("Q. The gynecologic medical community has not reached a consensus on the opinions that you have given to this Court today. Correct? A. That's right.").)

[4] IARC 2010 Monograph (Tersigni Cert. Ex. A72) (emphasis added); *see also* *(cont'd)*

almost entirely on the results of certain case-control studies, Drs. McTiernan and Carson admitted on cross-examination that "[t]he [IARC] Working Group believed that recall bias was a possibility inherent in the case-control studies and could not be ruled out,"[5] and that IARC recognized that "[it]t is possible that confounding by unrecognized risk factors may have distorted the results" of the case-control studies and could have "induce[d] the appearance of an association between the use of talc and ovarian cancer where there is none."[6]

Plaintiffs' experts were also forced to concede that their migration theory – i.e., that externally-applied talc migrates up through the reproductive tract to the fallopian tubes and ovaries – stands in contrast to IARC's conclusions that "the evidence for retrograde transport of talc to the ovaries in normal women is weak"[7] and that "[s]tudies in animals (rodents, langomorphs and non-human primates)

(cont'd from previous page)
7/25/19 Hr'g Tr. 916:13-917:4 (Dr. McTiernan acknowledging this conclusion); 7/29/19 Hr'g Tr. 1434:1-18 (Carson acknowledging same).

[5] IARC 2010 Monograph at 409. (*See also* 7/25/19 Hr'g Tr. 917:8-10 (Dr. McTiernan acknowledging that IARC could not rule out bias); 7/29/19 Hr'g Tr. 1435:7-10 (Dr. Carson acknowledging same).)

[6] IARC 2010 Monograph at 408. (*See also* 7/25/19 Hr'g Tr. 917:14-16 (Dr. McTiernan acknowledging that IARC could not rule out confounding); 7/29/19 Hr'g Tr. 1435:7-10 (Dr. Carson acknowledging same).)

[7] IARC 2010 Monograph at 411. (*See* 7/30/19 Hr'g Tr. 1665:8-15 (Dr. Clarke-Pearson acknowledging that the evidence was weak).)

showed no evidence of retrograde transport of talc to the ovaries."[8] In addition, Dr. Clarke-Pearson acknowledged that IARC considered many of the very same studies on which he relied in forming his migration opinions but found that because those studies were "conducted in women who were about to undergo gynecological surgery, most of whom had diseases or complications of the reproductive tract and organs that required surgery," their results may be "confounded by the . . . underlying pathologies."[9]

At the hearing, plaintiffs' experts attempted to downplay IARC's conclusions as outdated because they were based on the science as it stood in 2006.[10] But, as plaintiffs' experts were forced to admit, a number of the cohort studies showing no risk associated with talc use (Houghton, Gonzalez, Gates) were published ***after*** IARC reached its conclusions.[11] Indeed, the wider body of evidence that has emerged since the IARC monograph was written strengthens IARC's conclusion that a causal link has not been shown, contrary to plaintiffs'

---

8 IARC 2010 Monograph at 411.

9 (7/30/19 Hr'g Tr. 1661:14-1662:20.) Dr. Clarke-Pearson also agreed with the statement that "[n]one of the articles that you cite in support of your opinion regarding migration looked at whether talc can migrate from perineal application through the reproductive organs to the ovaries." (*Id.* 1665:16-1666:25.)

10 (*E.g.*, *id.* 1656:14-17 ("Q. You know IARC has not concluded that talc causes ovarian cancer. Correct? A. This is a 2010 publication based on data that preceded the working group coming to this conclusion.").)

11 (*See id.* 1657:8-1660:16 (Dr. Clarke-Pearson discussing the cohort studies).)

experts' suggestions.[12] Perhaps for this reason, Dr. McTiernan was forced to concede that any suggestion that IARC would be likely to reach a different conclusion today would be "speculati[ve]."[13]

Given the lack of scientific support for their opinions, it is not surprising that plaintiffs' experts' causation opinions are contradicted by the very studies and publications on which they rely, which is a significant red flag, as this Court has recognized elsewhere. *See Schepise v. Saturn Corp.*, No. CIV.A. 94-385 (MLP), 1997 WL 897676, at *17 (D.N.J. July 30, 1997) (Wolfson, J.) ("[R]eliance upon medical literature for conclusions not drawn therein is not an accepted scientific methodology.") (citation omitted). Even the Health Canada Draft Screening Assessment, on which plaintiffs and their experts have placed heavy emphasis,[14]

---

[12] As Dr. Ballman explained in her report, the cohort studies consist of "the most compelling new information provided by epidemiology studies since the IARC report," and these studies "confirm" what IARC had found – namely, "the lack of a statistically significant association between perineal/genital talcum powder use and the lack of a dose-response relationship." (Ballman Rep. at 44 (Tersigni Cert. Ex. C25).)

[13] (7/25/19 Hr'g Tr. 919:3-6.)

[14] (*See, e.g.*, *id.* 800:7-15 (Dr. McTiernan noting that she considered Health Canada's draft assessment in forming her opinion); *id.* 923:5-16 (Dr. McTiernan admitting that she focused on Health Canada draft assessment over FDA's findings); *id.* 935:21-24 (Dr. McTiernan testifying that her opinion is consistent with Health Canada's); *see also* 7/29/19 Hr'g Tr. 1281:24-1282:14 (Dr. Carson citing Health Canada draft assessment to support the notion that "transport from the perineum to the ovaries does occur"); 7/29/19 Hr'g Tr. 1316:1-8 (Dr. Carson stating that Health Canada "determined . . . that available data are indicative of a

(cont'd)

stops significantly short of concluding that there is a causal link between talc use and ovarian cancer. Among other things, Health Canada's Draft Screening Assessment expressly states that:

- "There is ***a lack of an available exposure-effect relationship in the human epidemiological data***. Many of the studies only assessed a single-dose level (ever versus never users). Furthermore, data with respect to the types of powder used by subjects or the amounts applied were not presented, and therefore a relationship between the concentration/dose of talc in the powder and the incidence of ovarian cancer could not be investigated;"[15]

- "There are limitations with the human epidemiological data. Potential sources of bias include selection bias due to low response rates or from limiting subjects, and exposure misclassification due to recall bias;"[16] and

- "[T]he specific mechanism(s) and cascade of molecular events by which talc might cause ovarian cancer ***have not been identified.***"[17]

In addition, Dr. McTiernan conceded that the Taher meta-analysis that is prominently cited in the Draft Screening Assessment did ***not*** conclude that a causal link has been established between talc and ovarian cancer, but only that "currently

---

(cont'd from previous page)
causal effect"); 7/29/19 Hr'g Tr. 1427:15-18 (Dr. Carson testifying that "the Health Canada report is clearly more current and more inclusive than any of the others that have been published thus far, and for that reason I gave it stronger consideration when I finally received it").)

[15] Draft Assessment at 2 (Tersigni Cert. Ex. A58) (emphasis added). (*See also* 7/29/19 Hr'g Tr. 1727:8-15 (Dr. Clarke-Pearson agreeing that Health Canada noted the lack of data supporting an exposure effect).)

[16] Draft Assessment at 28.

[17] Draft Assessment at 21.

available relevant data indicates that perineal exposure to talc powder is a ***possible*** cause of ovarian cancer in humans."[18] Notably, a PowerPoint presentation created by the authors of the Taher meta-analysis, and supplied to plaintiffs' counsel by one of their experts, concluded that the evidence shows only that talc would qualify as a "Group IIIA" agent under Health Canada's classification system – i.e., "Possibly Carcinogenic to Humans" – because, while epidemiological data "indicate an association between exposure and human cancer, . . . ***alternative explanations such as chance, bias or confounding cannot be excluded***."[19] The PowerPoint presentation, which plaintiffs' experts had not seen before the

---

[18] (7/25/19 Hr'g Tr. 956:23-957:7 (emphasis added).) In addition, the Taher meta-analysis identifies a number of biases inherent in the case-control studies that could have distorted study results and acknowledges that the relevant evidence "indicates that ***talc is not genotoxic***." Taher 2018 at 41-42 (Tersigni Cert. Ex. A137). The Taher meta-analysis, which has now been published in a European journal, includes additional language stating that "perineal application of talc may be a risk factor for ovarian cancer in some population subgroups," and does not in any way suggest that causation has been established. Notably, the published version also states that "the certainty of the evidence" underlying the meta-analysis "was classified as very low," "mainly due to the potential for recall bias in the included case control studies and the relatively short follow-up periods between exposure and outcome assessment in the included cohort studies." Taher et al., *Critical Review of the Association Between Perineal Use of Talc Powder and Risk of Ovarian Cancer*, 90 Reprod. Toxicol. 88, 98-99 & fig. 4 (2019) (attached as Ex. 1 to Cert. of Jessica L. Brennan ("Brennan Cert.").

[19] (Krewski PPT at 30 (Brennan Cert. Ex. 2) (emphasis added); 7/29/19 Hr'g Tr. 1435:11-17.)

hearing,[20] also states that the "specific mechanism(s) and cascade of molecular events by which talc might cause ovarian cancer have not yet been elucidated."[21]

Plaintiffs' experts also admitted under cross-examination that many of the other materials they rely on actually contradict their conclusions. For example:

Berge 2018. Dr. Clarke-Pearson cited the Berge meta-analysis as establishing a statistically significant increased risk of ovarian cancer from talc use.[22] Similarly, Dr. McTiernan testified that Berge is an "excellent" meta-analysis that supports her conclusion that the epidemiology is highly consistent.[23] But as these experts had to concede on cross-examination, the Berge study expressly states that "***the evidence is not consistent***" because "[s]everal aspects of our results, including the heterogeneity of results between case-control and cohort studies . . . ***do not support a causal interpretation of the association***."[24] In

---

[20] (7/30/19 Hr'g Tr. 1731:20-1733:7 (Dr. Clarke-Pearson testifying that he had not been made aware of and had not considered the PowerPoint presentation prior to giving testimony at the hearing).)

[21] (Krewski PPT at 17; *see also* 7/29/19 Hr'g Tr. 1435:11-1436:15 (Dr. Carson admitting that the PowerPoint presentation suggested a designation of talc as only "possibly carcinogenic" because "'alternative explanations such as chance, bias, or confounding cannot be excluded'") (citation omitted).)

[22] (7/30/19 Hr'g Tr. 1537:3-1538:1.)

[23] (7/25/19 Hr'g Tr. 830:3-12.)

[24] Berge 2018 at 248, 256 (Tersigni Cert. Ex. A11) (emphases added). (*See also* 7/30/19 Hr'g Tr. 1687:5-7 (Dr. Clarke-Pearson agreeing that the "authors of Berge [2018] do not state that talc causes ovarian cancer"); *id.* 1683:8-13 (conceding Huncharek 2003 "do[es] not support a causal relationship"); *id.*

(cont'd)

addition, Berge also notes that the association reported may "be attributed to bias" in the case-control studies and concludes that the "biological basis and plausibility of a possible carcinogenic effect of talc on the ovaries ***is still not understood and remains questionable***."[25]

Penninkilampi 2018. Dr. Clarke-Pearson testified at the hearing that the 2018 Penninkilampi meta-analysis supports both his conclusion that there is a causal connection between talc use and ovarian cancer, as well as the theory that talc initiates cancer by triggering inflammation in ovarian tissue.[26] On cross-examination, however, Dr. Clarke-Pearson conceded that Penninkilampi explicitly states that a causal link has ***not*** been established.[27] Dr. Clarke-Pearson also had to acknowledge that Penninkilampi does not provide any actual scientific evidence to support his inflammation theory, but instead merely posits that inflammation ***could*** be a mechanism by which talc could cause ovarian cancer.[28] In fact, Penninkilampi expressly concludes that the "potential mechanism by which genital

---

(cont'd from previous page)

1685:6-8 (conceding that "[t]he authors of [Gross and Berg 1995] did not conclude that talc causes ovarian cancer"); *id.* 1690:3-6 (conceding that "[t]he authors of [Langseth 2008] did not conclude . . . that talc causes ovarian cancer").)

25 Berge 2018 at 255 (emphasis added).

26 (7/30/19 Hr'g Tr. 1540:5-18, 1644:20-25.)

27 (*Id.* 1644:11-1645:13.)

28 (*Id.* 1643:1-5.)

talc is associated with an increased risk of ovarian cancer . . . ***remains unclear***."[29]

Rothman 2000. At the hearing, Dr. McTiernan relied on statements by Dr. Kenneth Rothman (discussed further in Section II.B, below) to the effect that the statistical significance of epidemiological studies should not be taken into account when considering whether their results are consistent.[30] But at the same time, Dr. McTiernan ignored Dr. Rothman's conclusion in 2000 that the "evidence to date does not indicate that talc can be 'reasonably anticipated to be a human carcinogen,'" given that: (1) the "weak positive association observed" in the literature could be the result of bias or confounding, and (2) there is a "lack of a plausible biologic mechanism."[31] As Dr. Diette noted at the hearing, Dr. Rothman has also pointed out that some of the epidemiological data regarding talc and ovarian cancer has identified an "inverse[]" dose response, "meaning the more [talc] you used," the more the product would be "protective from ovarian cancer."[32]

Merritt 2007. Dr. Clarke-Pearson testified at the hearing that Merritt

---

29 Penninkilampi 2018 at 45 (Tersigni Cert. Ex. A109) (emphasis added); *see also id.* at 48 (concluding that there is "need for further research on a potential mechanism by which ovarian cancer may be caused by talc, as this will allow a causal relationship to be established or rejected with more certainty").

30 (7/25/19 Hr'g Tr. 935:6-20.)

31 Rothman 2000 at 1, 8 (Tersigni Cert. Ex. A126).

32 (7/26/19 Hr'g Tr. 1193:4-21.)

supports the proposition that inflammation causes ovarian cancer, claiming that the paper shows that "the use of [the anti-inflammatory] aspirin on a continuous daily basis does reduce the incidence of ovarian cancer."[33] But Dr. Clarke-Pearson ultimately had to acknowledge that Merritt found that there was "[n]o overall association [between] ovarian cancer risk" and NSAID use, and that the ultimate conclusion of the paper was that "chronic inflammation ***does not*** play a major role in the development of ovarian cancer."[34]

Terry 2013. Dr. Carson cited Terry at the hearing as evidence of a "strong" and "compelling" association between talc use and ovarian cancer, and both Drs. McTiernan and Clarke-Pearson asserted that Terry demonstrates a dose-response relationship between the two.[35] But the Terry article does not support either of these propositions. Instead, as Dr. Carson admitted on cross-examination, Terry describes the reported association between talc and ovarian cancer as "modest."[36] In addition, Dr. McTiernan conceded that Terry found no dose-response relationship among users of talcum powder[37] – and Dr. Clarke-Pearson

---

33 (7/30/19 Hr'g Tr. 1639:7-11.)

34 (*Id*. 1631:8-18, 1634:10-23 (emphasis added) (citations omitted).)

35 (7/29/19 Hr'g Tr. 1411:18-1412:4; 7/25/19 Hr'g Tr. 791:2-794:1; 7/30/19 Hr'g Tr. 1613:23-1614:5.)

36 (7/29/19 Hr'g Tr. 1412:8-19.)

37 (7/25/19 Hr'g Tr. 888:8-889:23.)

acknowledged that government scientists have described Terry as demonstrating that "the trend across increasing lifetime number of applications was not statistically significant."[38]

In short, the testimony at the hearing demonstrated that plaintiffs' experts' causation opinions are belied by the very evidence cited to support them. This alone requires exclusion of their opinions.

II. THE HEARING HIGHLIGHTED THE UNRELIABILITY OF PLAINTIFFS' EXPERTS' BRADFORD HILL ANALYSES.

A. The Hearing Confirmed That There Is No Reliable Basis For Plaintiffs' Experts' Opinions Concerning Strength Of Association.

Plaintiffs' experts' testimony at the hearing essentially abandoned their position that Bradford Hill's "strength" consideration is satisfied by the talc-ovarian cancer epidemiological literature.

In his report and direct testimony at the hearing, Dr. Carson asserted that the "epidemiological studies support a strong association between the perineal use of talcum powder and ovarian cancer."[39] But these opinions unraveled during cross-examination, when Dr. Carson was forced to acknowledge that the studies he relied on showed only a 30 percent increased risk of ovarian cancer, which he agreed

---

38 (7/30/19 Hr'g Tr. 1613:3-1616:3 (quoting NCI PDQ (Tersigni Cert. Ex. A104)).)

39 (Carson Rep. at 9 (Tersigni Cert. Ex. C9); 7/29/19 Hr'g Tr. 1311:7-10 (finding "compelling" evidence of a strong association).)

would be regarded as a "weak or modest" association by epidemiologists.[40] Although Dr. Carson attempted to justify his use of the word "strong" based on the "translation of that increased risk to 3,000 ovarian cancer deaths in the United States every year," he admitted that such a conclusion is circular because it assumes that perineal talc use does in fact cause ovarian cancer.[41]

Dr. Clarke-Pearson could not even bring himself to claim that the association was strong on direct examination, instead testifying that there was "overwhelming support in the epidemiologic literature that talcum powder statistically increased a woman's risk of developing epithelial ovarian cancer by about 30 percent."[42] Of course, even if there were "overwhelming support" for a

---

[40] (7/29/19 Hr'g Tr. 1406:9-18.) Moreover, Dr. Carson was forced to concede that the very studies he cited recognize that the association is not strong. (*See id.* 1412:15-16 (acknowledging that Terry 2013 called the association "modest"); *id.* 1412:17-19 (Q. "They didn't call it strong or compelling. Correct?" A. "Not in this case, that's correct."); *id.* 1412:20-1413:6 (conceding that Berge 2017 called the association "weak").) Likewise, Dr. Carson acknowledged that IARC regarded the association reported by the case-control studies as "modest" and potentially the result of bias and confounding. (*Id.* 1434:23-1435:6.)

[41] (*Id.* 1408:13-16 ("It does rely on that assumption.").) Several of plaintiffs' experts who did not testify at the hearing similarly opined that this Bradford Hill factor was satisfied by re-defining "strength" in a similar manner. (*See, e.g.*, Smith-Bindman Rep. at 36-38 (Tersigni Cert. Ex. C36) (concluding strength of association is met because of a "large number of cancers"); Smith Rep. at 19 (Tersigni Cert. Ex. C16) (arguing strength of association is met for anything that "increases the risk of ovarian cancer by ANY consistent percentage" because ovarian cancer is "not a trivial occurrence").)

[42] (7/30/19 Hr'g Tr. 1542:9-14.)

weak association (which is not the case here), that would not magically make an otherwise weak association strong. Dr. Clarke-Pearson's effort to back away from his prior claims that the association is strong[43] was not surprising given that the association is "objectively weak in certain of the studies and non-existent in others,"[44] as Dr. Diette explained at the hearing.

Finally, the testimony at the *Daubert* hearing further discredited the theory expressed by several of plaintiffs' experts that strength of association is not important to the analysis because other objectively weak associations – such as those between hormone replacement therapy ("HRT") and breast cancer or secondhand smoke and lung cancer – are nevertheless considered causal. As Dr. McTiernan admitted, this case is "very different" from the reported association between HRT and breast cancer because the risk estimates for that association were based on randomized control trials – the gold standard for epidemiology – and prospective cohort studies that found a consistent association,[45] virtually

---

43 (*See* Clarke-Pearson Rep. at 8 (Tersigni Cert. Ex. C14) (asserting that "[s]trength and consistency" considerations are "supported by overwhelming epidemiologic evidence") (emphasis omitted).)

44 (7/26/19 Hr'g Tr. 1027:15-21; *see also id.* 1035:20-1036:15 (testifying that "you have to conclude that [the strength of association] is weak" and that relative risks close to 1 are "remarkably low").)

45 (7/25/19 Hr'g Tr. 909:6-911:23.)

eliminating concerns about recall bias and confounding.[46] And Dr. Diette explained at the hearing that in the case of both HRT and secondhand smoke, researchers were able to conclude that a low relative risk could nonetheless support a finding of a causal association because the other Bradford Hill factors were plainly satisfied, which is not the case here.[47]

In short, the hearing confirmed that plaintiffs' experts' opinions regarding strength of association are unreliable because they mischaracterize the objective magnitude of the association reported in the studies and rest on inapposite comparisons to established causal relationships.[48]

B. Plaintiffs' Experts' Testimony At The Hearing Confirmed That Their Opinions Regarding Consistency Are Unreliable.

The hearing also highlighted the unreliability of plaintiffs' experts' analyses with respect to the second Bradford Hill criterion: consistency of association.[49]

---

46 (*See* 7/26/19 Hr'g Tr. 1029:13-1032:13.)

47 (*Id.* 1030:20-1031:8.)

48 Notably, several of plaintiffs' experts who did not testify at the trial similarly had to concede at their depositions that the association is not strong. (*See, e.g.*, Kane Dep. 256:24-257:4 (Tersigni Cert. Ex. B45) ("I've seen 'weak' or 'moderate' used to describe a 1.3" risk ratio); *compare* Singh Rep. at 17 (Tersigni Cert. Ex. C40) ("strength of association . . . is significant"), *with* Singh Dep. 140:19-25 (Tersigni Cert. Ex. B47) ("I'm not looking at talc at 1.3 is a strong association.").)

49 (GC Mot. at 47-48 ("Consistency of association means that '[d]ifferent studies that examine the same exposure-disease should yield similar results,' and that an observed association should be 'repeatedly observed by different persons, in different places, circumstances and times.'") (alteration in original) (citations *(cont'd)*

First, plaintiffs' experts could not defend the position that the case-control and cohort studies are consistent with each other. As Dr. McTiernan conceded, there is not a single "prospective cohort study concluding that there was a statistically significant overall association between talc use and ovarian cancer."[50] In addition, Dr. McTiernan also conceded (as noted above) that the Berge 2018 meta-analysis expressly noted an inconsistency between cohort and case-control studies.[51] Specifically, Dr. McTiernan had to admit that Berge, which she referred to as an "excellent" study, concluded that there was a "'heterogeneity of results between the two groups of studies with an association generally detected in case-control studies but not in cohort studies.'"[52]

Dr. Carson admitted at the hearing that he simply ignored the cohort studies in concluding that consistency was satisfied,[53] testifying that he failed to include or

(cont'd from previous page)
omitted).)

50 (7/25/19 Hr'g Tr. 851:11-15.)

51 (*Id*. 828:22-829:6 (agreeing that the Berge abstract states that "[s]ome [epidemiological] studies suggest an association between genital use of talc powder and increased risk of ovarian cancer, but the evidence is not consistent") (citation omitted).)

52 (*Id*. 958:13-959:2 (citation omitted).) Dr. McTiernan similarly conceded that Penninkilampi 2018 concluded that the relevant epidemiological studies were inconsistent, with all of the cohort studies demonstrating the "possibility that there is in fact no association at all between genital talc use and ovarian cancer." (*Id.* 852:19-853:22.)

53 (*See* 7/29/19 Hr'g Tr. 1417:21-1418:17 (Dr. Carson acknowledging that he

(cont'd)

analyze the cohort studies because they did not "contribute[] to the opinions that [he] expressed,"[54] an apparent admission of improper cherry-picking. *See In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig.*, 26 F. Supp. 3d 449, 461-62 (E.D. Pa. 2014) (excluding expert whose "conclusions are drawn from trends she observed in a self-selected subset of supportive studies, not the totality of the epidemiological evidence"). Similarly, Dr. Clarke-Pearson acknowledged at the hearing that he did not mention, much less analyze, the cohort studies in his expert report.[55] Dr. Clarke-Pearson's purported justification for failing to include the cohort studies in his causation analysis was that he did not "consider [the cohort studies] useful in terms of going to the totality" of the evidence, either as "individual cohort studies or as a body of evidence."[56] The notion that an entire body of studies did not "contribute" to Dr. Clarke-Pearson's analysis and conclusions[57] highlights the unscientific nature of his efforts.

Plaintiffs' experts' related efforts to discredit the cohort studies as not being

(cont'd from previous page)
did not "analyze any of the cohort studies" in his report or identify any of them "by name in the body of [his] report"; or list any of them as references because he "did not feel they contained information that was necessary to cite").)

54 (*Id*. 1418:18-25.)

55 (7/30/19 Hr'g Tr. 1677:20-22.)

56 (*Id*. 1678:5-8.)

57 (*Id*. 1677:20-1680:7.) Several plaintiffs' experts who did not appear at the hearing likewise ignored the cohort studies in opining on consistency. (*See, e.g.*, Smith Rep. at 16; Wolf Rep. at 8, 15 (Tersigni Cert. Ex. C23).)

sufficiently powered were also refuted at the hearing.[58] For example, Dr. Carson admitted at the hearing that he did not perform any calculations related to the power of the relevant cohort studies that could possibly support his opinion that those studies were not sufficiently powered.[59] Instead, Dr. Carson merely assumed that this was the case because he believes that the "design of cohort studies . . . tend[s] to go toward the many cohort studies being underpowered."[60]

Dr. McTiernan disputed that the alleged power issues in cohort studies could be overcome by combining the data from the relevant studies in a meta-analysis,[61] but she made a number of concessions at the hearing that undermined that position. Most notably, Dr. McTiernan was forced to acknowledge on cross-examination that she had, in previous testimony, agreed with the proposition that "meta-analyses, with their larger combined sample sizes, can be used to overcome [a] lack of statistical power."[62] In addition, Dr. McTiernan admitted that the "excellent" Berge 2018 meta-analysis concluded that "the statistical power of the

---

58 (GC Mot. at 50-53.)

59 (7/29/19 Hr'g Tr. 1424:3-8 ("I did not determine the power of the cohort studies specifically.").)

60 (*Id.*; *see also id.* 1424:9-18 (Dr. Carson agreeing that his only analysis of the power of the cohort studies was to "read the studies, look[] at their conclusions, and" determine that "their conclusions were not that the effect didn't exist, but that they couldn't detect it").)

61 (*See* 7/25/19 Hr'g Tr. 861:6-14.)

62 (*Id*. 861:21-862:1.)

meta-analysis of the[] cohort studies to detect an RR of 1.25 similar to the result of the meta-analysis of case-control studies was 0.99" and thus, the allegedly low power of the cohort studies "cannot be invoked as [an] explanation of the heterogeneity of results."[63]

In short, the hearing confirmed that plaintiffs' experts' willingness to dismiss an entire body of studies could not be scientifically supported.

Second, plaintiffs' experts were forced to admit that the condom and diaphragm studies that find no association with ovarian cancer are inconsistent with the case-control studies involving perineal talc use that do find such an association. Indeed, Dr. McTiernan admitted that the very studies on which she and other plaintiffs' experts rely – including Berge 2018 and Penninkilampi 2018 – identify an odds ratio of ***less than*** 1.0 for ovarian cancer and talc-dusted diaphragm use.[64] As Dr. Diette explained, this reported negative association between the use of talc-dusted diaphragms and condoms is a significant inconsistency in the

---

63 (*Id*. 862:10-21 (citation omitted).) Several of plaintiffs' other experts likewise did not dispute Berge's calculation regarding the power of cohort studies when pressed at their depositions. (*See, e.g.*, Moorman Dep. 215:17-23 (Tersigni Cert. Ex. B39) ("I don't disagree with that"); Wolf Dep. 261:23-262:9 (Tersigni Cert. Ex. B30) (admitting that she had no basis to disagree with Berge).)

64 (*See* 7/25/19 Hr'g Tr. 907:11-908:19.) The negative association in Berge was statistically significant, which, according to Dr. McTiernan, indicates a protective effect. (*Id.* 906:9-907:25.)

epidemiological literature.[65] Dr. Saenz testified similarly, noting that the negative association reported in diaphragm studies "is a perfect example of" an inconsistency in the science.[66] In her words:

> Conceptually it makes no sense that putting talc on your diaphragm, which then goes in and sits at the mouth of your cervix, would reduce your risk of ovarian cancer; whereas, placing talc outside on the perineum, a good 7, 8 centimeters away would increase your risk of ovarian cancer. . . . [T]hat's part and parcel of the reason that I do not believe that the literature supports a causal role of talc in the development of ovarian cancer.[67]

Third, the testimony at the hearing demonstrated that even the case-control studies do not consistently show an association between talc use and ovarian cancer. Dr. McTiernan conceded at the hearing that only ***12 of the 24 case-control studies*** cited in the Berge 2018 meta-analysis showed an association between talc exposure and ovarian cancer that was statistically significant.[68] By contrast, the other half of the case-control studies involve confidence intervals that cross 1.0, meaning that the association between talc and ovarian cancer could actually be "null."[69] As discussed extensively in defendants' prior briefing, plaintiffs' experts have brushed aside this inconsistency within the case-control studies by attacking

---

65 (*See* 7/26/19 Hr'g Tr. 1055:8-19.)

66 (7/31/19 Hr'g Tr. 1835:19-22.)

67 (*Id*. 1835:22-1836:7.)

68 (7/25/19 Hr'g Tr. 842:13-845:7.)

69 (*Id*.)

the well-established concept of statistical significance as irrelevant and obsolete, and contending that studies with statistically insignificant results cannot be treated as though they show no association.[70] But the testimony adduced at the hearing made clear that this assault on statistical significance has failed to gain general acceptance in the scientific community.

For example, Dr. Carson admitted at the hearing that Health Canada recognizes the importance of statistical significance by categorizing studies with confidence intervals that cross 1.0 as showing "[n]o association" between exposure and disease.[71] Dr. Carson also conceded that it was "not improper" for Health Canada to report non-statistically-significant results as demonstrating "no association" under applicable scientific principles.[72] Dr. McTiernan similarly acknowledged that studies with non-statistically-significant results – i.e. confidence intervals crossing 1.0 – suggest that there could be a "null association" between an exposure and disease:

> Q. If the confidence interval, the range, includes 1.0, that is, it goes above and below 1.0, you cannot say that there is an association. Right?
>
> A. What we can say is that the association between exposure and disease could be null. So it could be. That's exactly what I

---

70 (GC Mot. at 61-66.)

71 (7/29/19 Hr'g Tr. 1430:19-22; *see also* Draft Assessment tbl. 6-1.)

72 (7/29/19 Hr'g Tr. 1430:23-25.)

said here, that it could be null.[73]

As Dr. McTiernan also testified, it is her "understanding" that the confidence interval in a study demonstrates "what the true relative risk might be," and a confidence interval that crosses 1.0 therefore indicates that a risk may not exist.[74] Dr. McTiernan thus conceded that she could not rule out the possibility that study results that were not statistically significant showed no risk.[75]

Finally, the testimony at the hearing confirmed that calls to "retire" statistical significance by certain epidemiologists, including statements by Dr. Rothman and a recent editorial in *Nature* co-authored by one of plaintiffs' retained experts, Sander Greenland, are theoretical and do not reflect the realities of the scientific world. As Dr. Diette put it:

> I can tell you that in the current world I work in of epidemiology and medicine we rely on p-Values, we rely on 95 percent confidence intervals, and I can't send a paper to a publication and say: By the way Sander Greenland said I shouldn't be doing this, so I don't want you to look at this . . . . There could be a better way, but at the moment these are not the rules of the game. These are somebody's opinion about what might happen.[76]

---

73 (7/25/19 Hr'g Tr. 925:2-8; *see also id*. 835:16-836:2.)

74 (*Id.* 841:4-10.)

75 (*Id.* 841:23-842:12.)

76 (7/26/19 Hr'g Tr. 1156:25-1157:22.)

C. The Hearing Underscored The Fact That Plaintiffs' Experts' Dose-Response Opinions Lack A Reliable Basis.

The testimony at the hearing similarly confirmed that plaintiffs' experts' analyses of dose response were unreliable and results-oriented.

As defendants have previously argued, evidence of dose response is integral to an assessment of whether talc use causes ovarian cancer.[77] *See, e.g.*, *Chapman v. Procter & Gamble Distrib., LLC*, 766 F.3d 1296, 1308 (11th Cir. 2014) (a dose response is "indispensable to proving the effect of an ingested substance" and "establish[ing] general causation"); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1242 (11th Cir. 2005) (dose response may be the "single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect") (citation omitted). Dr. Diette confirmed this point, explaining that dose response is an "important criterion" and that one "would expect to see [a dose-response relationship] for an exposure that's causing a disease."[78]

Yet, as plaintiffs' experts acknowledged at the hearing, dose-response data are absent in many studies, and the data that do exist are inconsistent.[79] For

---

77 (GC Mot. at 67-70.)

78 (7/26/19 Hr'g Tr. 1052:21-23, 1053:24-1054:3.)

79 Once again, plaintiffs' other experts made the same acknowledgments in their reports or depositions. (*See* Kane Rep. at 35 (Tersigni Cert. Ex. C38) (evidence of dose response "equivocal"); Smith-Bindman Rep. at 40 (dose-response "results are inconsistent"); Siemiatycki Dep. 123:8-14 (Tersigni Cert. Ex. B29) (any dose-response data "that exists today" "could be a chance finding").)

example, despite the claim in plaintiffs' expert reports that "[m]any of the 28 case control studies found evidence of a dose-response effect,"[80] Dr. Clarke-Pearson testified that the "case-control studies don't really address dose-response" at all.[81] Likewise, although plaintiffs' experts repeatedly touted the Health Canada Draft Screening Assessment at the hearing,[82] that draft assessment concluded that "[t]here is a lack of an available exposure-effect relationship in the human epidemiological data"; that "a relationship between the concentration/dose of talc in the powder and the incidence of ovarian cancer could not be investigated"; and that even among "studies that provided some evidence of increased risk of ovarian cancer with increasing perineal applications of talc," "none demonstrated both a clear dose-response trend and statistical significance."[83] Indeed, Dr. McTiernan was forced to concede at the hearing that the findings as to dose response in the epidemiological literature are at best inconsistent.[84]

---

80 (McTiernan Rep. at 65 (Tersigni Cert. Ex. C7).)

81 (7/30/19 Hr'g Tr. 1538:5-6.)

82 (*See, e.g.*, 7/25/19 Hr'g Tr. 800:7-15; 7/29/19 Hr'g Tr. 1316:1-8; 7/30/19 Hr'g Tr. 1725:1-10.)

83 Draft Assessment at 20-21. (*See also* 7/26/19 Hr'g Tr. 1053:8-20 (noting that the authors of the Health Canada assessment determined that the data did not demonstrate a dose-response relationship).)

84 (7/25/19 Hr'g Tr. 886:16-887:22.) As Dr. Diette explained at the hearing, the dose-response findings in the literature were "highly inconsistent," which is what would be expected "when you don't have a causal relationship." (7/26/19 Hr'g Tr. 1051:4-1054:3.) And while Dr. McTiernan emphasized Penninkilampi's

(cont'd)

Plaintiffs' experts' testimony at the hearing also revealed that the studies on which they rely to support their dose-response conclusions do not actually support those conclusions. For example, in their reports, Drs. McTiernan and Clarke-Pearson both claimed that Terry 2013 demonstrated a clear dose-response trend.[85] At the hearing, however, each acknowledged on cross-examination that the authors of Terry 2013 concluded that there was ***no trend*** in risk with increasing talc use.[86] Likewise, although Dr. McTiernan points to Cramer 1999 as evidence of a dose-response relationship,[87] the authors of that study found a significant trend ***only*** in models that included women who were non-genitally exposed; data limited to women with genital talc exposure showed no dose response.[88]

The hearing thus confirmed that a dose-response relationship has not been established, further demonstrating the unreliability of plaintiffs' experts' opinions.

(cont'd from previous page)

purported findings with respect to dose response (7/25/19 Hr'g Tr. 796:1-23), Penninkilampi 2018 itself cautions that the increased risk it reports for women with more than 3,600 lifetime applications was only "slight[]" and therefore "prone to recall bias," Penninkilampi 2018 at 45, 46.

85 (McTiernan Rep. at 54; Clarke-Pearson Rep. at 9.)

86 (*See, e.g.*, 7/25/19 Hr'g Tr. 888:3-16; 7/30/19 Hr'g Tr. 1615:21-1616:3.)

87 (McTiernan Rep. at 70.)

88 (7/25/19 Hr'g Tr. 897:25-901:20; Cramer 1999 at 353 (Tersigni Cert. Ex. A23).)

D. Plaintiffs' Experts' Minimal Hearing Testimony Regarding The Remaining Bradford-Hill Considerations Confirmed That Their Opinions On These Points Are Also Unreliable.

To the extent plaintiffs' experts addressed the remaining Bradford Hill criteria at the hearing, they did so only in the most perfunctory and conclusory way, further highlighting the unreliable nature of their analyses.[89]

Specificity. Dr. Clarke-Pearson testified that the specificity factor was "satisfied" because the increased risk purportedly associated with talc "is specific for ovarian cancer, not other cancers."[90] But this statement ignores the undisputed fact that ovarian cancer is not a single disease, but instead a number of different diseases, all with different genetic origins, risk factors and treatments.[91] As Dr.

---

89 The experiment and analogy factors were not covered extensively at the hearing. Biological plausibility is addressed separately in Part III.

90 (7/30/19 Hr'g Tr. 1542:16-20; *see also* 7/29/19 Hr'g Tr. 1311:25-1312:6 (Dr. Carson testifying that what "[w]e have shown through research is that [ovarian cancer] occurs more often in women who use talcum powder for hygienic purposes on a regular basis. So based on that information, I believe the specificity consideration is satisfied.").)

91 (*See* GC Mot. at 82-84; *see also* 7/23/19 Hr'g Tr. 290:1-293:16 (Dr. Neel explaining the various different subtypes of ovarian cancer and noting that it is improper to lump them together because they "are caused in different cells of origin by different types of mutations, and, generally, mutagenic agents cause different types of mutational processes"); 7/31/19 Hr'g Tr. 1813:19-1815:19 (Dr. Saenz detailing the varying origins, risk factors and treatments for the different subtypes of ovarian cancer).) Plaintiffs' experts do not dispute this. For instance, Dr. Judith Zelikoff wrote in her report that "[o]varian cancer comprises at least five distinct histological subtypes" (*see* Zelikoff Rep. at 19 (Tersigni Cert. Ex. C24)), though she did not consider them separately for her report (*see* Zelikoff Dep. 193:11-14 (Tersigni Cert. Ex. B31)).

Neel testified, any effort to determine the cause of ovarian cancer that does not differentiate among the various subtypes of the disease should be viewed with "skepticism" in light of these significant differences.[92] *See also Gannon v. United States*, 571 F. Supp. 2d 615, 626 (E.D. Pa. 2007) (specificity criterion not satisfied where exposure was associated with "at least half a dozen different tumors"; "[m]ost agents that cause cancer cause a single form of cancer"), *aff'd*, 292 F. App'x 170 (3d Cir. 2008).

Temporality. Plaintiffs' experts' testimony with respect to temporality was similarly perfunctory and insufficient to sustain their opinions. Dr. Carson, for example, stated only that "temporality is satisfied" because "the exposure must occur before the effect, [and] all of the studies that have looked at this have assessed retrospective talcum powder exposure."[93] Dr. Clarke-Pearson similarly mentioned temporality only once, asserting that it is "satisfied" because "there is a clear latency period sometimes decades from the exposure of the talcum powder in this case to the development of obvious ovarian cancers."[94] But none of plaintiffs' experts addressed the obvious fact that the temporal connection is unremarkable

---

92 (7/23/19 Hr'g Tr. 293:3-16.)

93 (7/29/19 Hr'g Tr. 1312:7-11.)

94 (7/30/19 Hr'g Tr. 1542:21-1543:6.) This latency theory is facially inconsistent with the supposedly speedy mutations Dr. Saed claims to have generated, as discussed further in Section III, below.

given that most ovarian cancers develop later in life and most women who use talcum powder begin doing so by their mid-20s.[95]

Coherence. Plaintiffs' experts were also unable to show that their theory that talc use causes ovarian cancer is coherent with existing scientific knowledge.[96] Indeed, plaintiffs' experts barely touched on coherence at the hearing, instead generally insisting that their causation theory is coherent because it is "drawn from the research that has been considered [and] do[es] not violate any central scientific laws or understanding."[97] This ignores defendants' arguments, set forth in their prior briefing, that plaintiffs' theory is incoherent because, *inter alia*, different subtypes of ovarian cancer have different etiologies; studies that have investigated the use of talcum powder on diaphragms and condoms have found no increased risk of ovarian cancer; and no studies have found an association between perineal talc use and vaginal cancers.[98] For these reasons, too, the hearing testimony confirmed the unreliability of plaintiffs' experts' Bradford Hill analyses.

III. THE HEARING HIGHLIGHTED THE SPECULATIVE NATURE OF PLAINTIFFS' EXPERTS' BIOLOGICAL PLAUSIBILITY OPINIONS.

Plaintiffs' experts have offered three different theories of biological

---

95 (GC Mot. at 93-94.)

96 (*Id*. at 84-88.)

97 (7/29/19 Hr'g Tr. 1312:25-1313:17 (Dr. Carson addressing coherence).)

98 (GC Mot. at 84-88.)

plausibility: (1) that cosmetic talc contains asbestos that causes cancer; (2) that it contains heavy metals that cause cancer; and (3) that talc itself is carcinogenic. The hearing highlighted the unscientific and speculative nature of each theory.

A. Plaintiffs Failed To Present Reliable Evidence That The Products Contain Asbestos At All, Much Less In An Amount Capable Of Causing Ovarian Cancer.

The alleged presence of asbestos in talc does not supply a biologically plausible mechanism through which talc use could cause cancer because: (1) plaintiffs' experts have not reliably shown that the relevant products contain asbestos; and (2) even if plaintiffs' experts' unreliable testing methods were accepted, they would only establish minuscule exposure, far below background levels and regulatory limits and even farther below the heavy occupational exposure that occurred in the studies linking asbestos and ovarian cancer.

1. Dr. Longo's Methodology Cannot Positively Identify Asbestos And Is Not Reproducible.

The *Daubert* hearing confirmed that Dr. Longo – a professional expert[99] – applied a subjective, litigation-driven methodology that was incapable of reliably detecting asbestos in defendants' talc products. Dr. Longo used two microscopy

[99] Dr. Longo "testif[ies] once to twice a week" (95 percent of the time for plaintiffs) in connection with talc cases and has done so "every week for the past five years." (7/24/19 Hr'g Tr. 542:16-21, 544:14-17.) This is a major turnaround for a man who once testified that the notion that cosmetic talc contains asbestos is an "urban legend," a phrase he later tried to disavow by pretending he did not know what it means. (*Id.* 556:15-558:6.)

methods – transmission electron microscopy ("TEM") and polarized light microscopy ("PLM") – both of which were conducted in a profoundly unscientific manner.

TEM. Dr. Longo's hearing testimony made clear that his TEM methodology failed to distinguish asbestos particles (an alleged cause of ovarian cancer) from cleavage fragments (which no scientific literature has linked to ovarian cancer).[100] Although Dr. Longo agreed that cleavage fragments can form in the same dimensions as asbestos fibers,[101] he conceded in response to questioning from the Court that his analysts did not attempt to distinguish between cleavage fragments and asbestos.[102] He also acknowledged his prior testimony that his analysts counted non-asbestiform cleavage fragment as asbestos, relying on the

---

100 Although plaintiffs argued otherwise in their *Daubert* opposition brief (*see* Pls.' Asbestos Opp'n at 40-42 (ECF No. 9892)), Dr. Longo agreed that cleavage fragments are not the same thing as asbestos (*e.g.*, 7/24/19 Hr'g Tr. 569:15-20 (agreeing that "[a] cleavage fragment is not asbestos" and that "[a] cleavage fragment is a crushed up piece of non-asbestiform rock"); *see also* 7/24/19 Hr'g Tr. 571:9-573:4 (agreeing that the EPA, OSHA and ISO definitions of asbestos exclude nonasbestiform minerals)).

101 (*E.g.*, *id.* 574:8-23 (agreeing that "long, thin cleavage fragments can actually resemble asbestos fibers"); *id.* 576:23-577:3 (agreeing that "the ISO standard on which [he] rel[ies]" states "that the crushing of non-asbestiform amphiboles generally yields elongated fragments that [conform] to the definition of a[n] [asbestos] fiber").)

102 (*Id.* 582:9-17 (testifying that his analysts "are not making that decision").)

EPA's "counting rules."[103]

As Dr. Longo agreed, however, the "counting rules" come from the EPA's Asbestos Hazard Emergency Response Act ("AHERA") regulations, which are designed for asbestos remediation in school buildings, where there is no question that the building previously contained asbestos.[104] They are ***not*** designed to distinguish between asbestos and cleavage fragments. Thus, Dr. Longo's application of the counting rules for purposes of detecting "asbestos" renders his opinions speculative and unreliable.[105] *See, e.g., Hanson v. Colgate-Palmolive Co.*, 353 F. Supp. 3d 1273, 1285-86 (S.D. Ga. 2018) (excluding asbestos-contamination opinion where expert's testing methodology was incapable of distinguishing asbestos from non-asbestos particles); *see also Zachary v.*

---

[103] (*Id.* 579:20-580:11; *see also id.* 578:18-579:8, 581:17-582:21 (Dr. Longo's analysts "are not making that decision" as to whether any particle is asbestiform).) Dr. Longo argued at the hearing that "[a]n analyst would not count" a cleavage fragment as asbestos "if he knew somehow" that it was a cleavage fragment from the outset (*id.* 579:9-19), but this is impossible to square with his repeated testimony that the only criterion used to determine whether a particle was asbestos when conducting TEM was application of the counting rules.

[104] (*Id.* 493:16-494:5, 583:9-585:1; *id.* 586:1-15 (agreeing that the AHERA counting rules come into play only after "there has been a determination that the school contained asbestos-containing materials"); *see also* Defs.' Asbestos Mot. at 29-35 (ECF No. 9736-3) (fully explaining the genesis of the AHERA regulations and why they are not applicable here).)

[105] Notably, the ISO protocol Dr. Longo relies on but did not fully follow (as set forth in detail in defendants' prior briefing), "in fact says that it is necessary to discriminate between the asbestiform and non-asbestiform analog of these minerals." (7/24/19 Hr'g Tr. 580:19-23, 581:10-16.)

Bridgestone/Firestone, Inc., No. 1:01-cv-531-GET, 2004 WL 5512956, at *3 (N.D. Ga. Oct. 2, 2004) (excluding expert who performed tests "under conditions that were not designed to approximate those of the accident in this case").[106]

The hearing also exposed the unreliability of Dr. Longo's efforts to overcome his inability to distinguish asbestos from cleavage fragments by claiming that 93 percent of the particles his analysts detected in their MDL testing were "bundles" of asbestos and thus could not have been cleavage fragments.[107] This highly convenient prevalence of "bundles" underscores the unreliable nature of Dr. Longo's opinions for two reasons.

First, it contrasts with Dr. Longo's testing in prior talc litigation, where he

---

[106] Dr. Longo purports to have attempted to confirm the mineral type of the particles detected via TEM through Energy Dispersive X-ray Analysis ("EDXA") and Selected Area Diffraction ("SAED") (*see, e.g.*, 7/24/19 Hr'g Tr. 488:18-499:4), but his analysts did not perform these steps reliably because, *inter alia*, they used subjective visual approximations rather than quantitative data for the former and failed to use multiple zone axis diffraction patterns when conducting the latter (*see* Defs.' Asbestos Mot. at 54-62, 66-72; Defs.' Asbestos Reply at 28-39 (ECF No. 10041)). Dr. Longo argued that the steps he omitted are not required under the EPA's AHERA methodology (*see* 7/24/19 Hr'g Tr. 504:1-508:13), but that methodology should not have been used here, as just explained, *cf. Hanson*, 353 F. Supp. 3d at 1280-81 (explaining how complete EDXA and SAED analyses are both required to prove asbestos detection in litigation).

[107] (7/24/19 Hr'g Tr. 592:22-25.) Dr. Longo has agreed that TEM "cannot tell you if you identify a single fiber whether or not that particle is asbestiform." (*E.g.*, *id.* 588:8-24, 589:10-15.) He believes, however, that "bundles" of fibers are "by definition" asbestos. (*E.g.*, *id.* 589:25-590:5, 594:13-15.) As such, in his view, characterizing particles as "bundles" allows him to avoid distinguishing between asbestos and cleavage fragments.

purported to detect roughly half individual fibers and half bundles.[108] Such a "sudden reversal" alone calls the reliability of Dr. Longo's opinions into serious question. *See Fireman's Fund Ins. Co. v. Canon U.S.A., Inc.*, 394 F.3d 1054, 1059 (8th Cir. 2005) (agreeing with district court's conclusion that a "sudden reversal" of opinion in response to new evidence that drew original opinion into question "seriously undermines the reliability" of the opinion).

Second, Dr. Longo's newfound prevalence of "bundles" is also unreliable because, as the hearing testimony made clear, he and his analysts – to whom Dr. Longo delegated every aspect of the testing – have no objective way of determining whether a particle is a single fiber or a bundle.[109] Dr. Longo's results show that all four of his analysts only agreed ***once*** as to whether a particle was a bundle,[110] and the particles he now calls "bundles" are visually indistinguishable from particles he formerly deemed to be single fibers.[111] Faced with these

---

108 (*Id.* 592:16-19 (53 percent of structures in pre-MDL report were bundles).)

109 Dr. Longo's testimony confirmed that he delegated every aspect of his MDL testing to analysts at his laboratory. (*See, e.g.*, *id.* 540:16-541:9.) Indeed, Dr. Longo has "never personally tested a talc sample for asbestos from start to finish." (*Id.* 540:12-15.)

110 (*See* Defs.' Asbestos Mot. at 46-47.) Dr. Longo claims that overall, his analysts agreed 72 percent of the time as to whether a particle was a single fiber or bundle (7/24/19 Hr'g Tr. 525:22-25), but this falls well below the industry accreditation standard, which requires that analysts agree 90 percent of the time (*see id.* 603:10-15).

111 (7/24/19 Hr'g Ex. 520A (Brennan Cert. Ex. 3) (comparison of images in pre-

(cont'd)

discrepancies, Dr. Longo's response – incredibly – is that "there is no right" answer as to whether or not a particle is a bundle.[112] This "anything goes" philosophy is the antithesis of methodological science and thus cannot satisfy *Daubert*. *See, e.g.*, *In re TMI Litig. Cases Consol. II*, 911 F. Supp. 775, 795-96 (M.D. Pa. 1996) (excluding expert who used subjective techniques that "expose[d] [his] methodology to a potentially high rate of error").

PLM. The hearing also confirmed that Dr. Longo's PLM testing was similarly unreliable. As an initial matter, Dr. Longo has never used PLM in previous talc litigation because he believed that PLM was not an "appropriate" method for testing talc for asbestos.[113] Dr. Longo's about-face – he now believes that PLM is among "the best instruments for [analyzing talc] for asbestos"[114] – is yet another "sudden reversal of opinion" that calls the reliability of his opinions into serious question. *See Fireman's Fund*, 394 F.3d at 1059; *see also, e.g.*, *In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig.*, No. 12-md-2342, 2015 WL

(cont'd from previous page)
MDL and MDL reports); *see also* 7/24/19 Hr'g Tr. 593:7-18, 594:16-595:8 (discussing these and similar images); Defs.' Asbestos Mot. at 45 (comparison showing that Dr. Longo's "bundles" look identical to single fibers identified in previous litigation).)

112 (*See* 7/24/19 Hr'g Tr. 526:2-6 ("The way this is measured is not – does everybody get it right because there is no right."); *see also id.* 603:1-9 (similar).)

113 (7/24/19 Hr'g Tr. 606:13-23.)

114 (*Id.* 475:12-13.)

7776911, at *16 (E.D. Pa. Dec. 2, 2015) ("It is improper for an expert to . . . mold[] his methodology . . . to confirm his preconceived opinion."), *aff'd*, 858 F.3d 787 (3d Cir. 2017).

In any event, the hearing made clear that Dr. Longo's PLM methodology, like his TEM methodology, was replete with similar subjectivity and reproducibility problems. For one thing, Dr. Longo's method for determining how much asbestos was in any PLM sample was to have his analyst visually compare what he saw to internal reference charts.[115] As Dr. Longo conceded at the hearing, these charts were not produced, and neither the employee who designed them nor the analyst who used them was made available to testify.[116] This undisclosed element of his method rendered Dr. Longo's PLM analysis impossible to replicate,[117] a factor that favors exclusion, as this Court has previously recognized. *See, e.g.*, *Bracco Diagnostics, Inc. v. Amersham Health, Inc.*, 627 F. Supp. 2d 384, 446 (D.N.J. 2009) (Wolfson, J.) (striking expert testimony where work product that

---

115 (7/24/19 Hr'g Tr. 609:11-611:20 (confirming that the analyst conducting PLM "compared" what he observed "against a weight percent standard" that Dr. Longo's lab had created).) Notably, the ISO protocol Dr. Longo relied on warns that "the accuracy and reproducibility of [such] visual estimates is very limited" (*id.* 613:17-20) and recommends a different procedure ("point counting"), which Dr. Longo did not use (*id.* 609:11-610:6).

116 (*Id.* 613:17-614:3, 615:16-22 (Dr. Longo stating that he "did not produce that data").)

117 (*Id.* 614:4-615:22.)

was "critical" for verifying accuracy was "never produced").

Even Dr. Longo's own effort to "replicate" his PLM results was an utter failure. As Dr. Longo conceded at the hearing, he sent his samples to a third-party laboratory (J3) for "verification," but that lab did not detect asbestos in ***any*** sample via PLM.[118] Thus, Dr. Longo's only effort to verify his results showed instead that he "could not reproduce his own results . . . using his own method." *In re Diet Drugs*, No. MDL 1203, 2001 WL 454586, at *13 (E.D. Pa. Feb. 1, 2001). Dr. Longo offered several speculative explanations for this discrepancy, including that his analyst spent significantly more time than the analyst at J3 (specifically, that unlike the J3 analyst, his own analyst examined each sample for two-to-six hours).[119] But Dr. Longo's testimony was disproven at the hearing because the reports his analyst generated showed that he analyzed as many as 13 samples in a day, meaning that he would have been working up to 78 hours a day if Dr. Longo's rationale were correct.[120] Moreover, as the Court observed, Dr. Longo has not investigated whether ***any*** of his explanations for the divergence between his and J3's results are correct, despite being aware of the issue since July 2018 and having

---

118 (7/24/19 Hr'g Tr. 618:13-619:8 (confirming that J3 tested 22 samples and did not detect asbestos, while Dr. Longo's lab detected asbestos in eight of the same samples).)

119 (*See, e.g.*, 7/24/19 Hr'g Tr. 623:22-625:6.)

120 (*See id.* 625:8-628:8.)

initially suggested follow-up testing.[121] This further highlights the unreliable and non-replicable nature of Dr. Longo's analysis.

2. Dr. Longo's Testing Is Irrelevant To General Causation Because He Did Not Conduct Any Exposure Analysis.

Dr. Longo's testimony also confirmed that his opinions should be excluded as irrelevant to causation because he did not conduct any sort of exposure analysis, even though his laboratory has that capability.[122] As a result, although Dr. Longo believes that talc users sustain "significant exposure" to asbestos,[123] all he can actually say with respect to exposure is that the amount of asbestos he alleges to have found in defendants' talc is "ultra trace" – akin to "looking for a needle in a haystack"[124] – and "well below" both the one-percent-by-weight threshold the EPA uses to define an "asbestos containing material" and OSHA's permissible exposure limit ("PEL") of one fiber per cc.[125] Nor has Dr. Longo undertaken any effort to

---

121 (*Id.* 629:2-632:6 (the Court observing that Dr. Longo does not "have an answer as to the differences" even though "[i]t has been a year"); *see also id.* 623:3-22, 624:23-625:7 (Dr. Longo called J3 analyst Lee Poye to discuss reasons for divergence and has proposed ways to test hypothesized explanations).)

122 (7/24/19 Hr'g Tr. 561:2-562:24, 565:1-6.) Similarly, Dr. Carson did not attempt to compare the levels of asbestos he believes are in talc to ambient background levels. (7/29/19 Hr'g Tr. 1341:22-1342:2.)

123 (*E.g.*, 7/24/19 Hr'g Tr. 561:2-4.)

124 (*Id.* 560:9-11, 641:21-642:9.)

125 (*Id.* 559:11-560:9, 563:2-20.) Dr. Longo has also previously testified that a person who used a product containing less than one percent asbestos 20 times a day for 40 years would not sustain "'a very large exposure.'" (*Id.* 566:7-567:25.)

refute the exposure analysis that was performed by defendants' expert, Dr. Moore, who demonstrated that, even ignoring the huge flaws in Dr. Longo's detection methods, the amount of asbestos he claims to have found could not pose a risk of cancer.[126] When asked about this analysis at the hearing, Dr. Longo expressed his disagreement with it, but he also claimed that he "ha[s]n't seen [Dr. Moore's] report" in the five months since it has been submitted to plaintiffs and "ha[s]n't done [a] study" to refute her findings.[127]

Dr. Longo's failure to conduct an exposure analysis is highly problematic because it is a fundamental tenet of toxicology that "the dose makes the poison."[128] As such, courts have excluded general causation opinions that "ignore[] the importance of . . . dose [and] duration of exposure." *Mallozzi v. EcoSMART Techs., Inc.*, No. 11-CV-2884 (SJF) (ARL), 2013 WL 2415677, at *8 (E.D.N.Y. May 31, 2013) (excluding a general causation opinion that brief inhalation of a product containing 1% peppermint oil can cause disease because it was premised

---

126 (Defs.' Asbestos Mot. at 85 (citing Moore Rep. at 52-56 (Tersigni Cert. Ex. C19)) (opining that even the highest amounts of asbestos that Dr. Longo claims to have found in the Products would result in cumulative lifetime exposures that are: (1) three times less than those associated with ambient, background exposure; (2) at least 4,000 times below the lifetime asbestos concentration associated with the OSHA PEL; and (3) at least 29,000 times below the level of tremolite asbestos considered to be safe with respect to mesothelioma); Defs.' Asbestos Reply at 47-48 (same).)

127 (*See* 7/24/19 Hr'g Tr. 564:1-25.)

128 Toxicology Reference Manual at 636 (Tersigni Cert. Ex. A46).

on literature studying the effects of topical application of pure peppermint oil); *see also Amorgianos v. Nat'l R.R. Passenger Corp.*, 137 F. Supp. 2d 147, 186-87, 190-91 (E.D.N.Y. 2001) (excluding testimony of plaintiffs' general causation expert in part because the expert extrapolated from materially different exposure scenarios, leaving an "analytical gap" between the literature and his conclusions), *aff'd*, 303 F.3d 256, 270 (2d Cir. 2002). These cases apply in spades here, because the epidemiological studies showing an association between asbestos exposure and ovarian cancer involve long-term exposure to huge quantities of asbestos in industrial settings.[129] There is thus a huge gulf between the relevant science and the opinions of Drs. Longo and Carson, who are essentially offering "any exposure" theories with respect to asbestos.[130] *See, e.g.*, *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671, 679-80 (6th Cir. 2011) ("[I]t is well-settled that the mere existence of a toxin in the environment is insufficient to establish causation

---

[129] *See, e.g.*, IARC 2012 Monograph at 256 (Tersigni Cert. Ex. A70) (noting that the majority of studies regarding asbestos and ovarian cancer involved "heavy occupational exposure"). (*See generally* Defs.' Asbestos Mot. at 84-86; Defs.' Asbestos Reply at 46-48.) In any event, as Dr. Diette explained, the most relevant body of epidemiological literature is the literature regarding talc, because even if talc contains asbestos, any effect of that asbestos would be "part of the epidemiology." (7/26/19 Hr'g Tr. 1189:8-1190:2.)

[130] (*See* 7/24/19 Hr'g Tr. 564:198-25 (Dr. Longo arguing that any asbestos exposure from talc is "additional" to background exposure and thus not irrelevant); 7/29/19 Hr'g Tr. 1339:21-22 (Dr. Carson testifying that "[a]ny amount of asbestos exposure to the ovary has the potential to cause ovarian cancer").)

without proof that the level of exposure could cause the plaintiff's symptoms."); *Boyer v. Weyerhauser Co.*, No. 14-cv-286-wmc et al., 2016 WL 705233, at *22-23 (W.D. Wis. Feb. 19, 2016) (striking asbestos experts who could not show "reliably that non-occupational exposures were comparable to those in the occupational setting, nor that the level of exposure met any scientifically recognized level to contribute substantially to contracting lung cancer").[131]

In short, Dr. Longo's opinions (and the asbestos-related opinions of plaintiffs' experts who piggyback on his report) would be inadmissible even if his testing had been reliably conducted because he does not establish the presence of a dangerous amount of asbestos in talc.

B. Plaintiffs Failed To Present Reliable Evidence That The Heavy Metals Purportedly Present In The Products Are Capable Of Causing Ovarian Cancer.

Several of plaintiffs' experts have also attempted to establish biological plausibility by opining that the Products contain heavy metals (i.e., cobalt, chromium and nickel) that contribute to their purportedly carcinogenic nature. Dr. Carson's hearing testimony underscored that such a position is speculative and unreliable.

First, Dr. Carson's testimony confirmed that there is no scientific evidence

[131] (*See also generally, e.g.*, Defs.' Asbestos Mot. at 83-84, 89-92 (collecting cases rejecting "any exposure" opinions and causation opinions where alleged exposure was not comparable to that in studies).)

linking exposure to the alleged heavy metals with ovarian cancer. In fact, Dr. Carson admitted at the hearing that ***none*** of the heavy metals has been identified as carcinogenic to the ovary by IARC,[132] and that there are no studies showing that exposure to cobalt, chromium or nickel combined with talc increases the risk of ovarian cancer.[133] Dr. Carson nonetheless speculated that, because these metals have been linked to ***other*** kinds of cancers, there is a "***suspicion*** that the same mechanism can operate in other tissue including ovarian epithelial tissue."[134] As Dr. Carson freely acknowledged, however, mere suspicion is not tantamount to a causal connection.[135] And it is axiomatic that "[e]vidence . . . that suggests a connection between . . . exposure and" one type of cancer "is not probative on the causation of" a different form of cancer, *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 197 (5th Cir. 1996), as plaintiffs themselves have recognized in their briefing.[136]

Second, Dr. Carson's hearing testimony further established that plaintiffs' experts' heavy-metals theory of biological plausibility is separately excludable for failure to consider dosage and exposure concentration. As Dr. Carson confirmed at

---

132 (7/29/19 Hr'g Tr. 1359:22-25.)

133 (*Id.* 1360:11-18.)

134 (*Id.* 1439:22-24 (emphasis added).)

135 (*Id.* 1439:1-1440:2.)

136 (Pls.' Bio. Plausib. Opp'n at 50 (ECF No. 9890) (arguing that "different tissues react differently to carcinogens").)

the hearing, he does not know the amount of chromium, cobalt or nickel allegedly contained in the Products.[137] Nor could he point to any other expert who ***has*** such knowledge.[138] As a result, Dr. Carson admittedly did ***not*** conduct a dose-response assessment.[139] Such a failure is especially glaring given his admission at the hearing that the trace metals purportedly present in talc are naturally present in our bodies, in food, in drinking water, in bottled water and in vitamins.[140] Indeed, Dr. Carson could not answer whether the blood or tissue levels of any trace heavy metals are higher in genital talc users as compared to non-talc users.[141] For this reason, too, his opinions are speculative and unreliable.[142]

---

137 (7/29/19 Hr'g Tr. 1362:15-21, 1363:3-7, 1365:7-10.)

138 (*Id.* 1366:5-9.)

139 (*Id.* 1367:6-9.)

140 (*Id.* 1363:20-1364:2.)

141 (*Id.* 1364:18-24.)

142 In his hearing testimony, Dr. Carson briefly addressed the theory that certain fragrances in the Products contribute to their carcinogenic nature, but he stressed that any such contribution "is minor." (*Id.* 1274:4-10; *see also id.* 1306:24-1307:4.) He also testified that his opinions regarding fragrances are reliant on the opinion of another expert, Dr. Michael Crowley (*see id.* 1367:15-19, 1368:20-24), essentially admitting that he engaged in improper parroting. *See Dura Auto. Sys. of Ind., Inc. v. CTS Corp.*, 285 F.3d 609, 614 (7th Cir. 2002) (expert "is not permitted to be the mouthpiece of" another scientist).

C. Plaintiffs' Experts' Hypothesis That Talc Itself Is Carcinogenic Lacks A Legitimate Scientific Basis.

1. Plaintiffs' Only Evidence That Talc Is Genotoxic Is Inherently Unreliable And Inadmissible.

The hearing also confirmed that Dr. Ghassan Saed's methods were unreliable and that his data do not support his conclusion that cosmetic talcum powder is genotoxic and can cause ovarian cancer; indeed, as Dr. Neel testified, Dr. Saed's work does not "even address that question," let alone answer it.[143]

(a) The Hearing Confirmed That Dr. Saed's Methods Were Unreliable.

The hearing first confirmed that Dr. Saed's opinions are inadmissible because: (1) Dr. Saed failed to follow his own methods; (2) he failed to use a relevant dose; (3) he did not run his experiments in triplicate or otherwise replicate them; and (4) his lab notebook was rife with errors.

First, the hearing confirmed that Dr. Saed failed to follow his own methodology. As Dr. Saed admitted at the hearing, he set out to test for cellular transformation using a neoplastic transformation assay – which he thought would be "critical in establishing [a] cause and effect relationship" – but never did so.[144] Dr. Saed also conceded that the cell proliferation test that he did perform cannot show transformation with any degree of "certain[ty]," since proliferation is a

---

143 (7/23/19 Hr'g Tr. 303:5-10.)

144 (*See* 7/22/19 Hr'g Tr. 58:8-12, 64:19-25, 121:2-19, 123:24-124:3.)

common response to foreign bodies among non-cancerous cells.[145] In addition, Dr. Saed acknowledged departing from his own methodology in other ways as well, for instance by failing to perform all of the tests for redox balance outlined in his proposal and failing to perform single nucleotide polymorphism ("SNP") testing for BRCA mutations.[146] Such departures from his own specified methods – including the "critical" neoplastic transformation assay – bespeak a lack of reliability and support exclusion. *See, e.g.*, *Amorgianos*, 303 F.3d at 268 (affirming exclusion where expert "failed to apply his own methodology reliably").

Second, Dr. Saed failed to even ascertain, much less use, a relevant dose, as the hearing further demonstrated. Dr. Saed admitted that he does not "know whether the concentrations [he] used . . . compare to actual human exposure" levels[147] and volunteered that it would be "[v]ery hard to correlate the two."[148] Indeed, even if he wanted to test an appropriate dose, he could not have, since he admitted that he does not know "the dose that women are exposed to in real life."[149] Instead, Dr. Saed simply started with a dose that "kill[ed]" his cells, then

---

145 (*See id.* 189:11-18, 190:6-19, 191:17-20.)

146 (*Id.* 124:6-125:3, 132:4-134:3.)

147 (*Id.* 148:6-10.)

148 (*Id.* 146:11-16.)

149 (*Id.* 147:10-11.)

"tapered . . . down" until he found a "not toxic" dose, and used that.[150] This, too, merits exclusion. *See, e.g., Bourne ex rel. Bourne v. E.I. DuPont de Nemours & Co.*, 189 F. Supp. 2d 482, 498 (S.D. W. Va. 2002) (excluding expert who relied on in vitro testing with "high doses" that were "'far removed' from the plaintiff's alleged exposure").

Third, Dr. Saed failed to adequately replicate his experiments. Although his proposal stated that "[a]ll experiments w[ould] be performed in triplicate," he did not follow that aspect of his proposal either. Dr. Saed explained at the hearing that instead of testing each cell line three times, he used "six different cell lines" and tested each of them once.[151] As the Court pointed out, "[t]hat's not testing in triplicate" because no individual cell line was tested multiple times.[152] This additional failure to follow basic scientific methods further underscores the unreliability of Dr. Saed's methods and opinions. *See, e.g., Rovid v. Graco Children's Prods., Inc.*, No. 17-cv-01506-PJH, 2018 WL 5906075, at *5-6 (N.D. Cal. Nov. 9, 2018) (excluding expert who failed to run "multiple tests" and thus

---

150 (*Id.* 147:1-4, 147:17-148:2.) Dr. Saed also testified that the doses he ultimately used were derived from those "published in the literature that talk about testing talcum powder." (*Id.* 50:5-16.) That is a dubious claim since, as discussed in defendants' reply in support of their motion to exclude Dr. Saed's testimony, Dr. Saed did not cite any of this literature to substantiate the doses he chose in his Proposal. (Saed Reply at 10 n.12 (ECF No. 10040).)

151 (7/22/19 Hr'g Tr. 152:2-5 (citation omitted); *id.*153:5-154:7.)

152 (*Id.* 51:14-18.)

could not "show that his results are reproducible or reliable").

Fourth, the hearing illustrated the shoddiness and unprofessionalism of Dr. Saed's lab notebook, which make it impossible to replicate his work.[153] *See Hanson*, 353 F. Supp. 3d at 1284 (excluding expert in part because "[b]y failing to record" his methodology, he "ensured no other analyst could replicate his work and test his findings").

In addition to all of the previously identified problems in Dr. Saed's lab notebooks, the hearing revealed for the first time a discrepancy in Dr. Saed's proliferation assay results that he could not explain. As explained at the hearing, Dr. Saed's lab notebook sets forth an ***eight***-row "well plate design" for the assay, the seventh and eighth row of which were designated for untreated ovarian cells (wells G1-3) and talc-treated ovarian cells (wells H1-3).[154] Oddly, however, Dr. Saed's raw data – the data he collected and analyzed for his 2019 publication – contained an additional ***ninth*** row, reporting data from wells whose cell line was not identified in the design or explained anywhere in Dr. Saed's lab notebooks (identified in pink):[155]

---

153 (7/23/19 Hr'g Tr. 308:1-20.)

154 (7/22/19 Hr'g Tr. 211:20-212:11.)

155 (*See* SAED000001-97(color) at SAED000087(color) (Tersigni Cert. Ex. B13); 7/22/19 Hr'g Tr. 199:21-203:7, 218:5-221:6.)

96 wells Plate design

	1	2	3	4	5	6	7	8	9	10	11	12
A	A2780 Unt			EL-1Unt								
B	A2780 100ug/ml			EL-1 100ug/ml								
C												
D	SKOV-3 Unt			TOV112 Unt								
E	SKOV-3 100ug/ml			TOV112 100ug/ml								
F												
G	Normal ovarian Unt			FT33 Unt								
H	Normal ovarian 100ug/ml			FT33 100ug/ml								

Well plate design

Raw data

9/6/2018					
1	2	3	4	5	6
0.1764	0.17	0.1767	0.1616	0.15	0.156
0.212	0.223	0.2261	0.2899	0.2873	0.2719
0.1225	0.1248	0.1232	0.192	0.2087	0.1961
0.2198	0.2126	0.2171	0.2604	0.251	0.2598
0.3042	0.3017	0.3269	0.1383	0.1402	0.1437
0.1593	0.1506	0.1598	0.253	0.2643	0.2539
0.1244	0.1202	0.1282	0.151	0.1541	0.15
0.103	0.115	0.112	0.1411	0.1414	0.1408
0.225	0.2248	0.2232	0.192	0.2087	0.1961

Raw Data

At the hearing, Dr. Saed struggled to explain the ninth row of the raw data table, offering various and incomprehensible responses, including the possibility that it might have been derived from "controls," ultimately prompting the Court to remark that "[e]very minute we go on it gets more confusing."[156] This confusion was compounded by a third table in the lab notebook reflecting Dr. Saed's reported data, which presented the data in still a different manner that did not correspond to either the well plate design or the raw data table. Specifically, as noted above, the

156 (*See* 7/22/19 Hr'g Tr. 212:16-217:13.)

well plate design suggested that the seventh and eighth rows of the raw data table should have corresponded to untreated and treated normal ovarian cells, respectively, while Dr. Saed's testimony suggested that the ***ninth*** row contained extra data that could be disregarded. But the reported data table treated the eighth and ninth rows of the raw data table as containing the results for untreated and treated normal ovarian cells, respectively, and disregarded the data in the ***seventh*** row of the raw data table:

Raw data

9/6/2018					
1	2	3	4	5	
0.1764	0.17	0.1767	0.1616	0.15	0.15
0.212	0.223	0.2261	0.2899	0.2873	0.271
0.1225	0.1248	0.1232	0.192	0.2087	0.196
0.2198	0.2126	0.2171	0.2604	0.251	0.259
0.3042	0.3017	0.3269	0.1383	0.1402	0.143
0.1593	0.1506	0.1598	0.253	0.2643	0.253
0.1244	0.1202	0.1282	0.151	0.1541	0.1
0.103	0.115	0.112	0.1411	0.1414	0.140
0.225	0.2248	0.2232	0.192	0.2087	0.196

Raw data table

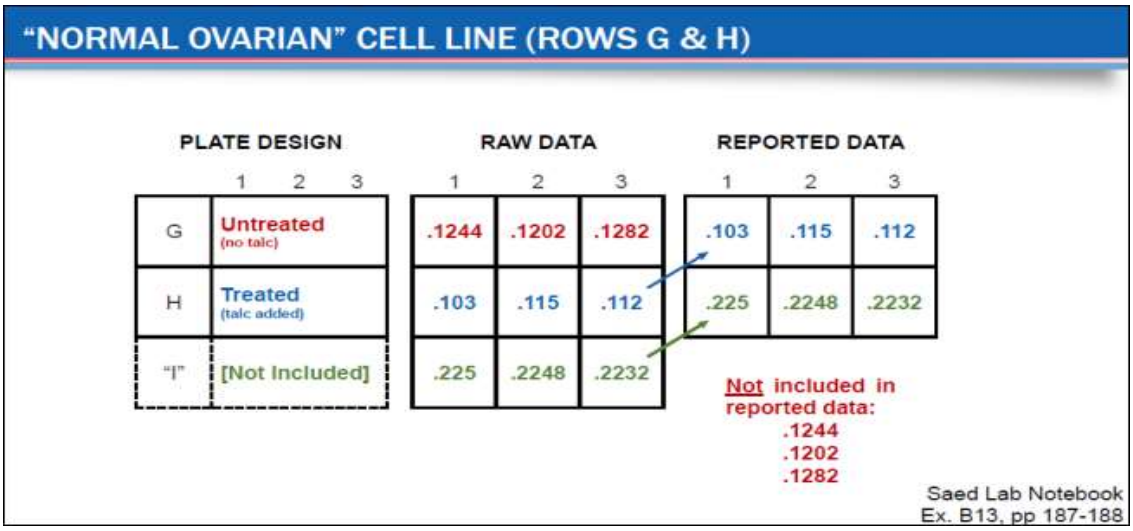
Cell type			
	OD 1	OD 2	OD 3
A2780 unt	0.1764	0.17	0.176
100 ug/ml	0.212	0.223	0.226
SKOV unt	0.2198	0.2126	0.217
100 ug/ml	0.3042	0.3017	0.326
TOV112 unt	0.192	0.2087	0.196
100 ug/ml	0.2604	0.251	0.259
EL-1 unt	0.1616	0.15	0.15
100 ug/ml	0.2899	0.2873	0.271
Normal overian unt	0.103	0.115	0.11
100 ug/ml	0.225	0.2248	0.223
FT33 unt	0.1411	0.1414	0.140
100 ug/ml	0.192	0.2087	0.196

Reported data table

Dr. Saed was unable to explain this discrepancy either,[157] which is extremely concerning because, if the rows in the raw data table indeed correlate with the well plate design – i.e., if the seventh and eighth rows in the raw data table reflect the testing supposedly performed in the seventh and eighth rows of the well plate – then the proliferation results for the untreated cells are ***higher*** than the results for treated cells in the raw data (in contrast to the reported data, which show the

157 (*See id.* Tr. 213:24-217:11, 218:5-221:6.)

opposite):



This would mean that the proliferation assay results reported in Dr. Saed's article are backwards and that talc actually ***decreased*** proliferation, rather than increasing it. In short, it is utterly impossible to verify Dr. Saed's purported conclusions regarding proliferation, even against his own data, and there is a significant probability that he reported inaccurate data in his published article.[158]

The hearing also shed further light on other lab notebook irregularities that

---

158 (*See* 7/22/19 Hr'g Tr. 220:11-221:6.) To the extent plaintiffs suggest that these errors are irrelevant because Dr. Saed's work has been subjected to peer review, that argument should be rejected because the peer-review process does not involve a review of lab notebooks, but instead assumes the integrity of the raw data underlying a submission. In any event, the peer-review process was also irregular. Most notably, the hearing confirmed that Dr. Saed's conflict-of-interest disclosures were insufficient, if not downright fraudulent. Dr. Saed falsely told *Gynecologic Oncology* that he had no conflicts of interest despite having been paid tens of thousands of dollars by plaintiffs' counsel (*see id.* 222:22-223:6), and while he mentioned "consulting" to *Reproductive Sciences*, he failed to tell that journal that he was consulting for plaintiffs' counsel or that the matter was ongoing (*see id.* 225:9-21).

defendants have identified in prior briefing. For example, the averages calculated in several places in the lab notebook appear to be wrong.[159] At the hearing, Dr. Saed contended for the first time that these were not errors at all, but rather the result of a computer formula that disregards "outliers."[160] But this explanation cannot be squared with Dr. Saed's deposition testimony, in which he dismissed the same averages as a "typo,"[161] and never mentioned that the process was computerized.[162] And even if his hearing testimony was truthful, any such computerized formula is not set forth in the lab notebook, has never been shared with defendants and could not be coherently explained by Dr. Saed at the hearing, making his work all the more impossible to replicate.[163]

Finally, Dr. Saed had to admit once again that data in his lab notebooks were whited out and that the notes were not written contemporaneously with the work they purport to memorialize.[164] Dr. Saed himself acknowledged that using white-

---

159 (*See* Saed Mot. 18-19, 54 (ECF No. 9736-2).)

160 (*See* 7/22/19 Hr'g Tr. 22:19-24:2.)

161 (2/14/19 Saed Dep. 450:21-451:14 (Tersigni Cert. Ex. B19).)

162 (*See* 7/22/19 Hr'g Tr. 184:14-187:12.)

163 (*See id.* 187:25-188:11.) As the Court pointed out, whatever formula Dr. Saed uses to exclude outliers is counterintuitive at best. For instance, Dr. Saed excluded 9.98 from the set of 9.98, 10.50, 11.68, even though 11.68 is more than twice as far away from the middle value. (*Id.* 24:19-25:2.)

164 (*Id.* 36:8-12 ("Q: Doctor, in Exhibits G and H, there are pages that have white-out. Correct? A: Yes. Your Honor, this is from the new hired lady."); *id.*

(cont'd)

out in a lab notebook is not proper laboratory practice,[165] and Dr. Neel testified that he has "***never seen anyone use white-out in [his] 30 years as a faculty member***."[166] The use of white-out was particularly concerning because the whited-out portions contained data that are at the heart of Dr. Saed's analysis, including the actual product being reviewed, the methodology utilized, and the dates on which analyses were conducted.[167] Dr. Saed contended that the white out only covered up his "methodology" and not his "original data," but this assertion ignores the fact that original data, including the identity of the product and the dates of entry, were altered, and in any event, whiting out "methodology" would be no virtue because Dr. Saed's methods are precisely the focus of this *Daubert* proceeding.[168]

Dr. Saed also admitted that pages were ripped out of his lab notebooks,

---

(cont'd from previous page)
168:19-169:17 (admitting lab notebook entries were not all "prepared at the time that the work was done").)

165 (*Id.* 177:17-23, 178:12-17.)

166 (7/23/19 Hr'g Tr. 308:21-24 (emphasis added).)

167 (7/22/19 Hr'g Tr. 178:25-179:7 ("[Q:] [Y]ou see there is an entry where there is white-out, and the words Johnson & Johnson are written over at the white-out. . . . That has to do with what product is being reviewed. Correct? A: Yes."); *id.* 180:6-10 ("Q: That's a methodology? A: Yes. Q: It is whited out and those words appear over it. Right? A: Yes."); *id.* 182:6-183:8 (Dr. Saed admitting that dates were whited out and altered).)

168 (*Id.* 178:2-11.)

which he acknowledged is "very bad laboratory conduct,"[169] and that entries in his lab notebooks were not contemporaneous.[170] When pressed about dates by the Court, Dr. Saed admitted that he does not have any memory of when things were entered.[171] Although Dr. Saed attempted to blame these departures from standard practices on a "research assistant" who "is always [making] mistakes," he also testified that he, not his research assistant, retains "ultimate responsibility for the content" of his lab notebooks.[172]

And finally, there is still no explanation for how Dr. Saed's initial reports that he had treated cells with talc for ***48 hours*** conveniently changed to ***72 hours*** after a reviewer for *Gynecologic Oncology* expressed doubt that exposure could cause mutations in such a short time period. When the Court questioned Dr. Saed about this issue, he claimed that there was "an error in the actual manuscript,"

---

169 (7/22/19 Hr'g Tr. 33:17-24 ("[W]e have a new research assistant from China, and she was not familiar with the practice, normal practice of lab notebooks. She wanted to keep everything related to talcum powder in one notebook. So she started a different project in those two pages. So she decided to take them out. I instructed her not to do it. This is very bad laboratory conduct.").)

170 (*Id.* 168:19-169:7.)

171 (*Id.* 176:12-14; *see also id.* 176:19-25 ("Q: With regard to other entries in the notebook that have date, can you tell whether those pages were created on the dates listed on the page or whether they were created later but backdated to the date the work occurred? A: I will try. I don't know. I can't tell all the time.").)

172 (*Id.* 182:21-23; *id.* 37:3-5.) In any event, if Dr. Saed's opinions are derived from an unreliable methodology, it is irrelevant who bears the blame for that unreliable methodology.

which he apparently attributed to unnamed "trainees like clinical residents and fellows."[173] But this testimony does not explain why Dr. Saed repeatedly referenced 48 hours of treatment time – not only in the manuscript, but also in his report and multiple abstracts.[174]

In sum, Dr. Saed's failure to adhere to basic rules concerning recordation of data, along with his lax and unscientific approach to his work, make it impossible to evaluate, let alone replicate, his purported experiments and call into serious question the integrity of his procedures and claimed results.

(b) The Hearing Also Confirmed That Dr. Saed's Results Would Not Support His Opinions Even If He Had Employed Reliable Methods.

The hearing also confirmed that, even ignoring the serious methodological errors and lapses in his work, Dr. Saed's results would not support his conclusion that there is a biologically plausible mechanism by which talc could cause ovarian cancer for two reasons: (1) Dr. Saed's in vitro experiments could not, whatever their results, show that talc is carcinogenic in living animals, much less in human beings, something ***Dr. Saed himself ultimately admitted***; and (2) Dr. Saed did not even show that talc is carcinogenic in a petri dish.

First, Dr. Saed failed to demonstrate that any claimed effects he produced in

---

173 (*Id.* 38:8-39:5.)

174 (*See id.* 157:18-164:10.)

vitro would occur in vivo, meaning that his results "remain one step removed" from what would be necessary to support a causation opinion. *In re Human Tissue Prods. Liab. Litig.*, 582 F. Supp. 2d 644, 663 (D.N.J. 2008).

It is a matter of basic science that cell transformations in a petri dish do not indicate a similar effect in the human body. As Dr. Neel explained in unrebutted and unchallenged testimony, some cells "behave in a semi-transformed way" in vitro "but do not form tumors" in animals or humans.[175] Thus, it cannot simply be assumed from biological effects in a petri dish that the same effect would be produced in humans. Dr. Saed essentially agreed with this principle at the hearing. Although he filibustered when questioned, he admitted having previously testified that he has ***never*** in his ordinary work "classified a substance as a carcinogen based on the result of an in vitro model" alone without in vivo replication.[176] And

---

[175] (7/23/19 Hr'g Tr. 307:9-13.)

[176] (7/22/19 Hr'g Tr. 94:7-14.) This was just one of more than 50 instances in which plaintiffs' experts were impeached with their own prior statements during the hearing. (**Saed:** 7/22/19 Hr'g Tr. 85, 86, 94, 95 , 117, 122, 138, 159-60, 166-67, 168-69, 174, 177, 185-86, 190; **Longo:** 7/24/19 Hr'g Tr. 546, 557, 567, 569, 578-79, 588, 613; **McTiernan:** 7/25/19 Hr'g Tr. 805-06, 844-45, 856-57, 861-62, 913; **Carson:** 7/29/19 Hr'g Tr. 1327, 1330, 1333-34, 1336, 1343, 1344-45, 1350-53, 1361-62, 1364, 1366, 1368, 1369, 1377, 1378-79, 1381, 1402, 1410-11, 1416; **Clarke-Pearson:** 7/30/19 Hr'g Tr. 1574, 1576, 1577, 1578, 1627, 1650, 1678, 1691, 1726.) By contrast, plaintiffs attempted to impeach defendants' experts only a handful of times, and none of these efforts was successful. (*E.g.*, 7/23/19 Hr'g Tr. 316-18 (attempted impeachment of Dr. Neel as to which the Court stated that "I think we're looking at different things" and that there was a "disconnect" between the question posed at the hearing and the supposedly contrary deposition

(cont'd)

he admitted that an in vivo model would "enhance[]" his results[177] – an opinion shared by those who reviewed his paper and rejected it in part because it lacked such a model, as he acknowledged.[178]

When pressed, Dr. Saed remarkably stated that he is simply "not interested in the in vivo effect" of talcum powder.[179] This assertion contradicts his deposition testimony that the only reason he did not perform animal studies was a lack of time and money[180] and that he was "planning to do more work" that included animal studies,[181] as well as his statement to the press in June that his "next project will involve injecting talcum powder directly into rats' reproductive systems," which he said he "'[h]ope[s] . . . will confirm [his] findings.'"[182] Dr. Saed's contradictory

---

(cont'd from previous page)
testimony); 7/31/19 Hr'g Tr. 1863-64 (attempted impeachment of Dr. Saenz as to which the Court remarked about the supposedly impeaching deposition colloquy, "That is a different question. Perhaps you have the wrong cite.").)

177 (7/22/19 Hr'g Tr. 98:2-4.)

178 (*Id.* 139:20-23 (noting reviewer comment that "cell line studies were not convincing").)

179 (*Id.* 98:19-20.)

180 (1/23/19 Saed Dep. 50:10-13 (Tersigni Cert. Ex. B12).)

181 (2/14/19 Saed Dep. 546:7-18.)

182 Richards, *Research Shows How Talc Powder May Change Ovarian Cells*, Cancer Therapy Advisor, June 18, 2019, https://www.cancertherapyadvisor.com/home/cancer-topics/gynecologic-cancer/talc-powder-may-change-ovarian-cancer-cells-new-research/. The same article quotes Dr. Paolo Boffetta, a co-author of the Berge meta-analysis, explaining that "we don't know if the same thing happens in vivo in an actual woman's ovary." *Id.*

and opportunistic statements around this question highlight both that he recognizes that such studies are required to validate his thesis and that he is (rightly) fearful that they will not do so, strongly suggesting that he has strategically avoided conducting them before this *Daubert* process concludes. This, too, supports exclusion of his opinions. *See Hanson*, 353 F. Supp. 3d at 1285-87 (excluding expert who "failed to perform . . . final step" of testing that might have supported or undermined his conclusions).

Nor did Dr. Saed's testimony at the hearing identify any existing animal studies that could plug the gaping hole in his own work. To the contrary, he admitted having relied on only a small portion of the relevant animal studies,[183] two of which were cited in his report and the third referenced in his deposition.[184] And the hearing confirmed that none of these studies is even relevant to, much less supportive of, Dr. Saed's conclusions. Dr. Saed admitted that the first study in his report, a 1969 study by Graham & Graham, does not mention talc at all, but instead is about asbestos exposure.[185] As for the second, Dr. Saed's report cited a 2004 study by Langseth & Kjærheim that was not about animals at all. At the hearing,

---

183 (7/22/19 Hr'g Tr. 102:17-22.)

184 (*See id.* 109:15-110:4.)

185 (*See id.* 103:22-25.)

Dr. Saed said he actually meant to cite a 2008 study by Langseth et al.,[186] but Dr. Saed did not know whether that was an animal study either, and it is not.[187] Accordingly, there is no basis to infer animal or human carcinogenicity from Dr. Saed's results, and his opinion should thus be excluded for lack of fit as well.

Second, the hearing also confirmed that Dr. Saed's findings do not even establish carcinogenic potential at an in vitro level. Most strikingly, the hearing revealed that ***plaintiffs' own experts*** do not believe Dr. Saed has shown carcinogenesis in vitro. Indeed, Dr. Clarke-Pearson explained to the Court that Dr. Saed "stopp[ed] short" of showing "malignant transformation."[188]

The hearing also confirmed that the biomarkers Dr. Saed purportedly observed are entirely irrelevant to the development of cancer, by his own admission. Dr. Saed tested the impact of talc on seven SNPs. But he was forced to

---

186 (*See id.* 106:13-20.)

187 (*See id.* 109:2-8; *see also* Langseth 2008 (Tersigni Cert. Ex. A88).) At his deposition, Dr. Saed attempted to bolster his opinions with reference to a 1993 animal study sponsored by the National Toxicology Program. (1/23/19 Saed Dep. 194:25-196:8; *see also* 7/22/19 Hr'g Tr. 109:13-18.) In fact, that study showed no increased risk of ovarian cancer, *see* Nat'l Toxicology Program, *Toxicology and Carcinogenesis Studies of Talc in F344/N Rats and B6C3F$_1$ Mice (Inhalation Studies)* (1993) (Brennan Cert. Ex. 4), and to the extent it suggested increased risk of other cancers, the FDA determined that, "because of serious flaws" in the study's data, it has "no relevance to human risk," *see* FDA Denial Letter at 3-4 (Tersigni Cert. Ex. A89).

188 (7/30/19 Hr'g Tr. 1544:14-17.) Beyond this, Dr. Clarke-Pearson confirmed that no other published study testing talc treatment of cells has observed "cancer . . . in a cell culture." (*Id.* 1649:3-14.)

admit at the hearing that he previously published that these seven SNPs had "***no association with ovarian cancer***,"[189] and his pre-litigation conclusion has not been "disproven" since.[190] As Dr. Neel explained, Dr. Saed's old opinion is indeed correct: none of the SNPs tested for this litigation is among the "about 100" that have been "associated" with ovarian cancer in "large scale genetic association studies called GWAS."[191]

Dr. Saed's oxidative stress results likewise fail to provide any support for his conclusions about ovarian cancer. As Dr. Saed had to acknowledge at the hearing, one of the *Gynecologic Oncology* reviewers expressly commented that Dr. Saed did "not support[]" his claim that "oxidative stress is a key mechanism to the

---

189 (7/22/19 Hr'g Tr. 131:20-24.)

190 (*Id.* 135:10-16.) Dr. Saed also acknowledged that an article he co-authored and had relied on previously for a supposed association between the CAT SNP and ovarian cancer had in fact studied an association between the SNP and ovarian cancer ***survival***, not development of the disease. (*Id.* 131:5-24.)

191 (7/23/19 Hr'g Tr. 304:4-305:18.) Dr. Saed's SNP findings should be rejected for the independent reason that they are "completely inconsistent with everything we know about modern molecular biology." (*Id.* 305:19-306:1.) As Dr. Neel explained, "it is impossible . . . for ***any substance***" to recode "a particular locus" of DNA "within 48 to 72 hours." (*Id.* 306:14-17 (emphasis added); *see also* Rejection Letter at 4 (Tersigni Cert. Ex. B23) (referring to the changes Dr. Saed purported to find as "surprising").) Dr. Saed all but admitted as much at his deposition and at the hearing, acknowledging that he knows of no other substance reported to cause these types of mutations that quickly. (*See* 7/22/19 Hr'g Tr. 164:15-165:21; 1/23/19 Saed Dep. 252:3-7.)

initiation and progression of ovarian cancer."[192] Nor does any other literature. At the hearing, Dr. Saed could not name a single study "that concludes that oxidative stress . . . causes ovarian cancer"[193] despite having been alerted to the need for such support by the *Gynecologic Oncology* reviewer nearly a year before the hearing. And he ultimately admitted that while "[o]xidative stress . . . has been ***observed*** in ovarian cancer patients,"[194] "[a]ssociation is different than causation."[195]

Not only are Dr. Saed's oxidative stress results irrelevant, but they are also contrary to the very research that plaintiffs have used to buttress them. As Dr. Neel explained, while plaintiffs have cited Buz'Zard & Lau 2007 as supposedly supporting Dr. Saed's findings, that study found that reactive oxygen levels "were actually lower in the treated cells than in the control cells," and it showed none of the "proliferation which Dr. Saed claims" to have found.[196] Similarly, Dr. Neel explained that the paper – Shukla, et al. 2009 – found no changes in gene expression in cells exposed to talc, meaning it supported the conclusion that talc was "essentially . . . biologically inert."[197]

---

192 (7/22/19 Hr'g Tr. 139:25-140:6.)

193 (*Id.* 141:4-142:10.)

194 (*Id.* 142:24-143:1.)

195 (*Id.* 141:15.)

196 (7/23/19 Hr'g Tr. 310:24-33:7.)

197 (*Id.* 311:17-24.)

The hearing also demonstrated that the elevated levels of CA-125 that Dr. Saed claimed to find do not support his causation opinions. Dr. Saed admitted that CA-125 "is not used to diagnose" ovarian cancer,[198] and when pressed by the Court, he could not name a single study "showing an association between elevated CA-125 levels and an increased risk of ovarian cancer."[199] As Dr. Cheryl Saenz explained, although CA-125 can be elevated in women with ovarian cancer, it is "a ***response*** marker," not a protein involved in "initiating the cancer," and in most women, CA-125 levels are only elevated after cancer has progressed to Stages III or IV.[200]

Finally, Dr. Saed conceded at the hearing that his measurement of alleged cell proliferation (which is, in any event, highly suspect, as discussed above) was insufficient to demonstrate carcinogenicity in vitro, because although increased proliferation offers an indirect "indication" that cells may undergo neoplastic transformation, such transformation remains far from "certain."[201] As Dr. Saed recognized, healthy cells "can experience a temporary increase in cell proliferation

198 (7/22/19 Hr'g Tr. 144:23-145:1.)

199 (*Id.* 145:16-20.)

200 (7/31/19 Hr'g Tr. 1846:9-1847:7 (emphasis added); *see also* 7/23/19 Hr'g Tr. 313:6-9 (Dr. Neel explaining "[t]here is no evidence . . . that [CA-125] is involved in the causation of the ovarian cancer").)

201 (7/22/19 Hr'g Tr. 191:17-20.)

in response to" foreign agents like talc,[202] and "temporary . . . proliferation . . . is a ***normal response of all normal cells to agents***."[203]

For all of these reasons, Dr. Saed's findings cannot provide the necessary "evidence to carry [Dr. Saed] all the way down the[] causal chain" to his ultimate conclusions on biological plausibility or causation. *In re Rezulin Prods. Liab. Litig.*, 369 F. Supp. 2d 398, 426-27 (S.D.N.Y. 2005).

2. Plaintiffs' Experts Also Lack Scientific Support For The Theory That Talc Causes Cancer Through Inflammation In The Fallopian Tubes And Ovaries.

(a) Plaintiffs' Experts Lack Reliable Evidence That Externally-Applied Talc Reaches The Fallopian Tubes And Ovaries.

The hearing also demonstrated that plaintiffs' experts lack any reliable evidence for a fundamental underpinning of all their biological plausibility theories – i.e., that talc applied externally to a woman's perineum could make it to the ovaries or fallopian tubes in the first place.[204]

First, the hearing confirmed that the theory that talc sprinkled on the external perineum can enter the vagina and migrate upward – against gravity, vaginal mucus and menstrual fluid – all the way through the vagina, the cervix, the uterus and the fallopian tubes, is not supported by anything other than the experts'

---

202 (*Id.* 189:11-18.)

203 (*Id.* 190:6-19 (emphasis added).)

204 (*See generally* Defs.' Bio. Plausib. Mot. at 18-47 (ECF No. 9736-1).)

own *ipse dixit*, and therefore must be excluded. *See, e.g.*, *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997). Dr. Clarke-Pearson conceded as much at the hearing, acknowledging that the carbon particle studies he relies on involved the insertion of particles into the vaginas of supine women who were given drugs to foster migration.[205] In fact, Dr. Clarke-Pearson agreed that ***no human or animal studies*** "demonstrate the migration of any particulate matter," much less talc, "from the external perineum . . . to the ovaries" or fallopian tubes.[206]

Without studies showing that talc applied perineally actually reaches the ovaries, plaintiffs' experts were left to provide utterly nonsensical hypotheses for how talc ***might*** reach the ovaries. In particular, Dr. Carson testified that "the female reproductive system is essentially an open channel,"[207] and that "vaginal installation" is thus "tantamount to perineal exposure."[208] But Dr. Carson admitted he has only studied the matter "casually," and he ***could not cite a single source*** to support his open-channel theory.[209] As Dr. Saenz explained, Dr. Carson's theory

---

205 (7/30/19 Hr'g Tr. 1663:9-1665:5.)

206 (*Id.* 1665:25-1666:25 (agreeing that "[n]one of the articles that [he] cite[s] in support of [his] opinion regarding migration looked at whether talc can migrate from perineal application through the reproductive organs to the ovaries").)

207 (7/29/19 Hr'g Tr. 1279:3-6.) Dr. Clarke-Pearson similarly testified that there is "no lid or door" between the vagina and the "cervix, uterus, fallopian tubes." (*See* 7/30/19 Hr'g Tr. 1559:9-16.)

208 (7/29/19 Hr'g Tr. 1399:4-20.)

209 (*Id.* 1400:2-4, 1400:21-1401:1.) The hearing also revealed that Dr. Carson is *(cont'd)*

makes no sense because the genital tract is self-evidently not "an open conduit."[210] After all, when a woman "swim[s] in the ocean, [she] do[es not] get out of the water and have a big gush of ocean water come out of [her] vagina."[211]

Finally, plaintiffs' experts' reliance on Heller 1996 to support the migration theory further shows that their opinions are unreliable, as testimony at the hearing also clarified.[212] For example, Dr. Carson stated on direct examination that this study "showed perineal cosmetic talc usage and the relationship of talc being found in ovarian specimens."[213] But he agreed on cross-examination that no such "relationship" was demonstrated; rather, the study found talc in the ovaries of all participants regardless of whether they used talc, and the "'quantity . . . detected in the study did not correlate well with the reported exposure.'"[214] As Dr. Saenz

---

(cont'd from previous page)
merely speculating in contending that the ovaries are susceptible to talc-induced cancer because they "lack" an "intrinsic elimination system." (*Id.* 1404:8-11.) Dr. Carson contended that talc can "stall[] and sequester[]" in the ovaries (*id.* 1403:25-1404:7), but he acknowledged that he had "not conducted any tests" or cited any literature to support his theory (*id.* 1404:12-1405:4).

210 (7/31/19 Hr'g Tr. 1889:7-10.)

211 (*Id.* 1889:10-13.)

212 (*See* 7/30/19 Hr'g Tr. 1671:7-1672:13 (Dr. Clarke-Pearson confirming that he cites Heller 1996 to support migration and arguing that it "showed talc particles in the ovaries"); 7/29/19 Hr'g Tr. 1283:25-1284:10 (Dr. Carson identifying Heller 1996 as "one paper that I've cited, that shows the presence of talc within ovarian tissue," supporting "a proposed mechanism for its insertion into the ovaries").)

213 (7/29/19 Hr'g Tr. 1280:19-21.)

214 (*Id.* 1372:19-1373:7.)

explained, what this study shows is that "perineal application itself does not account for finding talc in the ovaries."[215]

Second, plaintiffs' experts' testimony confirmed that not even they believe that the theory that talc somehow travels to the ovaries after being inhaled is scientifically viable. On direct examination, Dr. Carson volunteered that inhalation is merely a "secondary route of exposure" that is "***extremely minor***" and would lead to "***very insignificant***" exposure.[216] Dr. Clarke-Pearson offered a similarly tepid endorsement, characterizing inhalation as "very unlikely," and a "plausible" but not "probable" mechanism for migration.[217] And even if these experts' testimony were not construed as fully disavowing the inhalation theory, they otherwise confirmed that there is no research supporting it.[218] As the Court

---

[215] (7/31/19 Hr'g Tr. 1832:25-1833:9.) Plaintiffs and their experts attempted to resuscitate their reliance on Heller 1996 by postulating that the non-talc users in the study were diapered with talc as infants. (*See id.* 1876:4-16 (questioning Dr. Saenz on this); 7/30/19 Hr'g Tr. 1672:19-22 (Dr. Clarke-Pearson speculating that participants "may have gotten talc exposure when they were babies").) But as Dr. Saenz explained, "if they were diapered with baby powder, they are now of the age that at least 25 years have passed and we would expect to see in their ovaries either inflammation or ovarian cancer, if, indeed, that is the mechanism by which talc is inducing ovarian cancer." (7/31/19 Hr'g Tr. 1957:7-13.)

[216] (7/29/19 Hr'g Tr. 1282:20-1283:3 (emphases added).)

[217] (7/30/19 Hr'g Tr. 1563:16-1564:1, 1676:24-1677:1, 1694:10-15.) Dr. Clarke-Pearson additionally stated that he would find the inhalation theory even less likely if there is no asbestos in talc. (*Id.* 1694:24-1695:1.)

[218] (*See, e.g.*, 7/29/19 Hr'g Tr. 1369:14-17, 1369:18-24 (Dr. Carson confirming that he knows of no studies supporting inhalation theory and has not conducted his *(cont'd)*

suggested, plaintiffs' experts were just "throwing out . . . possibilities."[219]

In sum, the hearing confirmed that plaintiffs' experts' migration theories are speculative and ultimately, nonsensical, rendering their general causation opinions all the more unreliable.

(b) Plaintiffs' Experts Lack Scientific Evidence That Talc Causes Chronic Inflammation.

The hearing also showed that plaintiffs' experts do not have reliable evidence that talc itself causes chronic inflammation in the fallopian tubes or ovaries.[220]

No expert could identify a study that supported this hypothesis. For example, Dr. Clarke-Pearson agreed that he was not aware of a single published study that supports his opinion that perineal talc use causes chronic inflammation.[221] He also conceded that he does not know the dose at which talc

---

(cont'd from previous page)
own research on the issue; studies have merely "alluded to the possibility of inhalation being a secondary route of talc exposure to the ovaries"); *id.* 1371:2-9 (Dr. Carson claiming that inhalation is supported by "multiple speculation" in various articles).)

219 (*See id.* 1371:10-13.)

220 (*See generally* Defs.' Bio. Plausib. Mot. at 49-54.)

221 (7/30/19 Hr'g Tr. 1653:13-17 ("Q. And you cannot cite to any published study that supports your opinion that any amount of talc a woman uses perineally leads to chronic inflammation. Correct? A. That's correct.").) Dr. Zelikoff, who did not testify at the hearing, likewise testified at her deposition that she is unaware of any study reporting inflammation in perineal talc users. (*See* Zelikoff Dep. 357:22-358:4.)

can purportedly cause chronic inflammation leading to cancer.[222] As a result, the hearing confirmed that Dr. Clarke-Pearson is offering what is essentially an "any exposure" opinion – i.e., that any amount of talc that a woman uses can cause chronic inflammation leading to ovarian cancer.[223] As noted above (*see* pp. 42-43), courts routinely find such opinions unreliable.

Dr. Carson, too, was unable to point to studies showing that talc causes chronic inflammation. Instead, he hypothesized that talc can become "sequestered" during ovulation, when the emission of an egg ostensibly "leaves an open wound on the surface of the ovary" in which talc can become trapped during

---

[222] (7/30/19 Hr'g Tr. 1651:6-12 (Dr. Clarke-Pearson does not "know th[e] threshold" below which talc exposure is safe and has "not identified how much talcum powder must reach a woman's ovaries for her to undergo chronic inflammation that leads to cancer"); *see also id.* 1650:7-14, 1651:18-21.)

[223] (*Id.* 1651:13-17 (Dr. Clarke-Person testifying that "any amount of talcum powder that a woman uses" "increases the risk of causing ovarian cancer"); *see also id.* 1651:22-1652:24 (Dr. Clarke-Pearson testifying that a hypothetical product with 1% talc and 99% cornstarch could cause chronic inflammation leading to cancer).) Dr. McTiernan similarly opined that "one piece of one application of talc" could potentially cause inflammation leading to ovarian cancer. (7/25/19 Hr'g Tr. 795:13-25.) Aside from being inherently unreliable, these "any exposure" opinions are impossible to square with studies finding no association between the use of diaphragms or condoms dusted with talc and ovarian cancer. They also contradict plaintiffs' experts' previously stated opinions that studies addressing talc on diaphragms or condoms are irrelevant because such use does not result in the same exposure as daily perineal use. (*See, e.g.*, Clarke-Pearson Dep. 215:9-17 (Tersigni Cert. Ex. B10).)

the healing process, setting the stage for inflammation.[224] But when pressed by the Court to cite literature in support of such a theory, he could only reference Heller 1996 and Cramer 2007 – neither of which even describes (much less provides evidence for) such a process.[225] As to the inflammation theory more generally, Dr. Carson conceded that a study he cited for the proposition that talc causes inflammation (Okada 2007) addressed neither talc nor ovarian cancer.[226] And he agreed that Heller 1996, discussed above, which specifically looked at talc in human ovarian tissue, did not show any inflammation associated with the presence of talc.[227]

Unable to anchor his opinions to any human studies, Dr. Carson also pointed to several studies reporting that talc caused foreign body reactions in animals. But

---

224 (7/29/19 Hr'g Tr. 1283:4-1284:18.) Notably, in the talc litigation in New Jersey state court, Judge Johnson rejected the same hypothesis as lacking reliable support, citing the concession of plaintiffs' expert Dr. Graham Colditz that "there's got to be continuing studies to understand this whole process better." *Carl v. Johnson & Johnson*, Nos. ATL-L-6546-14, ATL-L-6540-14, 2016 WL 4580145, at *16-18 (N.J. Super. Ct. Law Div. Atl. Cty. Sept. 2, 2016).

225 Cramer 2007 (Tersigni Cert. Ex. A24) posited that talc might be sequestered in lymph nodes and cause immune dysregulation there, not that ovulation creates holes in the ovary that are filled by talc. Heller 1996 (talc) (Tersigni Cert. Ex. A60) does not mention sequestration at all.

226 (7/29/19 Hr'g Tr. 1380:1-1383:1 (Dr. Carson cites Okada 2007 for the proposition that talc "'causes inflammation and fibrotic reaction including the chemotaxis of inflammatory immune cells,'" but agreed that this study "doesn't look at talc at all" and "does not say anything about ovarian cancer").)

227 (*Id.* 1373:16-20.)

when pressed, Dr. Carson quickly conceded that none of the studies he relied on concluded that the observed inflammation led to neoplastic changes or cancer.[228] For example, although Dr. Carson testified on direct examination that the Hamilton 1984 study found that the "inject[ion of] talc into the ovarian bursa of rats . . . resulted in papillary transformation and the papillae that resulted may represent early neoplasia," he was later forced to concede that the authors actually reported finding ***no evidence*** of "frank neoplasia," or "precancer," in any exposed rat ovary, and the "Hamilton study ***did not*** conclude that chronic inflammation from talc led to neoplastic changes or cancer."[229] These concessions are no surprise; as defense expert Dr. Neel clarified, "[i]nflammation is a broad term," and studies that report that talc induced acute local reactions such as granulomas do not show that "talc cause[s] the kind of inflammation that is associated with cancer or cancer initiation."[230]

Faced with a lack of evidence supporting their inflammation theory, plaintiffs and their experts resorted to misrepresenting studies at the hearing. For

---

228 (*E.g.*, *id.* 1383:3-1385:11 (agreeing that Radic 1998 did not study the ovaries or show neoplastic changes); *id.* 1389:20-1390:23 (agreeing that Keskin 2009 does not show that inflammation can lead to neoplastic changes); *see also* 7/25/19 Hr'g Tr. 817:20-23 (Dr. McTiernan agreeing that Keskin 2009 found that "there was no neoplastic change in the rats who had the talc directly placed into their ovaries").)

229 (7/29/19 Hr'g Tr. 1298:14-17, 1385:21-1387:17 (emphasis added).)

230 (7/23/19 Hr'g Tr. 321:22-322:19.)

example, Dr. Clarke-Pearson heavily relied on the Penninkilampi 2018 meta-analysis as supposedly supporting the inflammation theory during his direct examination.[231] On cross-examination, however, he was forced to concede that this study actually stated that the mechanism by which talc use might cause ovarian cancer is "uncertain" – a conclusion he and plaintiffs' counsel "left out" of his direct examination.[232] As noted above, this Court and others have recognized that relying on studies for propositions they do not support is unreliable, *see, e.g.*, *Schepise*, 1997 WL 897676, at *17 (excluding expert who relied on studies that did not support his opinions), and the fact that plaintiffs' experts did so here only underscores the lack of evidence supporting the inflammation theory.

(c) Plaintiffs' Experts Have No Basis To Link Inflammation To Ovarian Cancer.

The hearing also revealed that plaintiffs' experts do not have reliable evidence linking chronic inflammation to ovarian cancer.

Dr. Clarke-Pearson agreed that "not all inflammatory conditions lead to cancer" and that "research regarding whether chronic inflammation can cause

---

231 (7/30/19 Hr'g Tr. 1540:5-18.)

232 (*Id.* 1635:19-1637:18; *see also id.* 1640:23-1641:12.) Dr. Clarke-Pearson also agreed that he failed to cite an additional meta-analysis (Berge 2018), which similarly states that the mechanism of carcinogenicity is "not understood and remains questionable." (*Id.* 1647:3-1648:20 (Dr. Clarke-Pearson "failed to put [Berge 2018] in [his] report").)

ovarian cancer is ongoing."[233] Although he contended that there is evidence that ovarian cancer is caused by chronic inflammation, he had to concede that studies that have directly examined the issue have not confirmed his theory.

In particular, Dr. Clarke-Pearson conceded that Merritt 2007 – a study that looked at (1) whether pelvic inflammatory disease ("PID") is associated with an increased risk of ovarian cancer and (2) whether the use of anti-inflammatory drugs is associated with a decreased risk of ovarian cancer – found neither to be the case, concluding as a result "that chronic inflammation is unlikely to be a cause of ovarian cancer."[234] Dr. Clarke-Pearson also conceded that Penninkilampi 2018 is in accord, reporting among other things that "NSAIDs findings have not supported the chronic inflammation theory."[235]

Defense expert Dr. Saenz confirmed that "the epidemiologic literature on [whether anti-inflammatory drugs reduce ovarian cancer risk] is very inconsistent," and explained that as such, "prescribing NSAIDs as a method to reduce the risk of ovarian cancer is not something that's accepted by the gynecologic oncology

---

233 (*Id.* 1648:21-24, 1653:18-23.)

234 (*Id.* 1630:21-1635:12.)

235 (*Id.* 1641:12-1642:2.) Dr. Clarke-Pearson additionally pointed to illustrations in several review articles (Balkwill 2001 and Shan & Liu 2009) to attempt to show how inflammation caused by incessant ovulation leads to ovarian cancer. (*Id.* 1567:5-1570:20.) But these review articles did not contain any original research and expressly labeled the inflammation theory a "hypothes[i]s." (*Id.*; *see also id.* 1628:5-1630:3.)

community."[236] She further explained that the relationship between PID and ovarian cancer is likewise "fairly inconsistent," with an association reported only for borderline tumors (which are essentially different diseases than high grade serous ovarian cancer, and are not the focus of this MDL proceeding).[237] Finally, both Drs. Saenz and Neel explained that the known precursors to ovarian cancer – STIC lesions and p53 mutations – are not associated with inflammation.[238] Dr. Saenz testified that she has "looked at the tissues that have been stained to identify cells that contain p53 mutations and cells that have STIC lesions or tubes that have STIC lesions, and there is ***no associated inflammation*** with these tissues, even

---

[236] (7/31/19 Hr'g Tr. 1821:10-23; *see also id.* 1934:25-1935:25 (explaining that a review article shown to her on cross-examination (Savant 2018) "only further demonstrates the inconsistencies . . . because using low dose aspirin daily decreased the risk of a woman getting cancer" whereas "non-aspirin NSAIDs actually increased her risk of getting cancer").) Dr. Diette similarly testified that studies examining the effect of anti-inflammatory drugs produced "a mixture of findings," including "a positive risk for NSAIDs use, . . . which is inconsistent with the idea that NSAIDs would be protective against ovarian cancer." (7/26/19 Hr'g Tr. 1057:2-9.)

[237] (7/31/19 Hr'g Tr. 1843:5-18; *see also id.* 1930:24-1931:13 (similar).) Dr. Saenz further explained that there are no data showing that women with other known inflammatory conditions, such as ulcerative colitis or Crohn's disease, have an increased risk of ovarian cancer. (*Id.* 1843:19-1844:2.)

[238] (*E.g.*, 7/23/19 Hr'g Tr. 297:15-299:10 (Dr. Neel explaining, *inter alia*, that studies have "looked at STICs from normal patients and patients undergoing risk reduction surgery for BRCA1 and BRCA2 lesions and found no evidence of increased inflammation"); *see also* Shih Rep. at 26-27 (Tersigni Cert. Ex. C20) (study of cells with p53 mutations and STIC lesions "did not observe chronic inflammation in the p53 signatures and STIC lesions").)

though the cancer is already in the process of developing."[239] As Dr. Neel summarized, this evidence collectively shows that "although several authors have suggested chronic inflammation might play a role in ovarian cancer, that remains a hypothesis and basically speculation at this point."[240]

In sum, the hearing made clear that the notion that chronic inflammation causes ovarian cancer is a hypothesis at best and thus cannot support an opinion on biological plausibility. *See, e.g., In re Accutane Prods. Liab.*, 511 F. Supp. 2d 1288, 1296 (M.D. Fla. 2007) (rejecting expert's theory of biological plausibility because it had "not been verified by testing" or "peer-reviewed" and therefore amounted to nothing more than "an educated guess").

IV. PLAINTIFFS' EXPERTS' TESTIMONY MAKES CLEAR THAT THEY CONDUCTED SCIENCE FOR THE COURTROOM, NOT FOR THE LABORATORY.

The law is clear that litigation experts must "employ[] in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152 (1999).

---

239 (7/31/19 Hr'g Tr. 1842:5-15 (emphasis added); *see also id.* 1944:2-17 (Dr. Saenz similarly explaining that if ovarian cancer is "in the process of developing and you are proposing that chronic inflammation is the inciting event, that should be there, where the precancer is, and it's not").) Dr. Saenz further explained that, having operated on "somewhere between 1,500 [and] 1,800" ovarian cancer patients, she has only seen evidence that "cancer itself . . . can be inflammatory" – there has been no "evidence of a chronic inflammatory response that would have been incited by a foreign body." (*Id.* 1838:21-1840:1.)

240 (*See, e.g.*, 7/23/19 Hr'g Tr. 300:18-23.)

Accordingly, as explained in detail in defendants' General Causation *Daubert* motion, courts are especially skeptical of experts whose litigation opinions lack connection to their scientific work or contradict their prior published views.[241] Recent caselaw confirms this fundamental point. *See, e.g.*, *Davis v. McKesson Corp.*, No. CV-18-1157-PHX-DGC, 2019 WL 3532179, at *26 (D. Ariz. Aug. 2, 2019) (excluding testimony of expert in part because court identified "stark inconsistencies" between a speech given by the expert outside litigation and his expert report that were "very concerning" and "suggest[ed] that he has not employed the same level of intellectual rigor to his opinions as he does in practice").

Several notable examples illustrate that plaintiffs' experts ***are not*** employing the same level of rigor in the courtroom that characterizes their activities in the field. ***First***, Dr. Clarke-Pearson's opinions in this litigation are at odds with the advice he has given his patients in over 40 years of practice as a gynecological oncologist. In fact, during his 40 years of "research," "teaching" and "practice," Dr. Clarke-Pearson has never warned his patients about the alleged dangers of talc.[242] Moreover, he has never recommended increased screening or monitoring

---

241 (*See* GC Mot. at 113-14.)

242 (*See* 7/30/19 Hr'g Tr. 1573:10-1574:1 (discussing "40 years" of "research[] and t[eaching] and practice[]"); *id.* 1574:2-7 (agreeing he never told a patient talcum powder caused ovarian cancer); *see also id*. 1576:22-1577:1 ("Q. Your

(cont'd)

for ovarian cancer based on a patient's prior talc use.[243] Indeed, the questionnaire and other forms presented to patients in Dr. Clarke-Pearson's practice do not ask gynecologic patients about their history of talc use,[244] although he testified that he plans to update his intake forms to include a question about talc use in the future, "when we finish getting rid or using up the forms we currently use."[245]

When asked at his deposition whom he has informed of his opinion that talc is a cause of ovarian cancer (other than plaintiffs' counsel), Dr. Clarke-Pearson said he had only communicated it to one "friend" who was a past president of the Society of Gynecologic Oncology, and that he only did so after preparing his report for this litigation, although at the hearing, Dr. Clarke-Pearson stated that he has since additionally informed the CEO of ACOG that he is serving as an expert

---

(cont'd from previous page)
patients are moms and sisters and aunts, and you don't advise them that they should not be purchasing an over-the-counter product for their family that you believe causes ovarian cancer. True? A. That's true.").)

243 (*Id*. 1574:17-21.)

244 (*Id*. 1575:10-17 ("THE COURT: I guess the question is, though, any of your patients that come in put aside whether they have been diagnosed with ovarian cancer, if they are coming in for appointments of any kind, is there something that's used in the questionnaire, do your forms anywhere ask that of any of the gynecological patients? THE WITNESS: Not at this time.").) Dr. Clarke-Pearson testified that until now, the intake forms have not asked patients about their history of talc use because knowing whether or not a patient used talc previously is "not going to help [him] take care of that patient that has ovarian cancer," and he does not want his patients to feel "guilty" for using talc. (*Id*. 1575:2-9, 1576:14-21.)

245 (*Id*. 1723:15-25.)

for plaintiffs and believes that ACOG should investigate the connection between talc and ovarian cancer.[246] And when Dr. Clarke-Pearson appeared on Fox News in 2014, in a segment designed to warn women about ovarian cancer risks, he did not mention talc use because – as he admitted – he "didn't believe" at the time that talc use causes ovarian cancer.[247]

Plainly, Dr. Clarke-Pearson does not view talc as a cause of ovarian cancer outside of the context of this litigation.

Second, Dr. McTiernan's testimony underscored that her opinions constitute advocacy rather than a neutral interpretation of data as an independent scientist. While plaintiffs touted Dr. McTiernan's testimony before Congress regarding the purported connection between talc and ovarian cancer as evidence of her expertise and supposed leadership on this issue, the circumstances surrounding her testimony make clear that she was acting as a biased advocate, not an independent scientist. Specifically, Dr. McTiernan met with plaintiffs' counsel prior to the hearing, shared her statement with them prior to the hearing, and even rode to the hearing with them.[248]

Dr. McTiernan's testimony also established that the opinions she has offered

---

246 (Clarke-Pearson Dep. 67:8-68:18; *see also* 7/30/19 Hr'g Tr. 1723:1-17.)

247 (7/30/19 Hr'g Tr. 1590:17-1592:12.)

248 (7/25/19 Hr'g Tr. 927:10-24.)

here contradict those she has taken outside of litigation. Most notably, while Dr. McTiernan has denied in this litigation that there is a hierarchy of epidemiological evidence,[249] she admitted that, at the time she was a panelist at the WCRF, the WCRF issued a publication, which stated that "[t]he hierarchy of epidemiological evidence places cohort studies above case-control studies," and that "[b]ecause case-control studies are particularly prone to recall and other bias, they were not routinely reviewed."[250] She also admitted that the same report explained that "[c]ohort studies are likely to be the main source of evidence owing to the long latent period for cancer to develop and also to their prospective design."[251] Thus, it is clear that Dr. McTiernan, too, has failed to adhere to the rigors of her field in developing her opinions and is instead advancing a litigation-driven agenda.

Third, Dr. Carson's publicly professed views also differ from those he has expressed in this litigation. Specifically – and contrary to the central tenet of toxicology that dose is relevant to toxicity – Dr. Carson repeatedly testified that "any exposure to a carcinogen increases [the] risk of cancer to some extent."[252]

---

249 (*See, e.g.*, McTiernan Dep. 116:24-117:5 (Tersigni Cert. Ex. B2); *id.* 118: 19-24 ("Q. So it is your view that there is no generally accepted hierarchy of epidemiological evidence? A. I think it depends entirely on what the question is.").)

250 (7/25/19 Hr'g Tr. 869:12-870:5, 871:1-17.)

251 (*Id.* 869:12-870:5.)

252 (7/29/19 Hr'g Tr. 1319:14-16.)

However, Carson admitted that just a few months ago, he was quoted in the newspaper stating that the levels of airborne benzene – a Group 1 carcinogen – in the areas surrounding Deer Park, Texas, did not rise to the levels of a health effect concern,[253] effectively acknowledging the principles of dose that he has ignored here. This highlights that Dr. Carson is taking a different approach to the issue of dose in the courtroom than in his publicly expressed scientific views.

For these reasons, too, plaintiffs' experts' testimony must be excluded.

CONCLUSION

For the foregoing reasons, the Court should exclude plaintiffs' experts' general causation testimony.

Dated: October 7, 2019

Respectfully submitted,

/s/ *Susan M. Sharko*
Susan M. Sharko
DRINKER BIDDLE & REATH LLP
600 Campus Drive
Florham Park, New Jersey 07932
Telephone: 973-549-7000
Facsimile: 973-360-9831
E-mail: susan.sharko@dbr.com

John H. Beisner
Jessica D. Miller
SKADDEN, ARPS, SLATE,
MEAGHER & FLOM LLP
1440 New York Avenue, N.W.

253 (*Id.* 1319:21-1320:1, 1320:15-23.)

Washington, D.C. 20005
202-371-7000

Attorneys for Defendants Johnson & Johnson and Johnson & Johnson Consumer Inc.