UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	MDL No. 2741 Case No. 16-md-02741-VC
This document relates to: *Karen Delorme-Barton v. Monsanto Co.*, Case No. 18-cv-01427-VC	**PRETRIAL ORDER NO. 289: ORDER ON MOTIONS TO EXCLUDE EXPERTS TOMASETTI, NAVARRO, AND SLACK** Re: Dkt. Nos. 16944, 16945, 16947

The plaintiff's motion to exclude the testimony of Dr. Cristian Tomasetti is denied. The motions to exclude Dr. Willis Navarro and Dr. Graham Slack—motions that attack these experts mainly for their reliance on Tomasetti's research—are largely denied. This order assumes a familiarity with the Court's prior orders on general and specific causation and the Ninth Circuit's opinion in *Hardeman*. *See generally In re Roundup Products Liability Litigation*, 390 F. Supp. 3d 1102 (N.D. Cal. 2018) (Pretrial Order No. 45, Dkt. No. 1596); *In re Roundup Products Liability Litigation*, 358 F. Supp. 3d 956 (N.D. Cal. 2019) (Pretrial Order No. 85, Dkt. No. 2799); *Hardeman v. Monsanto Company*, 997 F.3d 941 (9th Cir. 2021). It also assumes a familiarity with the record related to Tomasetti, Navarro, and Slack, including Tomasetti's testimony at the December 12, 2023, *Daubert* hearing and the exhibits then introduced into the record. *See* Hearing Tr., Dkt. No. 17736; Hearing Exs., Dkt. No. 17662.

I

Tomasetti, like other Monsanto general causation experts, opines that there is no convincing evidence that glyphosate is associated with NHL. That view is based on largely the

same literature that has previously been the subject of the general causation inquiry in this litigation. *See* Expert Report of Cristian Tomasetti at 13–32, Greenwald Decl. ISO Pls.' Mot. Ex. F, Dkt. No. 16947-7 ("Tomasetti Report"); *see generally* Pretrial Order No. 45, Dkt. No. 1596. Delorme-Barton barely mentions this aspect of Tomasetti's opinions (except insofar as she argues that Tomasetti is unqualified to offer any part of his testimony). But Tomasetti offers something new: he attempts to explain why most cases of NHL develop without exposure to any known risk factors. Previously, experts in this case have described such cases as "idiopathic," meaning they lack a known cause. This is the primary focus of Delorme-Barton's attack.

Tomasetti says that a significant portion of the genetic mutations that drive cancers are not caused by hereditary or environmental factors, but instead by random errors that occur during natural processes of cell replication. To put it more plainly, he says it is "bad luck" that causes most genetic mutations that drive cancer, rather than inherited traits or exogenous carcinogens. Tomasetti uses a shorthand to refer to the three factors he says are responsible for cancer-causing genetic mutations: random gene replication errors are abbreviated as "R," environmental and lifestyle factors as "E," and hereditary factors as "H."

With respect to Non-Hodgkin Lymphoma, Tomasetti says that "[t]he current evidence indicates that NHL is one of the most random ('bad luck') cancers we know of, almost completely due to R factors." Tomasetti Report at 12. He estimates that less than 5% "of all the mutations found in NHL are due to E or H factors," with the remaining 95% attributable to random replication errors. *Id.* Tomasetti adds that "[i]n the absence of an exposure to a proven cancer-causing E or H factor it is reasonable to attribute a given cancer to R." *Id.*

Tomasetti explains that cancer is the result of an accumulation of harmful genetic mutations, known as "driver mutations," that cause a cell to grow and divide out of control. *See* Tomasetti Report at 3; *accord* American Cancer Society, *Gene Changes and Cancer* 1, 5–6 (Aug. 31, 2022), *available at* https://www.cancer.org/cancer/understanding-cancer/genes-andcancer/gene-changes.html, Hearing Ex. 119, Dkt. No. 17662-12 ("American Cancer Society"). According to Tomasetti, the prevailing scientific view prior to the publication of his

research was that all or most driver mutations could be attributed to either hereditary factors or environmental factors. Tomasetti Report at 2–3. For cancers where no such factors had been identified, people tended to assume that they existed but were undiscovered.

In 2015, Tomasetti and his co-author Dr. Bert Vogelstein, an eminent oncologist, published a paper reporting that about two-thirds of the variations in cancer risk across different tissues in the body could be explained by differences in the rates at which stem cell division occurred in those tissues.[1] Those tissues with more stem cell divisions, and thus with more opportunities for random mutations to accumulate, had a higher cancer risk than those with fewer divisions and fewer opportunities for random mutations. The broad implication was that endogenous processes of cell division had a large and underemphasized role in cancer development. Tomasetti is trained as an applied mathematician/biostatistician, and the methods of his research—in this and other studies—are essentially mathematical: the study analyzed data regarding the cancer risk of various tissues, along with available estimates of the rate of stem cell division in each tissue. Statistically, the results of this study were robust. Tomasetti Report at 9.

Tomasetti's 2015 paper produced a strong, and often critical, scientific response. IARC, for example, published criticisms of the paper that targeted its estimates of the number of cells and stem cell division rates in each tissue, its omission of certain tissues with large incidences of cancer (like breast and prostate), and the geographical limitations of the underlying data, which was derived only from the United States. Tomasetti Report at 10–11.

A follow-up paper in 2017 addressed some of these concerns; it used worldwide cancer data and included additional types of cancer in its analysis.[2] The 2017 paper also took a different methodological approach based on cancer genome sequencing and epidemiological data. It essentially replicated the results of the first study, again finding to a high degree of statistical

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[1] Cristian Tomasetti & Bert Vogelstein, *Variation in Cancer Risk Among Tissues Can Be Explained by the Number of Stem Cell Divisions*, 347 Science 78 (2015), Greenwald Decl. ISO Pls.' Mtn. Ex. E, Dkt. No. 16947-6.

[2] Cristian Tomasetti et al., *Stem Cell Divisions, Somatic Mutations, Cancer Etiology, and Cancer Prevention*, 355 Science 1330 (2017), Greenwald Decl. ISO Pls.' Mtn. Ex. D, Dkt. No. 16947-5 ("Tomasetti (2017)").

significance that "for all cancer combined, at most 29% and 5% of all mutations were respectively attributable to E and H factors, with the remaining 66% presumably to R." Tomasetti Report at 11. The 2017 paper also provides the support for Tomasetti's conclusion that NHL is more random than the average cancer, with 95% of its mutations attributable to R. *Id.* at 12.[3]

Tomasetti has continued this line of research in subsequent years, and his work appears to have continued to lend additional support, through a variety of methodologies, to his basic conclusion that random replication errors drive a significant majority of cancer-causing genetic mutations across all types of cancer. *See id.* at 12–13; Hearing Tr. 60:6–63:2 (Tomasetti discussing corroborating findings of his research since 2017). Tomasetti's theory also appears to have moved from an initial phase of scientific dispute to a certain level of acceptance, at least as far as its core thesis is concerned. The American Cancer Society, for example, explains on its website that driver mutations

> [s]ometimes . . . happen when a cell's DNA is damaged, such as after being exposed to radiation or certain chemicals. But often these mutations occur randomly, without having an outside cause. For example, during the complex process when a cell divides to make 2 new cells, the cell must make another copy of all of its DNA, and sometimes mistakes (mutations) occur while this is happening. Every time a cell divides is another chance for gene mutations to occur. The number of mutations in our cells can build up over time, which is why we have a higher risk of cancer as we get older.

American Cancer Society at 4.

II

Delorme-Barton first targets Tomasetti's qualifications, arguing that he should not be permitted to offer a general causation opinion in this case because his training is in applied mathematics rather than in medicine, toxicology, epidemiology, or in some specific NHL-related expertise. But Tomasetti has devoted his career to studying the origins of cancer, and he has

[3] The paper's supplementary materials contain the cancer-specific data and calculations on which this conclusion is based. *See* Hearing Tr. at 52:22–53:4; 92:10–95:14; *see also* Hearing Ex. 36, Dkt. No. 17662-1.

spent his professional life in schools of medicine, schools of public health, and hospital systems. *See* Cristian Tomasetti CV, White Decl. ISO Def.'s Opp'n Ex. 8, Dkt. No. 17001-9. At present, Tomasetti is the Director of the Center for Cancer Prevention and Early Detection and the Director of the Division of Integrated Cancer Genomics at City of Hope, a network of cancer hospitals and research centers. Previously he was Associate Professor in the Department of Oncology at Johns Hopkins University School of Medicine, and before that he held a fellowship at the Harvard School of Public Health and the Dana-Farber Cancer Institute. *Id.* Any supposed deficiencies in Tomasetti's knowledge of hematology or oncology can be used to undermine his credibility, but they do not show him to be unqualified. "So long as the expert's testimony is 'within the reasonable confines of his subject area,' a lack of particularized expertise generally goes to the weight of the testimony, not its admissibility." Pretrial Order No. 45 at 7 (citing *D.F. ex rel. Amador v. Sikorsky Aircraft Corp*., No. cv-00331-GPC-KSC, 2017 WL 4922814, at *14 (S.D. Cal. Oct. 30, 2017); *Avila v. Willits Environmental Remediation Trust*, 633 F.3d 828, 839 (9th Cir. 2011); *United States v. Garcia*, 7 F.3d 885, 889-90 (9th Cir. 1993)). Tomasetti is qualified to offer all the opinions disclosed in his expert report, including those about cancer etiology, the application of his research to NHL, and the weakness of the evidence for an association between glyphosate use and NHL.[4]

Delorme-Barton says that Tomasetti's research has been heavily criticized by the scientific community, and that these criticisms have exposed his methodology as unreliable. But Delorme-Barton gives only a partial, and therefore misleading, picture of the reaction to

[4] In response to questioning by the Court, Tomasetti made the case that genetic medicine has made his own expertise more than merely sufficient in cases like this. *See* Hearing Tr. 81:4–81:19 (explaining that cancer research "has become very mathematical"). Later, he said that he believed genetic analysis of "mutational signatures"—again a mathematical endeavor—will play an increasingly important role in the specific causation analysis in cases like this one in the future. *See id.* at 73:11–76:19. Even with allowances made for self-promotion, Dr. Tomasetti's points had some force. They also affirmed the Court's growing sense that the law giving special deference to medical doctors in the domain of specific causation, and especially the performance of "differential diagnoses," is outmoded. Not only do most doctors not perform such analysis in their actual practice, but it may soon be the case—if it isn't already—that such analysis requires an expertise that many medical clinicians do not have.

Tomasetti's research. It is certainly true that the 2015 paper was the target of a critical response. However, in subsequent years Tomasetti accounted for these methodological critiques, beginning with the 2017 paper. And at the *Daubert* hearing, Tomasetti offered scientifically reasonable explanations why some specific criticisms should not shake confidence in his results. For example, he explained that a statistical "sensitivity analysis" had shown that the results of his 2015 study would be substantially unaffected by any inaccuracies in the underlying estimates of stem cell division rates. Hearing Tr. at 33:24–36:3. As noted above, Tomasetti replicated his basic findings in his later work, and his theory has entered the scientific mainstream to some degree. *See, e.g.*, American Cancer Society at 5–6.[5] It is telling that nearly 85% of the critical publications cited by Delorme-Barton date from the period between Tomasetti's 2015 paper and his 2017 paper. *See* Bibliography of Critical Articles, Greenwald Decl. ISO Pl.'s Mot. Exclude Ex. G, Dkt. No. 16947-8.

To be sure, the implications of Tomasetti's research have been a subject of serious debate, and to an extent this remains the case.[6] The critical literature reveals that Tomasetti's theory may be vulnerable to the claim that it oversimplifies the complex process of cancer development. For example, one critical piece argues that random replication errors might mediate the effects of environmental carcinogens, which would undermine Tomasetti's apparent claim to "partition the etiological determinants of a disease so that their relative effects add up to 1." *See* Perduca (2018). But while this criticism shows there is room to debate Tomasetti's

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[5] *See also, e.g.*, Song et al., *Cancer Prevention: Molecular and Epidemiologic Consensus*, 361 Science 6409 (2018), White Decl. ISO Def.'s Opp'n Ex. 18, Dkt. No. 17001-19 ("Song (2018)") ("However, not all cancers are preventable by changes in environment or lifestyle. Recent research . . . has shown that mutations due to random mistakes during normal DNA replication (R) play a major role in cancer etiology, along with environment and lifestyle (collectively denoted E) and heredity (H).").

[6] *See, e.g.*, Anya Plutynski, *Is Cancer a Matter of Luck?*, 36 Biol. & Phil. 2 (2021), Greenwald Decl. ISO Pls.' Mot. Ex. H, Dkt. No. 16947-9 (analyzing the literature discussing Tomasetti and Vogelstein's research and, among other things, critiquing their use of the term "luck"); Victoria Perduca et al., *Stem Cell Replication, Somatic Mutations and Role of Randomness in the Development of Caner*, 34 Eur. J. Epidemiology 439 (2019) Greenwald Decl. ISO Pls.' Mot. Ex. J, Dkt. No. 16947-11 ("Perduca(2018)") (arguing that "the claim that cancer is mostly explained by intrinsic random factors is unsupported by data and theoretical models").

conclusions and the way he presents them, it does not come close to exposing Tomasetti's theory as an "unreliable nonsense opinion[]." *City of Pomona v. SQM N. Am. Corp.*, 750 F.3d 1036, 1044 (9th Cir. 2014). Nor do any of the other critical articles filed by Delorme-Barton. Instead, the record as a whole leaves the impression that Tomasetti's basic theory has achieved general acceptance, while important debate continues about its implications. And even if Tomasetti's testimony touches on matters that remain debated or debatable, that does not make it unreliable. It is not the Court's task to "t[ake] sides on questions that are currently the focus of extensive scientific research and debate—and on which reasonable scientists can clearly disagree." Pretrial Order No. 45 at 46 (*quoting Milward v. Acuity Specialty Prod. Grp., Inc.*, 639 F.3d 11, 22 (1st Cir. 2011)) (alteration in original). Delorme-Barton might wish to draw from the debate over Tomasetti's work on cross-examination or through another expert. But she has not shown that Tomasetti is a practitioner of junk science.

Delorme-Barton also makes a narrower argument that, regardless of the validity of Tomasetti's theory in general, his opinions about NHL are unreliable. She specifically targets a figure offered by Tomasetti—namely that 95% of mutations in NHL are caused by random replicative errors rather than environmental factors. First, Delorme-Barton says that Tomasetti's calculation, and the application of his theory to NHL, are based on incorrect assumptions about the biological mechanisms through which NHL develops. For example, Tomasetti's figures are based on data about the rate of division of hematopoietic stem cells, while Delorme-Barton says that the focus should be on B-cells, which do not divide spontaneously. Monsanto responds that this argument exploits a linguistic ambiguity: NHL is observed in mature B-cells, so it is sometimes said to "originate" there. But, as Tomasetti explained at the hearing, the cancer is also properly understood to "originate" in the stem cells that, through a process of division and cellular differentiation, give rise to those B-cells and pass their genetic mutations to them. Hearing Tr. at 36:4–38:13. Based on the information in the record, this explanation—which

Plaintiff has not rebutted—is scientifically plausible.[7]

A second criticism of Tomasetti's opinions about NHL concerns the data and calculations underlying Tomasetti's 95% figure. That data, as Delorme-Barton points out, has baked-in assumptions about what environmental "E" factors do and do not cause NHL. The 2017 paper—which is where the 95% figure comes from—relied on data maintained by a nonprofit called Cancer Research UK ("CRUK"). CRUK's data is, in relevant part, based on a 2011 publication by D.M. Parkin in the *British Journal of Cancer*.[8] Hearing Tr. 50:16–50:21. For a variety of cancers including NHL, the Parkin study calculated the proportion of cases attributable to relevant environmental factors. The factors considered were those that met a set of selection criteria based on the strength of the evidence supporting their carcinogenic properties and availability of data on exposure levels. *See* Parkin (2011) at S2. Glyphosate was not one of the factors included. Delorme-Barton says that this shows Tomasetti's calculations to be unreliable and his figures to be misleading, for "all probable causes of NHL were not included as E and were counted as R, thereby artificially skewing the calculation towards a greater percent attributed to R."[9] Pl.'s Reply, Dkt. No. 17053 at 4.

At the hearing, Tomasetti did not hide from the fact that glyphosate was not included as an environmental carcinogen in the underlying data. Hearing Tr. at 76:20–77:17. He also said that if new environmental causes of NHL were discovered, it was true that his calculation of the

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[7] Tomasetti also offered a reasonable response to the argument that his theory fails to account for the importance of chromosomal translocations in NHL. Tomasetti testified that his research uses the term "mutation" in the most general sense, such that it encompasses both point mutations and translocations. Hearing Tr. at 14:14–20 ("I want to clarify, even in all of my research, when I say 'mutations,' I mean mutations in the most general sense. Mutations are—go from point mutations, which is the example I give now, to deletion/insertions, chromosomal translocations, chromosomal loss, even, epigenomics events and so on.").

[8] D.M. Parkin, *The Fraction of Cancer Attributable to Lifestyle and Environmental Factors in the UK in 2010*, 105 British J. Cancer S2 (2011), Hearing Ex. 113, Dkt. No. 17662-11 ("Parkin (2011)").

[9] At the *Daubert* hearing, Delorme-Barton tried to make the Parkin data seem unreliable for a variety of other reasons, but none of these efforts succeeded. And in a footnote to her motion, Delorme-Barton offers a brief argument that the 95% figure should be excluded because Dr. Tomasetti refuses to identify the data or how he calculated it. But this is not the case. Both the data and the calculations were identified and discussed at length at the *Daubert* hearing.

proportion of mutations attributable to R would correspondingly decrease. *Id.* at 77:18–77:22. But while this shows that the figure has limitations, it does not show that his methods were scientifically unreliable. Most importantly, the record does not support the view that Tomasetti's methods were designed to "artificially" inflate the proportion of mutations attributable to R. In fact, Tomasetti explained that the analysis in his 2017 paper was designed to *overestimate* the effect of E factors, and that glyphosate was excluded solely because the Parkin study—which was then the best available data—did not include it. *See* Hearing Tr. at 53:6-55:20. And today, Tomasetti has evaluated the evidence and concluded that glyphosate is not a cause of NHL, so the exclusion of glyphosate is also consistent with his considered opinion on that subject. *See* Tomasetti Report at 13–32. While the exclusion of glyphosate from the underlying data may impose limitations on the conclusions that the 95% can be used to support—something discussed in connection with the "fit" analysis below—the methods used to generate it do not offend *Daubert*.

Finally, Delorme-Barton argues that Tomasetti's opinions, even if reliable, will mislead rather than help the jury. She argues, in other words, that Tomasetti's testimony does not "'fit' the question[s] the jury must answer" in this case. Pretrial Order No. 45 at 7 (quoting *Daubert II*, 43 F.3d at 1321 n.17). "In elucidating the 'fit' requirement, the Supreme Court noted that scientific expert testimony carries special dangers to the fact-finding process because it 'can be both powerful and quite misleading because of the difficulty in evaluating it.' . . . Federal judges must therefore exclude proffered scientific evidence under Rules 702 and 403 unless they are convinced that it speaks clearly and directly to an issue in dispute in the case, and that it will not mislead the jury." *Daubert II*, 43 F.3d at 1321 n.17 (citing *Daubert I*, 509 U.S. at 595) (internal citations and some quotation marks omitted).

Whether Delorme-Barton's "fit" argument is correct depends, first, on what sorts of evidence would "logically advance[] a material aspect of" Monsanto's case. Pretrial Order No. 45 at 7 (citing *Messick v. Novartis Pharms. Corp.*, 747 F.3d 1193, 1196 (9th Cir. 2014)). Delorme-Barton takes an excessively narrow view of this question. She is correct that

Tomasetti's random replication error theory and his 95% figure will not independently help the jury decide whether glyphosate causes NHL. Nor can Tomasetti by himself explain what caused Delorme-Barton's NHL. He does not try to do so. But for expert evidence to be relevant under Rule 702, it only needs to support the conclusion that Delorme-Barton has failed to show by a preponderance of the evidence that glyphosate caused her NHL. And Tomasetti's opinions help give meaning to something that experts have largely glossed over in earlier *Roundup* cases. Specifically, experts from both sides have agreed that a large percentage of NHL cases are idiopathic, meaning they lack a known cause. *See* Pretrial Order No. 85 at 4; *see also, e.g.*, *Hardeman* Trial Tr. at 1217:24–1218:11, Dkt. No. 3113. One of Monsanto's attacks on plaintiffs' specific causation experts has been that they fail to adequately rule out idiopathy. And one of the main claims of Monsanto's own specific causation experts has been that a given plaintiff's NHL was likely of idiopathic origin. With Tomasetti, rather than arguing that most NHL has no known cause, Monsanto can argue that random gene replication errors cause most NHL, and it can contextualize that argument within a larger theory of cancer etiology. That will advance Monsanto's case. It will also be helpful to the jury, not least because it replaces the otherwise vague concept of idiopathy with a more positive, substantive explanation.

That said, the core of Delorme-Barton's attack on Tomasetti's "fit" goes to a deeper question: When Tomasetti talks about "mutations," is he really saying anything about causation? The basic point is this. Tomasetti's research conclusions are framed not in terms of the causes of *cases* of cancer, but the causes of *mutations* that cause cancer. This distinction matters, as Tomasetti briefly acknowledged both in his expert report and on direct examination at the *Daubert* hearing. Specifically, Tomasetti disclaimed that his research supports the conclusion that two-thirds of "cancer cases" are attributable to bad luck. Hearing Tr. 47:7–48:10; Tomasetti Report at 12–13. Instead, he said, his research conclusions are about the variation in cancer risk across tissues (presumably referring to the 2015 paper) and the proportion of mutations due to R (presumably referring to the 2017 paper). Hearing Tr. 48:3–10. Importantly, Tomasetti's own publications show that it is not necessarily straightforward to go from speaking in terms of

mutations due to R and speaking in terms of *cancers* due to R. Consider Tomasetti's 2017 paper, which notes that "cancer etiology and cancer preventability . . . are not equivalent." Tomasetti (2017) at 5. The paper then provides the following illustration:

> A cancer in which 50% of the mutations are due to R can still be preventable. The reason for this is that it generally requires more than one mutation to develop the disease. A cancer that required two mutations is still preventable if one of the mutations was due to R and the other due to an avoidable environmental factor.

Id. Given these complications—which Tomasetti certainly does not foreground—one might wonder whether his testimony does, in fact, fit the questions put to the jury in this case. It is fair enough for Tomasetti to speak carefully in terms of "mutations" in the name of scientific precision. But for his testimony to help rather than confuse the jury, there has to be some reliable way to connect his claims about the cause of genetic mutations in NHL to claims about the cause of Delorme-Barton's case of NHL.

Ultimately, the Court is persuaded that this connection can be made. Tomasetti says in his expert report that "[i]n the absence of an exposure to a proven cancer-causing E or H factor it is reasonable to attribute a given cancer to R." *Id.*; *see also* Hearing Tr. 71:7–19 ("[I]n general, unless you find some specific and proven factor, the reality is that the default assumption should be that it was just the natural process of our body that yielded that cancer, and that's what we see in our patients."). Those opinions are consistent with the 2017 paper, which explains that for certain cancers without known environmental causes—the example given is prostate cancer—"a very high fraction of the driver gene mutations . . . can be attributed to R." Tomasetti (2017) at 4. Therefore, the paper says, Tomasetti's findings shed light on the *causes* of those cancers, which had previously been poorly understood. *Id*. At the *Daubert* hearing, Tomasetti also testified that a 2020 publication of his lent independent support to the conclusion that "in the majority of the other cases, you don't need anything else but R factors to get to cancer." Hearing Tr. 60:6–15; *see also* Song (2018) at 1317 ("However, not all cancers are preventable by changes in environment or lifestyle."). In other words, Tomasetti does make claims about causation, including the claim that cancer can be caused by replication errors alone. When those claims are made about cancers

which, like prostate cancer, have few or no well-established environmental causes, the claims are sufficiently grounded in the peer-reviewed literature and do not "involve any logical leaps so great and so lacking in support as to render them inadmissible." Pretrial Order No. 45 at 48 (citing *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)). Tomasetti believes NHL to be a cancer with few well-established environmental causes, so claims about "R" causing NHL cases are sufficiently grounded in Tomasetti's own research and the rest of his testimony.[10]

There are, to be sure, limitations on what Tomasetti's theory can say. And there is a danger that his testimony could be misleading if these limitations are not made clear. Consider Tomasetti's opinion that 95% of mutations in NHL are caused by random replication errors. It would be impermissible for Tomasetti to say that because 95% of mutations in NHL are due to random replication errors, it is therefore very unlikely that glyphosate causes NHL. Because of the way the figure was calculated, that would be question-begging. The conclusion—that glyphosate is not a carcinogen—is an assumption of the argument. It would also be impermissible for Tomasetti to say that, *even if glyphosate were a proven cause of NHL*, there would be a 95% chance that a given case of NHL was the result of random replication errors, *despite any exposure to glyphosate*. That statement would be inconsistent both with his testimony at the *Daubert* hearing and without any evident foundation in his published research. *See* Tomasetti Report at 13 ("*In the absence of an exposure to a proven cancer-causing E or H factor* it is reasonable to attribute a given cancer to R.").

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[10] Part of Delorme-Barton's "fit" argument is that it is impossible to reason from Tomasetti's "population-based conclusions" to an individual case, at least where there is no way to attribute a particular mutation in a patient to a particular cause. *See* Pls. Mot. Exclude at 17–20. If correct, this argument would likely mean that all of Delorme-Barton's own experts would have to be excluded. Their testimony depends on reasoning from "population-based" epidemiological evidence to the conclusion that glyphosate caused a particular patient's cancer, and there are no biomarkers or genetic signatures that can show a given case of NHL (or a given genetic mutation) was caused by glyphosate. *See* Pretrial Order No. 85 at 4. At any rate, the record shows that reasoning from Dr. Tomasetti's population-level analysis is possible and scientifically reliable. Monsanto's specific causation experts can do essentially what Delorme-Barton's specific causation experts will do: reason that (1) random replication errors are a known cause of NHL and that (2) Delorme-Barton had no exposures to other known causes of NHL, and conclude that (3) random replication errors therefore likely caused Delorme-Barton's NHL. *See* Pretrial Order No. 85 at 5.

But Tomasetti never says either of these things. Nor are these impermissible inferences necessary for his testimony to fit the questions put to the jury. That is because Tomasetti's 95% figure *can* reliably be used to show that, in the absence of exposure to a proven environmental or lifestyle carcinogen, there is a very substantial chance that a given case of NHL was the result of random replication errors.

III

Delorme-Barton also moves to exclude two of Monsanto's other experts: Dr. Willis Navarro, M.D. and Dr. Graham Slack, M.D. Both Navarro and Slack offer specific causation opinions about Delorme-Barton's cancer. A large portion of the attacks on Navarro and Slack concern their reliance on Tomasetti's theory. Because those arguments have already been addressed, the Court focuses on the remaining arguments for exclusion.

Navarro is a hematologist and oncologist. Since 2005, he has spent most of his time working in biotechnology and drug development but has continued to spend about 10 percent of his time in clinical practice. Navarro Depo. Tr. at 35:1–12, Greenwald Decl. ISO Mot. Ex. D, Dkt. No. 16944-5. He opines that exposure to glyphosate did not increase Delorme-Barton's risk of NHL or cause her NHL. Expert Report of Dr. Willis Navarro at 18, Greenwald Decl. ISO Mot. Ex. C, Dkt. No. 16944-4 ("Navarro Report"). Instead, he says, Delorme-Barton's NHL "was most likely caused by the accumulation of random and unrepaired genetic events over time, as is the case with most cancers." *Id*.

First, Navarro is qualified to offer the full range of his opinions, despite his more limited clinical practice in recent years. *See Kennedy v. Collagen Corp.*, 161 F.3d 1226, 1231 (9th Cir. 1998) (stating that "[d]isputes as to the strength of [an expert's] credentials . . . go to the weight, not the admissibility, of his testimony") (quoting *McCullock v. H.B. Fuller Co.*, 61 F.3d 1038, 1044 (2d Cir. 1995)) (alteration in original).

Second, Delorme-Barton argues that, while Navarro purports to rely on Tomasetti's research for his conclusions about the cause of her cancer, his deposition testimony revealed that he holds opinions inconsistent with Tomasetti's. When asked to explain the "accumulation of

genetic hits" that he said caused Delorme-Barton's follicular lymphoma, Navarro initially attributed them not to random genetic replication errors but to a variety of "environmental carcinogens":

> By virtue to the fact that we're all exposed to background radiation just from everywhere, we're exposed to environmental carcinogens, barbecued meats; you name it. There are multiple genetic stressors, if you will, things that can potentially impact DNA. They're everywhere; flying in an airplane at altitude increases your exposure to cosmic rays. That constitutes a nontrivial amount of radiation from, from that kind—those types of exposures are ubiquitous, and over time, we, we all experience those as we grow older and multiple hits take place.

Navarro Depo. Tr. at 52:3–13. He then added: "I'm sorry. I just, just to clarify, environmental factors plus just errors that occur over time in DNA repair that we all have." *Id.* This portion of the deposition testimony arguably casts doubt on the reliability of Navarro's analysis, since the first part of his answer is in tension with Tomasetti's views, which he otherwise purports to rely on. On balance, though, this does not warrant exclusion. Navarro can cite and rely on Tomasetti's research without espousing a theory of cancer etiology that is identical to his. Such inconsistencies may be good fodder for cross-examination of Navarro or Tomasetti, and they may persuade the jury to assign less weight to their views. But they do not warrant exclusion.

Delorme-Barton's other arguments also fail. She says that Tomasetti's population-based studies cannot be used to derive conclusions about individual cases, but this is wrong for reasons discussed above in connection with Tomasetti. She argues that Navarro cannot discuss Delorme-Barton's age as a risk factor because his opinion related to age and cancer is not specific to NHL, but to cancer generally. This is not the case. *See* Navarro Report at 17–18. Finally, she faults Navarro for failing to "rule[ ] out every possible environmental factor for the older patients he has treated" because he does not ask his older patients if they have been exposed to glyphosate. But it would be strange for Navarro to ask his patients about glyphosate because, after assessing the scientific literature, he has concluded that glyphosate is not carcinogenic. Navarro's testimony is admissible.

Next is Dr. Graham Slack. Slack is a practicing pathologist at BC Cancer in Vancouver,

British Columbia, and a Clinical Associate Professor of Pathology in the Department of Pathology and Laboratory Medicine at the University of British Columbia. His clinical experience for more than a decade "has been focused exclusively on the diagnosis and pathologic assessment of lymphomas, leukemias, and lymphoproliferative disorders." Expert Report of Dr. Graham Slack at 2, Greenwald Decl. ISO Mot. Ex. D, Dkt. No. 16945-5 ("Slack Report"). Slack opines that, "[l]ike the vast majority of patients with lymphoma, Ms. Delorme-Barton's [NHL] is most likely due to random genetic alterations accumulated over a lifetime." *Id.* at 18. He says that risk factors for Delorme-Barton's cancer "include age and Caucasian race/ethnicity," and that both factors increased her risk of developing NHL. *Id.*

Slack's opinions are largely admissible. Slack's reliance on Tomasetti is unproblematic. As a practicing physician who also conducts research focused "on elucidating the pathologic features and molecular underpinnings of lymphoid malignancies," Slack is qualified to opine about the application of Tomasetti's research to Delorme-Barton's case. Slack Report at 2. In fact, Slack's expert report offers some of the biological details that Delorme-Barton faults Tomasetti for neglecting: it describes the development of follicular lymphoma at the cellular and genetic levels and links this process to the role of random gene replication errors. *See* Slack Report at 4–7. Slack's opinion that age and race are potential risk factors for NHL is also admissible. Delorme-Barton's objection to these opinions—that they cannot be squared with Slack's conclusion about random replication errors causing Delorme-Barton's cancer—go to the weight of the testimony, not its admissibility. *See, e.g., Whitewater W. Indus., Ltd. v. Pac. Surf Designs, Inc.*, No. 3:17-CV-0111-8-BEN-BLM, 2019 WL 2211897, at *11 (S.D. Cal. May 22, 2019).

Daubert does require the exclusion of one aspect of Slack's testimony. His report includes a broad observation about how NHL cases have declined on a per-population basis even as Roundup use has gone up. Slack Report at 10. One could easily imagine a Monsanto expert properly using this information to pound their point home. But neither Slack (in his deposition) nor Monsanto (in its opposition brief) have adequately explained the data behind the assertion

that NHL cases have gone down while Roundup use has gone up, much less grappled with whether other factors (such as the decline in use of other, potentially more dangerous herbicides) could explain the apparent trends. *See* Slack Depo. Tr. at 194:5–195:16, Greenwald Decl. ISO Mot. Ex. A, Dkt. No. 16945-2. Accordingly, Slack may not include discussion of this issue in his testimony. But his opinions are otherwise admissible.

IT IS SO ORDERED.

Dated: January 30, 2024

VINCE CHHABRIA
United States District Judge