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17 **SUPERIOR COURT OF THE STATE OF CALIFORNIA**
18 **COUNTY OF SAN FRANCISCO**

20 DEWAYNE JOHNSON,

21 Plaintiff,

22 vs.

23 MONSANTO COMPANY,

24 Defendant.

Case No. CGC-16-550128

**MEMORANDUM OF POINTS AND
AUTHORITIES IN SUPPORT OF
MONSANTO COMPANY'S MOTION
FOR JUDGMENT NOTWITHSTANDING
THE VERDICT**

Hon. Judge Suzanne R. Bolanos

Hearing Date: October 10, 2018
Time: 2:00 p.m.
Department: 504
Trial Date: June 18, 2018

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1 **INTRODUCTION**

2 Monsanto Company (“Monsanto”) has produced glyphosate-based herbicides (the
3 “Formulation”) in the United States and much of the rest of the world for more than 40 years.
4 These herbicides have a safety record supported by a body of studies more extensive than almost
5 any other chemical in regular use anywhere. The studies include rigorous registration studies—
6 done by multiple registrants including Monsanto—required by U.S. EPA and European regulators
7 to demonstrate the safety of the Formulation, as well as many studies by independent scientists,
8 ranging from small laboratories to the National Cancer Institute. Based on these studies,
9 regulators across the world have concluded on multiple occasions, after multi-year evaluations,
10 that glyphosate is *not* a human carcinogen.

11 Under these circumstances, a jury verdict proclaiming that the Formulation caused
12 Plaintiff’s cancer and that Monsanto’s behavior in relying on the science and the regulators was so
13 egregious as to warrant a \$250 million punitive damages award requires exceptional scrutiny.
14 That extraordinary verdict cannot be sustained for multiple reasons.

15 ***First***, the evidence Plaintiff presented on causation was insufficient as a matter of law to
16 establish the Formulation was a “substantial factor” in causing his cancer. To prove causation,
17 Plaintiff relied on epidemiological studies, animal and mechanistic studies, and a differential
18 diagnosis from a single physician. But Plaintiff and his experts repeatedly admitted that the
19 epidemiology—the most important type of evidence—was “not causal” and that it showed a risk
20 well below the threshold required under California law to establish causation. Plaintiff’s reliance
21 on animal or mechanistic studies was similarly flawed because he presented no evidence that
22 linked the outcomes of any of those studies to human forms of cancer much less that linked the
23 Formulation with his specific form of Non-Hodgkin’s Lymphoma (“NHL”), mycosis fungoides
24 (“MF”). And because it did not account for the extent and timing of exposure to the Formulation,
25 and due to multiple other methodological flaws, the differential diagnosis is legally incapable of
26 supporting causation.

27 ***Second***, the individual legal theories Plaintiff presented fail for various independent
28 reasons. Plaintiff presented his design defect claim to the jury based exclusively on the consumer

1 expectation test, but it does not fit this case as a matter of law. *See Trejo v. Johnson & Johnson,*
2 13 Cal. App. 5th 110, 158-59 (2017). For the failure-to-warn claims, the evidence was insufficient
3 to prove that the “generally recognized and prevailing best scientific and medical knowledge” had
4 established the Formulation had a risk of cancer. *Carlin v. Superior Court*, 13 Cal. 4th 1104,
5 1112, 1116 (1996). Rather, the undisputed evidence disproved the claim: the state of the art
6 reflected in the domestic and foreign regulatory opinions and the scientific literature before and
7 after IARC’s classification is that glyphosate is *not* carcinogenic.

8 ***Third***, the punitive damages award cannot stand. There was no evidence, much less clear
9 and convincing evidence, that Monsanto’s executives or managing agents acted with “malice or
10 oppression” in marketing a product repeatedly certified by competent regulatory authorities as not
11 carcinogenic. Further, there was no evidence that any alleged “despicable” conduct resulted in
12 harm to Plaintiff. For all of these reasons, the Court should grant JNOV in Monsanto’s favor.

13 **ARGUMENT**

14 **I. STANDARD FOR JUDGMENT NOTWITHSTANDING THE VERDICT**

15 A party is entitled to JNOV when “there is no evidence of sufficient substantiality to
16 support” the jury’s verdict. *See Magic Kitchen LLC v. Good Things Int’l, Ltd.*, 153 Cal. App. 4th
17 1144, 1154 (2007); *see also* Cal. Code Civ. Proc. § 629(a). The plaintiff must “produce evidence
18 which supports a logical inference in his favor and which does more than merely permit
19 speculation or conjecture.” *Jones v. Ortho Pharm. Corp.*, 163 Cal. App. 3d 396, 402 (1985)
20 (affirming nonsuit where plaintiff failed to demonstrate medication caused cancer). “Substantial
21 evidence is not synonymous with ‘any’ evidence. To constitute sufficient substantiality to support
22 the verdict, the evidence must be reasonable in nature, credible, and of solid value; it must actually
23 be substantial proof of the essentials which the law requires in a particular case.” *Osborn v. Irwin*
24 *Mem’l Blood Bank*, 5 Cal. App. 4th 234, 284 (1992) (internal citation and quotations omitted).
25 Such evidence was wholly lacking here.

26 **II. THERE WAS NO SUBSTANTIAL EVIDENCE THAT THE FORMULATION**
27 **CAUSED PLAINTIFF’S MYCOSIS FUNGOIDES.**

28 The Court should grant Monsanto JNOV on all of Plaintiff’s claims because there was not

1 substantial evidence to support an essential, common element—that the Formulation was, to a
2 reasonable medical probability, a “substantial factor,” or proximate cause, of his injury. *Trejo*, 13
3 Cal. App. 5th at 110. California law recognizes that causation is “especially troublesome” with
4 cancer because “it is frequently difficult to determine the nature and cause of a particular
5 cancerous growth.” *Jones*, 163 Cal. App. 3d at 403. Given these uncertainties, California law
6 uses special guiderails that prohibit finding liability where causation is merely medically
7 “possible” but does not rise to the level of “reasonable medical probability.” *Id.* “A possible
8 cause only becomes ‘probable’ when, in the absence of other reasonable causal explanations, it
9 becomes *more likely than not* that the injury was the result of its action. This is the outer limit of
10 inference upon which an issue may be submitted to the jury.” *Id.* (emphasis added). Under this
11 standard, a plaintiff does not satisfy the reasonable medical probability standard when the
12 evidence establishes a “less than 50-50 possibility” of causation. *Simmons v. W. Covina Med.*
13 *Clinic*, 212 Cal. App. 3d 696, 702-03 (1989). If the probabilities “are at best evenly balanced, it
14 becomes the duty of the court to direct a verdict for the defendant.” *Jennings v. Palomar*
15 *Pomerado Health Sys., Inc.*, 114 Cal. App. 4th 1108, 1118 (2003) (emphasis in original).

16 Plaintiff’s case for causation included three types of evidence. First, Plaintiff’s expert
17 witnesses testified about various epidemiological studies that purported to link the Formulation or
18 glyphosate to cancer. Second, witnesses described animal and mechanistic studies that attempted
19 to associate glyphosate exposure with rodent tumors or cell damage. Third, based on a differential
20 diagnosis, a single physician testified that Plaintiff’s exposure to the Formulation caused his MF.
21 But Plaintiff’s entire case for causation was flawed from the ground up because he did not present
22 any evidence, much less substantial evidence, that there was more than a 50-50 possibility that the
23 Formulation caused his disease. Plaintiff’s failure to present substantial evidence of causation—
24 general or specific—mandates JNOV in Monsanto’s favor on all claims.

25 **A. Plaintiff’s Epidemiological Studies Are Not Evidence of Causation.**

26 Plaintiff conceded at trial—including in counsel’s closing argument—that the
27 epidemiology does not support causation here. Tr. at 5072:16-20 (“Nobody is saying [the
28

1 epidemiology] gets you there. Nobody.”).¹ Witness after witness agreed with this assessment.
2 Dr. Portier, one of Plaintiff’s experts, candidly testified: **“I can’t conclude it’s causal. . . . The**
3 **effects are small,”** (Tr. at 1964:13, 1965:1-3), **“I can’t really rule out chance,”** “I can’t rule out
4 that there aren’t confounders,” and “you can’t make a firm statement about glyphosate from the
5 epidemiology data alone.” *Id.* at 1965:2-7. Another one of Plaintiff’s experts, Dr. Neugut, agreed
6 that **“the epidemiology alone is not sufficient to show a causal link.”** Tr. at 2679:1-5, 2736:25-
7 2737:3; 2679:1-5. These witnesses were right to affirmatively reject causation. There simply is
8 no epidemiology study that satisfies the legally required standard for causation.

9 The strongest and most current evidence discussed at trial, a 2018 study by the prestigious
10 National Cancer Institute (“NCI Study”), refutes any possible epidemiological basis for causation.
11 An independent, long-term, prospective cohort study that followed over 50,000 pesticide
12 applicators, the NCI Study is the largest and most statistically-powerful epidemiological study and
13 unequivocally found “no association between glyphosate use and NHL overall or any of its
14 subtypes.” Neugut Tr. at 2745:7-13; Portier Tr. at 2357:19-23. The Formulation, the NCI Study
15 found, was slightly *inversely* correlated with NHL (.87 relative risk ratio).

16 Moreover, what little Plaintiff’s experts had to say about the NCI Study could not blunt its
17 ultimate finding, rejecting any association between glyphosate-based products (such as the
18 Formulation) and NHL at any level of exposure and for any subtype. Plaintiff touted a series of
19 older, smaller studies but none of these studies satisfied California’s threshold for causation. An
20 epidemiological study is not competent evidence of causation unless it shows “a relative risk
21 greater than 2.0.” *Cooper v. Takeda Pharms. Am, Inc.*, 239 Cal. App. 4th 555, 593 (2015). This
22 threshold ensures that there is at least a “50% probability that the agent at issue was responsible
23 for a particular individual’s disease.” *Id.* In contrast, when the relative risk is “less than two” it
24 “actually tends to disprove legal causation, as it shows that [the product] does not double the
25 likelihood of [disease].” *Daubert v. Merrell Dow. Pharm., Inc.*, 43 F.3d 1311, 1321 (9th Cir.

26 _____
27 ¹ All trial transcripts, deposition transcripts, exhibits, and orders cited herein are attached for the
28 Court’s convenience to the Declaration of Sandra A. Edwards in Support of Defendant Monsanto
Company’s Motion for New Trial and Motion for Judgment Notwithstanding the Verdict.

1 1995);² *In re Johnson & Johnson Talcum Powder Cases*, 2017 WL 4780572, at *14 (Cal. Super.
2 Ct. 2017) (“*Talcum Order*”).³

3 Plaintiff’s epidemiology evidence did not meet this standard. Plaintiff’s expert Dr.
4 Neugut—Plaintiff’s only certified epidemiologist—testified that the risk ratio from epidemiology
5 studies he examined fell below the mandated 2.0 ratio. The best he could do was to testify that the
6 risk ratio from case control studies fell in a range between 1.3 and “possibly 1.5.” Tr. at 2614:17-
7 21 (emphasis added). Although he attempted to characterize this as “a statistically significant
8 increased risk,” *id.*, Dr. Neugut’s bottom-line concession that the risk ratio was less than 2.0 is all
9 that matters under California law. *See Simmons*, 212 Cal. App. 3d at 702-703 (“A less than 50-50
10 possibility that defendant’s omission caused the harm does not meet the requisite reasonable
11 medical probability test of proximate cause.”); *Daubert*, 43 F.3d at 1321 (“A relative risk of less
12 than two ... actually tends to disprove legal causation....”).

13 The small size of these studies, especially as compared to the NCI Study, is yet another
14 reason to doubt their value, especially as compared to the NCI Study. Of the case-control studies
15 Dr. Neugut presented to the jury, Hardell had 8 exposed cases,⁴ Orsi had 12, Eriksson had 29, De
16 Roos (2003) had 36, and McDuffie had 51.⁵ By contrast, the NCI Study, which found no

18 ² Whether addressing this issue in the context of expert admissibility or sufficiency of the
19 evidence, numerous federal and state courts in and outside of California have agreed that a relative
20 risk of 2.0 or greater is necessary for epidemiological evidence to be probative of causation. *See*
21 *Daubert*, 43 F.3d at 1321 (holding under California law that expert testimony was inadmissible
22 where epidemiology did not support a 2.0 risk); *Merck & Co., Inc. v. Garza*, 347 S.W.3d 256
23 (Tex. 2011) (vacating judgment for insufficient evidence and holding “when parties attempt to
24 prove general causation using epidemiological evidence, a threshold requirement of reliability is
25 that the evidence demonstrate a statistically significant doubling of the risk”); *see also In re*
26 *Lipitor*, 150 F. Supp. 3d 644, 650 (D.S.C. 2015); *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217,
27 1225-28 (D. Colo. 1998); *Sanderson v. IFF*, 950 F. Supp. 981, 1000 (C.D. Cal. 1996); *Hall v.*
28 *Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1403 (D. Ore. 1996).

³ California Rules of Court 8.1115 permits citation to persuasive trial court orders such as this one.

⁴ Exposed cases are an important metric of the statistical power of the studies and allow one to compare the relative sizes of different studies. Neugut Tr. at 2686:21-25.

⁵ Neugut Tr. at 2690:21-23; 2692:4-13, 2695:8-2698:7; *see also* Mucci Tr. at 4238:12-18 (describing number of exposed cases in Hardell as “quite low”), 4248: 9-17 (Eriksson had “only 29 exposed cases”), 4247: 7-14 (Orsi had “very small number of exposed cases, only 12”), 4246:20-22 (De Roos 2003 “by pooling together these three studies, they had 36 exposed cases,

1 association between the Formulation and NHL, had 440 exposed cases. Mucci Tr. at 4285:21-
2 4286:9. It is thus 8.6 times larger than McDuffie (Plaintiff’s largest case-control study), and 55
3 times larger than Hardell (Plaintiff’s smallest). It is more than three times larger than all of the
4 case-control studies *put together*. Neugut Tr. at 2690:21-23, 2692:4, 2695:8-2698:7; *see also*
5 Mucci Tr. at 4238:12-18, 4248: 9-17, 4247: 7-14, 4246:20-22, 4242:3-8.

6 There are still more reasons why the small studies Plaintiff’s counsel relied upon do not
7 constitute substantial evidence of causation. Reflecting on their small size and lack of power, all
8 of the case-control studies Plaintiff relied on presented enormous confidence intervals, and in the
9 analyses adjusted for other pesticides that Dr.Neugut presented to the jury, none yielded
10 statistically significant results. Tr. at 2702:25-2703:3; 2682:10-15; (Hardell: 1.85 (.55 to 6.2);
11 Orsi: 1.0 (.5 to 2.2); Eriksson: 1.51 (.77 to 2.94); De Roos (2003) 1.6 (.9 to 2.8); McDuffie 1.2
12 (0.83 to 1.74).⁶ Because Dr.Neugut admitted that a statistically significant increased risk was
13 essential to determine whether there is a causal association (Tr. at 2685:4-8), the lack of statistical
14 significance effectively ended Plaintiff’s epidemiology argument. *See, e.g., McMunn v. Babcock*
15 *& Wilcox Power Generation Grp., Inc.*, 2013 WL 3487560, at *15 (W.D. Pa. July 12, 2013)
16 (“Step one looks to whether there is a statistically significant association between a substance and
17 a specific disease.... If no association between the exposure and the disease is supported by the
18 scientific literature, there is no basis to find a causal relationship exists and the analysis should end
19 there.”); *In re Lipitor Mktg.*, 174 F. Supp. 3d 911, 924-925 (D.S.C. 2016) (collecting cases). As
20 shown in the confidence intervals, none of these point estimates was statistically significant,
21 meaning that chance could not be ruled out as the explanation for any apparent association,

22 _____
23 so, again, not a really large study”); 4242:3-8 (McDuffie was “larger than Hardell, [but] still a
fairly small number of exposed cases.”).

24 ⁶ Dr. Portier agreed: Tr. at 1878:12-15 (McDuffie 2001: “It’s clearly not a statistically significant
25 result.”); 1885:17-22 (Hardell 2002: “The second [line] is adjusted for pesticides, other pesticide
26 use. . . . And there you see it’s clearly not statistically significant”); 1887:3-6 (DeRoos 2003:
27 “The second method of analysis of the same data but a different method of analysis, using what’s
28 called Bayesian statistics, shows a positive finding but not statistically significant.”); 1895:13-20
(Eriksson 2008: “A. If I remember correctly, I think they did two different types of analyses, but,
yes, they adjusted for other pesticides. Q. And what happens to the risk ratio when they did that?
A. It dropped. It drops to 1.5, and it’s no longer statistically significant.”); 1898:19-21 (Orsi 2009:
“Q. And it has a ratio of .5 and 2.2, so it’s not statistically significant? A. Correct.”).

1 making them meaningless for Plaintiff’s case. Neugut Tr. at 2702:25- 2703:3 (“Q. On the Forest
2 plot that you presented to the jury, there was no statistically significant result. A. Correct.”). *See*
3 *e.g., Dunn v. Sandoz Pharm. Corp.*, 275 F. Supp. 2d 672, 681 (M.D.N.C. 2003) (“Statistically
4 insignificant results do not constitute proof that Parlodel causes stroke.”); *Caraker v. Sandoz*
5 *Pharm. Corp.*, 188 F. Supp. 2d 1026, 1034 (S.D. Ill. 2001) (“This Court, however, rejects the
6 plaintiffs’ experts’ opinions inasmuch as they rely on selective use of statistically insignificant
7 data from epidemiological studies.”).

8 Moreover, when the small studies Plaintiff relied on were pooled together, they showed a
9 lower relative risk ratio. The NAPP study—a pooled study of North American case control
10 studies (including the De Roos (2003) data)—showed an even lower, non-statistically significant
11 risk ratio of **1.13** (CI = 0.84 to 1.51), which was further reduced to **0.95** (CI = 0.69 to 1.32) when
12 proxy respondents were eliminated. Portier Tr. at 2451:3-5; 2338:24-2339:7. And meta-analysis
13 of the epidemiology studies on which Plaintiff’s experts relied—which did not include either the
14 NAPP or NCI studies—still demonstrated a risk ratio of only **1.3**. Neugut Tr. at 2613:6-12;
15 Portier Tr. at 1914:5-8. That is too low to establish causation.

16 Finally, Monsanto anticipates Plaintiff may point to alleged “dose-response” findings in
17 McDuffie and Eriksson in which estimates above 2.0 were calculated at certain exposure levels.
18 Apart from being inconsistent with Dr. Neugut’s actual conclusion, these publications do not aid
19 Plaintiff for multiple reasons. *See, infra* at 12-14. These data are (1) confounded by other
20 pesticides, contrary to the requirements of California law, *see In re Lockheed Litigation Cases*, 23
21 Cal. Rptr. 3d 762, 777 (2005), *rev. dismissed by* 83 Cal. Rptr. 3d 478; (2) inconsistent with later
22 and more comprehensive analyses in the NAPP and NCI Study showing no increased risk
23 whatsoever even at the highest dose levels (Portier Tr. at 1914:5-8, 2451:3-5, 2338:24-2339:7;
24 Neugut Tr. at 2613:6-12); and (3) insufficient under California law, which looks at the
25 epidemiology evidence overall and do not find substantial evidence of causation based on an
26 isolated data point above 2.0. *Talcum Order*, at *14.

27
28

1 **B. The Animal and Genotoxicity Studies Are Not Evidence to Support Causation**
2 **as a Matter of Law.**

3 Plaintiff also relied on animal and genotoxicity studies to prove causation but the evidence
4 about these studies fell short in multiple ways.

5 **No evidence tying animal studies to disease in humans or Plaintiff.** Plaintiff’s reliance
6 on animal studies could not establish causation because none of his witnesses ever linked the
7 results in any way to cancer in humans or to NHL or MF—the studies were legally irrelevant and
8 not evidence of causation. Indeed, missing from Plaintiff’s case was evidence that addressed
9 either of the extrapolation problems associated with animal studies: species extrapolation, which
10 is the “differences between human beings and the animals studied,” and dosage extrapolation,
11 which is “how to extrapolate from the high doses given to animals to the lower doses to which
12 human beings may be subjected.” *Lockheed Litig. Cases*, 23 Cal. Rptr. 3d at 779; *see also*
13 *General Electric Co. v. Joiner*, 522 U.S. 136, 144 (1997) (rejecting use of animal studies to prove
14 causation in humans because doses, routes of exposure, and types of cancer all differed); *Redfoot*
15 *v. B.F. Ascher & Co.*, 2007 WL 1593239, at *11, n.18 (N.D. Cal. June 1, 2007) (“Extrapolations
16 of animal studies to human beings are generally not considered reliable in the absence of a
17 scientific explanation of why such extrapolation is warranted.”).

18 Dr. Portier’s testimony highlights this evidentiary void. He testified about his analysis of
19 various rodent tumors (which the worldwide regulatory bodies rejected) but offered no reliable
20 extrapolation of that analysis to any form of cancer in humans. Without this missing evidence, the
21 jury had no way to know if the same mechanisms associated with rodent tumors exist in humans
22 and, if so, what exposure levels are required to produce the same outcome. In fact, one of
23 Plaintiff’s experts even admitted that some scientific, epidemiological basis must exist before
24 animal studies can support a finding of causation in humans. Sawyer Tr. at 3683:13-17. There
25 was no such evidence here.

26 Plaintiff also offered no evidence that linked any of the animal studies with his specific
27 cancer, MF. Again, Dr. Portier’s testimony is instructive. He testified about an old study
28 involving mice and kidney tumors but never provided any link between that type of mouse tumor

1 and MF in humans. *See Joiner*, 522 U.S. at 144 (rejecting animal data as showing cancer where,
2 *inter alia*, animals developed different type of tumors than plaintiff). While Dr. Portier also
3 testified about mice lymphomas, the evidence did not even support extrapolating the lymphoma
4 results from male to female mice or even to another species of animal—rats—which are
5 indisputably more biologically similar to mice than humans. Tr. at 2200:13-15; 2151:16-2155:1;
6 Foster Tr. at 4539:1-4540:7. Whether the same studies can be extrapolated to MF in humans was
7 left to pure guesswork.

8 Equally unavailing was Plaintiff’s reliance on the so-called “tumor promotion” or 2010
9 George study. Again, the study was irrelevant to causation because none of Plaintiff’s experts
10 attempted to extrapolate the results—which purported to show *non-cancerous*⁷ “tumors” in rats—
11 to the occurrence of NHL or MF in humans.⁸ In any event, the study is also well known for its
12 flaws, leading every reviewing agency and scientific body (as well as IARC) to dismiss it as
13 unreliable. Portier Tr. at 1863:21-25.⁹

14 **Mechanism studies not linked to human outcomes.** The mechanism studies Plaintiff
15 introduced are similarly irrelevant to causation. The reason for this is that mechanism studies are
16 one step further removed from what Plaintiff must prove to establish causation: these studies only
17 purport to show possible pathways by which a substance might cause cancer. As Dr. Portier
18 explained, genotoxicity does not show that a substance can cause cancer generally because a
19 genotoxic compound “may not lead to critical mutations that are important for carcinogenesis.”
20 Tr. at 2257:10-20; 2258:15-17. Plaintiff’s experts, however, made no claim that his MF was
21 caused by the types of effects (e.g., “oxidative stress”) allegedly seen in high-dose cell testing
22 systems used in the mechanistic studies. This is unsurprising considering it was undisputed at trial
23 that MF is likely of unknown or epigenetic origin. Kuzel Tr. at 4790:3-4. If no one knows

24 _____
25 ⁷ Dr. Portier admitted they were *benign* skin growths, not carcinomas. Tr. at 1863:3-12, 2229:13-
2230:6.

26 ⁸ As set forth in Monsanto’s Motion for New Trial, both IARC and EPA declined to use the
27 George study, finding it to be “poor” and “inadequate in protocol, conduct or reporting.” *See*
Motion for New Trial at 13-14.

28 ⁹ *See* PX 784 at p. 34 (IARC); DX 2481 at 0070 (EPA).

1 precisely what causes MF, it logically follows that Plaintiff’s experts could not, and did not, link
2 the mechanism studies to MF.

3 * * * * *

4 Plaintiff’s reliance on animal and mechanistic studies lacked any evidence of the critical
5 bridge to human outcomes that was necessary for them to prove causation under California law.
6 Without this evidence, the jury was improperly allowed to make its own extrapolation and fill in
7 the missing link to causation that Plaintiff failed to provide. *See Domingo ex rel. Domingo v. T.K.*,
8 289 F.3d 600, 606 (9th Cir. 2002) (excluding expert testimony where proffered opinion did not
9 provide sufficient support to extrapolate animal studies to humans).

10 **C. Dr. Nabhan’s Differential Diagnosis Was Insufficient To Establish Causation.**

11 Plaintiff’s final piece of causation evidence was the differential diagnosis offered by Dr.
12 Nabhan, the only expert who attempted to link Plaintiff’s MF specifically with the Formulation.
13 For his differential diagnosis, Dr. Nabhan purported to “rule in” the plausible causes of Plaintiff’s
14 MF and then “ruled out” the least plausible causes of Plaintiff’s MF, so he was left with only
15 Plaintiff’s race and his exposure to the Formulation. Tr. at 2853:19-2854:2. *See Cooper*, 239 Cal.
16 App. 4th at 593-94 (explaining differential diagnosis). But there is every reason to doubt Dr.
17 Nabhan’s conclusion considering his concession that Plaintiff “could well be someone who would
18 have developed mycosis fungoides when he did, whether he was exposed to glyphosate or not.”
19 Tr. at 3002:21-3003:4. Given this testimony, it is perhaps unsurprising that Dr. Nabhan’s
20 differential diagnosis was legally flawed at every step: he did not properly rule in the Formulation
21 as a potential cause of Plaintiff’s MF, he failed to rule out unknown causes of MF, and he entirely
22 ignored the timing and extent of Plaintiff’s exposure.

23 **1. Dr. Nabhan Had No Scientific Basis To Rule In the Formulation as a**
24 **Possible Cause.**

25 Given the lack of any scientific basis for concluding that the Formulation causes cancer
26 (discussed above), there was no scientific basis for Dr. Nabhan to “rule in” the Formulation as a
27 potential cause. None of the putative bases Dr. Nabhan offered for doing so are legally sufficient.
28

1 conclusion about what dose of glyphosate could possibly cause cancer in humans. *Id.* at 2673:5-8
2 (“Q. . . . IARC reached no conclusion about a dose of glyphosate that could cause cancer in
3 humans, right? A. I don’t think they typically do that.”). IARC’s self-described limitations in
4 the Monograph preclude it from being used to determine that Plaintiff’s exposure to the
5 Formulation was a plausible cause of Plaintiff’s MF.

6 **b. Dr. Nabhan’s Cherry-Picked Epidemiology Does Not Establish**
7 **Specific Causation.**

8 Dr. Nabhan’s other basis for “ruling in” glyphosate involved plucking three data points
9 from various studies—which he claimed showed a “doubling” of the risk—while ignoring all
10 contrary data, including the conclusions of Plaintiff’s actual epidemiologists. This methodology is
11 also legally insufficient to support “ruling in” glyphosate.

12 Two of the three data points Dr. Nabhan selected—from the Eriksson and McDuffie
13 studies—were legally incapable of evidencing causation because they were not adjusted for other
14 pesticide use at all. A study that analyzes chemicals together and without distinguishing between
15 them cannot logically, or legally, prove that any particular chemical causes cancer. *In re Lockheed*
16 *Litig. Cases*, 23 Cal. Rptr. 3d at 774 (“We conclude that the multiple-solvent studies provide no
17 reasonable basis for an opinion that any of the solvents here at issue can cause disease.”); *see also*
18 *In re Roundup*, 2018 WL 3368534, at *8 (explaining that reliable epidemiology must determine
19 whether the study adequately considered confounding variables and possible sources of bias, and
20 “[o]ne important possible source of confounding in the studies relevant here is exposure to other
21 pesticides”). Yet despite admitting the importance of separating out and adjusting for other
22 factors,¹¹ (Nabhan Tr. at 2917:13-16), the risk ratios Dr. Nabhan relied on from the McDuffie and
23 Eriksson studies were *not* adjusted for other pesticides. Tr. at 2915:7-12 (McDuffie); 2917:8-10,
24 2918:10-12 (Eriksson).¹² In the case of Eriksson, Dr. Nabhan chose to present unadjusted data
25

26 ¹¹ All of Plaintiff’s experts who discussed epidemiology testified about the problem of
27 confounding and the importance of adjusting for other factors. Neugut Tr. at 2679:1-5; Portier Tr.
at 1964:23-25; Nabhan Tr. at 2922:5-2924:17.

28 ¹² Tr. at 2913:11-15 (“Q. Okay. And, Doctor, this McDuffie study, it does not adjust for -- let me

1 despite the fact that the same paper had presented another analysis (“multivariate” analysis) that
2 was at least partially adjusted for other pesticides,¹³ and found a non-statistically significant risk
3 ratio of 1.51. Portier Tr. at 1895:13-20.¹⁴ Dr. Nabhan’s reliance on this unadjusted data was
4 litigation advocacy and “junk science,” as Judge Chhabria observed in conjunction with the
5 federal MDL proceedings. 3/14/18 MDL Hearing on Daubert Mot at 54:3-5.

6 For his third data point—De Roos (2003)—the authors published *both* logistical and
7 hierarchical regression analyses. According to the authors, the goal of the hierarchical regression
8 analysis was to *decrease false positives*, and they found a non-statistically significant risk ratio of
9 1.6 using that method. Portier Tr. at 1887:3-6; Nabhan Tr. at 2826, 2935:20-22. Nevertheless, Dr.
10 Nabhan chose the “logistic regression” number—despite the fact that Plaintiff’s own
11 epidemiology expert (Dr. Neugut) used the “hierarchical” regression number. Nabhan Tr. at
12 2928:18-25; Neugut Tr. at 2693:9-22. Dr. Nabhan also ignored that the data from De Roos (2003)
13 was included into the larger NAPP dataset, and then showed an attenuated 1.13 RR (0.95 when
14 adjusted for proxies), which was not statistically significant. And, perhaps most egregiously, he
15 ignored that the De Roos (2003) paper stated a chemical-specific study was necessary, and then
16 further ignored that very study (the NCI study), (Tr. at 2936:2-23), which concluded that
17 “glyphosate exposure was not associated with cancer incidents overall.” Tr. at 2942:12-2943:4.¹⁵

18 In short, cherry-picked data that ignores most of the epidemiology evidence, including the
19 largest study (the NCI Study), is not competent evidence to “rule in” glyphosate as a potential
20 cause, particularly where Dr. Nabhan’s testimony is contrary to the conclusions of the actual

21 _____
22 just say this: There's no control for other pesticides in this study; is that right? A. Not that I'm
aware of.”); *id.* at 2917:4-10, 2918:10-14; 2919:6- 2920:15.

23 ¹³ Nabhan Tr. at 2920:11-15 (“Q. And, actually, Doctor, you didn't tell the jury this morning, but
24 when you do the multi-variate analysis, you get no statistically significant result for glyphosate;
isn't that right? A. In this paper, that is correct.”).

25 ¹⁴ Even before adjusting for other pesticides, the risk ratio cited by Dr. Nabhan from Eriksson was
not statistically significant. Nabhan Tr. at 2828:15-20 (Eriksson).

26 ¹⁵ In addition, none of these studies looked specifically at MF. Thus, as in the *Talcum* case, these
27 studies do not support ruling in the Formulation as a cause of *Plaintiff’s* illness. *Talcum* Order at
*13-14. As Dr. Kuzel testified—unrebutted—the causes and risk factors for each subset of NHL
28 are different, and there are known causes for some subtypes that are known not to be causes for
others. Tr. at 4731:19-4734:20; 4742:10-15.

1 epidemiology experts Plaintiff called to testify. Dr. Nabhan’s testimony thus compares
2 unfavorably even with the testimony the Court in the *Talcum* case ruled insufficient as a matter of
3 law. *Talcum* Order at *14 (although expert “ruled in” talc as basis for differential diagnosis based
4 on two epidemiology studies showing relative risk above 2.0, testimony was insufficient because
5 epidemiology did not support that conclusion and did not establish causation for specific subtype
6 of plaintiff’s cancer).

7 **2. Dr. Nabhan Failed To Rule Out Idiopathic Causes.**

8 Dr. Nabhan’s “differential diagnosis” is legally inadequate to prove causation for the
9 additional reason that he failed to rule out idiopathic causes—which constitute a *majority* of NHL
10 causes—as potential causes for Plaintiff’s cancer.

11 A differential diagnosis cannot support a finding of specific causation where the majority
12 of the instances of the disease are of unknown origin. In *Hall v. Conoco Inc.*, for example, the
13 Tenth Circuit found that “because the evidence had pointed to idiopathic causes in most cases of
14 acute myeloid leukemia,” “the district court could reasonably view the failure to rule out
15 idiopathic causes as a fatal error tainting the differential diagnosis.” 886 F.3d 1308, 1314 (10th
16 Cir. 2018). Likewise, in *Tamraz v. Lincoln Elec. Co.*, 620 F.3d 665, 675 (6th Cir. 2010), the Sixth
17 Circuit reversed admission of “differential diagnosis” testimony where idiopathic causation
18 “currently accounts for the vast majority of Parkinson’s Disease cases, making it impossible to
19 ignore and difficult to rule out.” In *Bland v. Verizon Wireless*, 538 F.3d 893, 897 (8th Cir. 2008),
20 the Eighth Circuit found that “[w]here the cause of the condition is unknown in the majority of
21 cases, an expert cannot properly conclude, based upon a differential diagnosis,” the plaintiff’s
22 “exposure to freon was ‘the most probable cause’ of [his]exercise-induced asthma.”¹⁶ While
23 “California has rejected the notion that an expert must ‘exclude *all* possibilities’ in reaching a
24 specific causation opinion,” the expert must do so when there is “substantial evidence of an

25 _____
26 ¹⁶ See *Hall*, 886 F.3d at 1314-15 (collecting many cases); *Restatement (Third) of Torts* § 28 cmt 4
27 (2010) (noting that differential diagnosis is “most useful when the causes of a substantial
28 proportion of the disease are known When the causes of a disease are largely unknown,
however, differential etiology is of little assistance”); Federal Judicial Center’s *Reference Manual
on Scientific Evidence*, at p. 618 (“[F]or diseases for which the causes are largely unknown . . . a
differential etiology is of little benefit.”).

1 alternative explanation for the disease.” *Cooper*, 239 Cal. App. 4th at 585-586.¹⁷

2 The evidence here was undisputed and indisputable: NHL generally, and MF specifically,
3 is an idiopathic cancer. Dr. Nabhan repeatedly admitted that the cause of *the majority* of NHL
4 cases is idiopathic. Tr. at 2990:6-14; 2812:8-10; 2997:17-23; 2998:16-21. And the evidence of
5 Drs. Kuzel and Kim that the causes of MF specifically are *entirely* idiopathic was also undisputed.
6 Kuzel Tr. at 4790:3-4; Nabhan Tr. at 2994:21-2996:1. Dr. Kim—Plaintiff’s own physician and a
7 world renowned expert in MF—testified: “But right now, the scientific fact –not my opinion, the
8 scientific fact is that so far there is no established cause for this particular rare disease.” Nabhan
9 Tr. at 2995: 12-14. Dr. Kuzel likewise testified, “I would say every case of mycosis fungoides is
10 of unknown etiology.” Tr. at 4790:3-4.

11 As *Hall, Tamraz*, and numerous other cases make clear, Dr. Nabhan could not possibly rule
12 out unknown causes for NHL and MF when the majority of cases are of idiopathic origin. And Dr.
13 Nabhan did not even try. Instead, he made a speculative leap from Plaintiff’s exposure to the
14 Formulation before his MF diagnosis to the unsupported conclusion that this exposure must have
15 been the cause. A differential diagnosis that does not rule out causes of unknown origin when it is
16 undisputed that they constitute a majority of the cases is speculative and cannot prove causation.
17 *See Talcum* Order, at *15 (finding plaintiff’s expert’s testimony on specific causation to be “mere
18 speculation” based, in part, on expert’s testimony that it was “probable” the cause of plaintiff’s
19 cancer was unknown).

20 **3. Dr. Nabhan Did Not Consider Plaintiff’s Exposure or Latency.**

21 Finally, Dr. Nabhan’s differential diagnosis also failed to establish specific causation
22 because Dr. Nabhan did not consider (1) Plaintiff’s exposure level, and (2) whether it was possible
23 for Plaintiff to have contracted MF within one to two years after his first exposure.

24 Dr. Nabhan, like Plaintiff’s other experts, admitted that “minimal exposure may not be that
25 significant” in causing NHL. Tr. at 2835:3-10. It follows that Dr. Nabhan could not rule in the
26

27 ¹⁷ In *Cooper*, the Court rejected the challenge to the differential diagnosis because the defendant
28 had only raised a “bare conceivability of another possible cause,” not substantial evidence of one.
239 Cal. App. 4th at 585-586. There is substantial evidence of an idiopathic cause here.

1 Formulation unless he considered what level of exposure *is* significant in causing NHL and
2 whether Plaintiff was subjected to that amount of exposure. But Dr. Nabhan did not opine on
3 either of these issues. *Id.*; see *Henricksen v. Conoco Phillips Co.*, 605 F. Supp. 2d 1142, 1161-62
4 (E.D. Wash. 2009) (excluding specific-causation opinion as inherently unreliable where all experts
5 agreed that benzene-induced diseases are dose dependent, yet specific-causation expert failed “to
6 analyze or evaluate (his own *or any other expert’s*)¹⁸ information pertaining to dose or the actual
7 level of Plaintiff’s exposure.”).

8 Dr. Nabhan also testified that he had no data on latency and no opinion about it. Tr. at
9 3010:22-3011:1. The most he could muster was that: “I think it’s very difficult to pinpoint a
10 particular duration, but I would say—I would struggle—or have I would have a tough time linking
11 both together if the lag time was less than a year.” Tr. at 3043:19-3044:8. In contrast, Dr. Kuzel
12 testified—again unrebutted—that MF takes “years” to develop biologically. Tr. at 4748:5-14.
13 Without competent evidence that Plaintiff’s MF could have been caused in the short period
14 between when he was first exposed to the Formulation and the onset of his disease (at most 18
15 months), Dr. Nabhan should not have been allowed to “rule in” the Formulation as a cause of
16 Plaintiff’s MF in his differential diagnosis.

17 **D. Dr. Sawyer Did Not Opine on Specific Causation and His Testimony Does Not**
18 **Demonstrate that the Formulation Was a Substantial Cause of Plaintiff’s MF.**

19 Dr. Sawyer’s testimony was also insufficient to prove causation. He never actually
20 testified that the Formulation substantially contributed to Plaintiff’s MF. In fact, when asked
21 about causation, Dr. Sawyer changed the subject and responded that Plaintiff was “heavily
22 exposed” to the Formulation. Tr. at 3596:15-3597:4. But the level of exposure on its own does
23 not necessarily mean anything about causation. To be sure, Dr. Sawyer later testified that
24 Plaintiff’s exposure “puts him approximately in the middle of the human epidemiologic studies
25 that show human cancer.” Tr. at 3674:25-3675:16. But those are the same studies that do not
26 _____

27 ¹⁸ Although Dr. Sawyer testified that Plaintiff’s exposure was theoretically sufficient to cause
28 NHL, Dr. Nabhan did not rely on that opinion—or the opinion of any other of Plaintiff’s experts.
Tr. at 3674:25-3675:16; *see also Talcum Order*, at *14.

1 meet the legal threshold under California law to establish causation. Plaintiff cannot bootstrap
2 these inadequate studies with Dr. Sawyer’s testimony to reach causation especially when
3 Plaintiff’s counsel and experts agree the epidemiology is not sufficient to establish causation.

4 **III. PLAINTIFF’S CLAIMS FAIL AS A MATTER OF LAW ON MULTIPLE**
5 **INDEPENDENT GROUNDS.**

6 Aside from failing to prove causation, each of Plaintiff’s legal theories fails as a matter of
7 law for other legal reasons. First, the design defect claim fails because Plaintiff chose to present a
8 legal theory (“consumer expectation”) that does not apply to this case as a matter of law and for
9 which he presented no evidence. Second, the failure-to-warn claims fail because the evidence
10 affirmatively refuted any duty to warn, given the undisputed state of the best prevailing scientific
11 and medical knowledge.

12 **A. Plaintiff’s Design Defect Claim Fails as Matter of Law.**

13 “A bedrock principle in strict liability law requires that the plaintiff’s injury must have
14 been caused by a ‘defect’ in the [defendant’s] product.” *O’Neil v. Crane Co.*, 53 Cal. 4th 335, 347
15 (2012). “A design defect exists when the product is built in accordance with its intended
16 specifications, but the design itself is inherently defective.” *Trejo*, 13 Cal. App. 5th at 142. In this
17 case, Plaintiff elected to pursue its claim only under the consumer-expectation test for design
18 defect, despite Monsanto’s urging that the risk-benefit test was the only one that could possibly fit
19 the case.¹⁹ Plaintiff’s “consumer expectation” theory fails for two reasons: first, the consumer
20 expectation test does not, as a matter of law, apply in these circumstances; and second, there was
21 no substantial evidence from which a jury could reach a verdict even under that theory.

22 **1. The Consumer-Expectation Test Was Not Appropriate as a Matter of**
23 **Law.**

24 The consumer-expectation test is “reserved for cases in which the everyday experience of
25 the product’s users permits a conclusion that the product’s design violated minimum safety
26 assumptions.” *Trejo*, 13 Cal. App. 5th at 156. “[W]hen the ultimate issue of design defect calls
27 _____

28 ¹⁹ Tr. at 4893:13-21, 4900:8-4904:17; Pl.’s Proposed Substantive Jury Instrs. at 1 (May 8, 2018).

1 for a careful assessment of feasibility, practicality, risk, and benefit, the case should not be
2 resolved simply on the basis of ordinary consumer expectations.” *Id.* In a jury case, “the trial
3 court must initially determine as a question of foundation, within the context of the facts and
4 circumstances of the particular case, whether the product is one about which the ordinary
5 consumer can form reasonable minimum safety expectations.” *Saller v. Crown Cork & Seal Co.,*
6 *Inc.*, 187 Cal. App. 4th 1220, 1233 (2010).

7 In *Trejo*, the court rejected the plaintiff’s assertion that the consumer-expectation test
8 should apply to his developing Stevens-Johnsons Syndrome from taking over-the-counter Motrin,
9 since “it could be said that any injury from the intended or foreseeable use of a product is not
10 expected by the ordinary consumer. If this were the end of the inquiry, the consumer expectation
11 test always would apply and every product would be found to have a design defect.” 13 Cal. App.
12 5th at 158-59. Given that the claim before it involved technical detail and medical testimony, the
13 court held the consumer expectation test inapplicable. *Id.*; *see also Morson v. Superior Court*, 90
14 Cal. App. 4th 775 (2001). *See Trejo*, 13 Cal. App. 5th at 159 (noting that four other California
15 cases “indicate that the consumer expectation test does not apply merely because the consumer
16 states that he or she did not expect to be injured by the product”). In *Morson*, the Court
17 recognized “the special problem here of reconciling products liability law that has developed in
18 the context of merchandise, such as soda bottles and automobiles, with the body of knowledge that
19 deals with medical and allergic conditions and their genesis.” 90 Cal. App. 4th at 791. The court
20 further acknowledged that “the consumer expectation test can be applied even to very complex
21 products, *but only where the circumstances of the product’s failure are relatively*
22 *straightforward.*” *Id.* at 792 (example of defective automobile exploding while idling at
23 stoplight). The consumer-expectation test is *not* helpful when “the alleged circumstances of the
24 product’s failure involve technical and mechanical details about the operation of the
25 manufacturing process, and then the effect of the product upon an individual’s health.” *Id.*

26 Like *Trejo* and *Morson*, Plaintiff’s claims turn on complex scientific details about how the
27 Formulation works *and* expert testimony about the “effect of the product upon [Plaintiff’s]
28 health.” *See id.* As *Trejo* makes clear, the consumer expectation test does not apply simply

1 because Plaintiff did not expect the Formulation to give him cancer. That would create an
2 exception to swallow the rule, and the applicability of the consumer expectation test would be
3 unbounded. Plaintiff’s own evidence that the Formulation—like Motrin in *Trejo*—can be used
4 safely reinforces that the test does not apply. Dr. Sawyer testified that the Formulation is safe to
5 use and he has safely used it for decades. Tr. at 3601:14, 3602:10. This testimony makes the
6 issue of whether a design defect exists complicated, as it shows that there are many factors that
7 come into play as to when and how the product can be used safely. In short, the ultimate issue of
8 design defect in this case “calls for a careful assessment of feasibility, practicality, risk, and
9 benefit,” not for an assessment of consumer expectations. *Trejo*, 13 Cal. App. 5th at 159. The
10 extensive expert testimony presented in this case should be enough on its own to establish that the
11 ordinary consumer could not reasonably appreciate the complex scientific issues of safety and risk
12 at play here.

13 **2. The Evidence Under the Consumer Expectation Test Was Insufficient.**

14 For the jury’s verdict to stand under “the consumer expectation” test, the Court must find
15 substantial evidence that (1) the Formulation is a product about which an ordinary consumer can
16 form reasonable expectations, (2)the Formulation did not perform as safely as an ordinary
17 consumer would have expected, (3)causation, and (4) harm. Pl.’s Proposed Substantive Jury
18 Instrs. at 17 (May 8, 2018). The lack of evidence on causation is dispositive of the second and
19 third elements of the consumer-expectation test. Because the evidence does not support that the
20 Formulation caused Plaintiff’s cancer, it likewise does not support the jury’s conclusion that the
21 product did not perform as safely as an ordinary consumer would have expected. Additionally,
22 Plaintiff has presented no evidence that the *design of the product*, i.e. the Formulation as opposed
23 to pure glyphosate, caused Plaintiff’s MF. To the contrary, Dr. Nabhan testified that glyphosate
24 and the Formulation are “interchangeable.” Nabhan Tr. at 2788:7-9.²⁰

25 _____
26 ²⁰ No substantial evidence established that the Formulation is more carcinogenic than pure
27 glyphosate, particularly in the instance of Plaintiff’s cancer. And Plaintiff cannot claim that the
28 product at issue was glyphosate because (1) that was not the theory he presented to the jury; and
(2) imposing categorical liability on glyphosate would be against California law. *See Poosh v.*
Philip Morris USA, Inc., 904 F. Supp. 2d 1009, 1025-26 (N.D. Cal. 2012) (applying California

1 The evidence also did not “support a finding that the ordinary consumer can form
2 reasonable minimum safety expectations” about the Formulation. *See Saller*, 187 Cal. App. 4th at
3 1234; *see also* May 17, 2018 Order on Deposition Designations and Certain Proposed Jury
4 Instructions at p. 4-5. There was no evidence the Formulation or its effects are part of the
5 “everyday” experience of the ordinary consumer or that minimum safety standards for the
6 Formulation are common knowledge of the ordinary consumer. Plaintiff himself was not an
7 ordinary consumer; he was certified as a qualified applicator and purchased the product from a
8 special distributor, not a retail store. *Johnson Tr.* at 3225:18-23, 3303:22-3304:19. He had
9 specialized training on how to mix and apply the product safely. *Id.* at 3229:5-19, 3313:12-16.

10 In light of the complex expert evidence on the critical issues in this case, the consideration
11 of safety expectations for the Formulation are simply beyond the purview of ordinary consumers.
12 JNOV must be granted on the design-defect claim for this additional reason.

13 **B. Monsanto is Entitled to JNOV on the Failure to Warn Claims.**

14 The jury’s verdicts on strict liability and negligent failure to warn are not supported by
15 substantial evidence: there is no evidence any cancer risk was known or knowable generally or by
16 Monsanto or that a failure to warn actually caused Plaintiff’s injury.²¹

17 **1. The Alleged Cancer Risks Were Not Known or Knowable in Light of**
18 **the Prevailing Scientific and Medical Knowledge.**

19 The jury’s failure to warn verdict cannot stand unless there was substantial evidence that
20 the probable risks of NHL and MF were known or reasonably knowable at the time of distribution
21 in light of the “generally recognized and prevailing best scientific and medical knowledge.”

22 *Carlin*, 13 Cal. 4th at 1112, 1116; *Valentine v. Baxter Healthcare Corp.*, 68 Cal. App. 4th 1467,
23

24 law and rejecting that cigarette company could be liable for defective design of cigarettes because
25 “[t]aken to its logical conclusion, the argument . . . would mean that the only remedy for this
26 alleged design defect would be a ban on the manufacture and sale of any cigarettes containing
27 nicotine”; *see also* Restatement (Second) of Torts 402A, cmts. k, j; *Brown v. Superior Court*, 44
28 Cal. 3d 1049, 1059-60 (1988); *Oaks v. E.I. Du Pont de Nemours & Co.*, 272 Cal. App. 2d 645
(1969).

21 Judge Karnow’s Order dated May 17, 2018, granted Plaintiff’s motion for summary judgment
on Monsanto’s preemption defenses. Because that Order is a final adjudication of these defenses,
Monsanto will not reassert them here but maintains that they are meritorious and preserves them.

1 1483-84 (1999). The mere possibility of risk does not trigger a duty to warn. *See Carlin*, 13 Cal.
2 4th at 1115-16 (failure-to-warn claim requires evaluating “whether available evidence established
3 a causal link”).

4 Even if Plaintiff had managed to prove a causal link between the Formulation and NHL or
5 MF, there was not substantial evidence that the probable risks of NHL were generally recognized
6 or prevailing in the scientific community at the time when the Formulation was distributed, which
7 had to have been prior to Plaintiff’s diagnosis in 2014.²² The evidence showed just the opposite.
8 As is true today, the scientific and regulatory communities were virtually uniform in the belief that
9 glyphosate and glyphosate-based herbicides do not cause NHL. Until the 2015 publication of the
10 IARC Monograph, *every scientific and regulatory agency* that had examined the issue concluded
11 that glyphosate was unlikely to cause cancer and that no warning was necessary. Portier Tr. at
12 2100:18-2101:22, 2098:13-23, 2104:21-2105:18, 2111:2-9, 2121:11-19; *see also* CACI 1205
13 Comments (risk must be “generally recognized,” “prevailing in the relevant scientific
14 community,” and “represents the best scholarship available,” not a minority viewpoint); *Ramirez*
15 *v. Plough, Inc.*, 6 Cal. 4th 539, 556 (1993) (regulatory findings “deserve[] serious consideration”).
16 And the NCI Study definitively refutes whatever tentative support the IARC Monograph might
17 have provided for the existence of a connection between the Formulation and NHL.

18 The IARC Monograph is not evidence that any risk about NHL was known or reasonably
19 knowable. The IARC Monograph was not published until 2015—three years *after* Plaintiff’s first
20 exposure in 2012, one year *after* Plaintiff’s MF diagnosis in 2014, and, thus, well after
21 distribution. Even putting the timing aside, the IARC Monograph is not evidence to establish
22 knowledge of a risk of NHL or that a warning was required because it is a hazard assessment and
23 not a risk assessment and thus does not support that cancer is a probable risk from exposure to the
24 Formulation. Portier Tr. at 1717:7-12, 1741:25-1742:15. Plaintiff’s own treating physicians told
25 him that there was no known cause for MF and no evidence establishing the Formulation as a
26 cause of MF. Johnson Tr. at 3324:20-3325:19. That was true at the time of his diagnosis and

27 _____
28 ²² There was no evidence that Plaintiff’s exposure after 2014 made his MF worse or changed his
prognosis. Nabhan Tr. at 2864:19-2865:6; Kuzel Tr. at 4777:16-22.

1 remains true today. Thus, Plaintiff presented no evidence that any risk was known or reasonably
2 knowable to the scientific community during the relevant time period.

3 **2. Monsanto Did Not Know, Nor Should It Have Known, of the Alleged**
4 **Risk and There Was No Evidence that a Reasonable Manufacturer**
5 **Would Warn.**

6 The negligent failure to warn verdict also cannot stand because there was not substantial
7 evidence that Monsanto knew or should have known of the risk and that its conduct in failing to
8 warn fell below the standard of care of a reasonable manufacturer under the same or similar
9 circumstances. CACI 1222 (plaintiff must prove “that [defendant] knew or reasonably should
10 have known that the [product] was dangerous”); *Kase v. Metalclad Insulation Corp.*, 6 Cal. App.
11 5th 623, 644 (2016). JNOV should be granted where the plaintiff fails to present competent
12 evidence of the applicable standard of care. *Stephen v. Ford Motor Co.*, 134 Cal. App. 4th 1363,
13 1367 (2005) (affirming nonsuit in favor of defendant where plaintiff did not have any expert
14 testimony or evidence establishing the applicable standard of care).

15 Plaintiff’s evidence came up short in almost every way. He presented no evidence on the
16 standard of care. And he presented no evidence that a reasonable manufacturer would warn given
17 the status of the science. In short, there was no evidence—none—that a reasonable manufacturer
18 knew or should have known that the Formulation could cause MF at any relevant time. To the
19 contrary, the evidence established Monsanto reviewed the vast scientific data and concluded there
20 was no risk to warn about, while complying with all regulatory requirements and gaining
21 regulatory approval. Goldstein Dep. at 329:18-330:11. Indeed, Monsanto actually conducted
22 *more* tests on the Formulation than required by regulators. Farmer Dep. at 433:9-434:2. Plaintiff
23 failed to establish Monsanto did not act reasonably or in accordance with the applicable standard
24 of care.

25 **3. Any Failure to Warn Did Not Cause Plaintiff’s Injury.**

26 Finally, no substantial evidence supported a finding that a warning would have changed
27 Plaintiff’s exposure or prevented his disease. *See, e.g., Huitt v. S. Calif. Gas Co.*, 188 Cal. App.
28 4th 1586, 1604 (2010); *In re Zyprexa Prods. Liab. Litig.*, 2009 WL 1850970, at *14 (E.D.N.Y.
June 22, 2009); *Rosburg v. Minn. Mining & Mfg. Co.*, 181 Cal. App. 3d 726, 735 (1986). Rather,

1 the unrefuted evidence established:

- 2 • Plaintiff already wore personal protective equipment because he thought the Formulation
3 was dangerous. “Q. So why did you decide to wear all that? Because I knew I was
4 applying a chemical, and if it could kill weeds, I’m pretty sure it could kill me, is the way I
5 looked at it, so I didn’t play with that stuff at all. I took it seriously, and that’s why I wore
6 anything I could to protect myself.” Johnson Tr. at 3237:11-16.
- 7 • Plaintiff kept spraying after he suspected the Formulation caused cancer and allegedly was
8 told by his employer that it caused cancer. *Id.* at 3235: 6-25.
- 9 • Plaintiff kept spraying after he asked his treating physician whether he should stop
10 spraying and she wrote to the School Board requesting that Plaintiff not be exposed. *Id.* at
11 3154:2-16.
- 12 • Plaintiff kept spraying after the first acute exposure incident, even though he was
13 concerned about his health and safety. *Id.* at 3266:13-15.

14 In short, because there was no evidence that a different warning would have changed his exposure
15 or diagnosis, Monsanto is entitled to JNOV on the failure to warn claim.

16 **IV. MONSANTO IS ENTITLED TO JNOV ON PUNITIVE DAMAGES BECAUSE**
17 **THE EVIDENCE WAS NOT SUFFICIENT TO SUPPORT THE VERDICT.**

18 Punitive damages cannot be warranted unless there was clear and convincing evidence that
19 Monsanto *knew* or *should have known* the Formulation could cause NHL or MF and failed to take
20 appropriate actions based on that knowledge. *State Farm Mut. Auto. Ins. Co. v. Campbell*, 538
21 U.S. 408, 419 (2003). It would require extraordinary evidence to show that Monsanto did know
22 such a thing when the consensus of those who make such evaluations for the public safety—EPA
23 and the other national and international regulators—agree that the Formulation does *not* cause any
24 human cancer. But no such extraordinary evidence (or *any* competent evidence) was offered to
25 demonstrate such knowledge. This Court initially recognized the paltry evidence for punitive
26 damages, noting that, even “cobbled together,” the evidence “just barely meets the threshold to
27 allow [punitive damages] to go to the jury.” 7/30/18 Tr. at 4027:4-8. The evidence is now in and
28 it was not enough. Reviewing the evidence in the light most favorable to Plaintiff, the “best that
can be said is that there was (and is) an on-going debate in the scientific and medical community”
about whether the Formulation “more probably than not” causes NHL; this does not and cannot
give rise to a duty to warn, much less punitive damages. *Talcum* Order, at *16.

1 **A. Punitive Damages Are Disfavored and Subject to Exacting Proof Requirements.**

2 Punitive damages are “disfavored” because they create the “anomaly of excessive
3 compensation.” *Piscitelli v. Friedenber*g, 87 Cal. App. 4th 953, 980 (2001). The standard for
4 punitive damages is deliberately high. California requires clear and convincing evidence of both
5 (1) an “intent” to cause harm or a “willful and knowing disregard for the safety of others,” and
6 (2)a “despicable” act that is “so vile, base, or contemptible that it would be looked down upon and
7 despised by reasonable people.” CACI 3945; Cal. Civ. Code, § 3294(c)(1). The Legislature’s
8 amendment of Section 3294(c)(1) to add “despicable” in 1987 “represent[ed] a new substantive
9 limitation on punitive damage awards,” that “must be found” along with the preexisting intent or
10 “willful and conscious disregard” element. *College Hosp. Inc. v. Superior Court*, 8 Cal. 4th 704,
11 725 (1994).

12 Not only must the wrongful actor engage in “despicable conduct” that “consciously
13 disregard[ed] the safety of others,” that conduct must be perpetrated, authorized, or ratified by an
14 officer, director, or managing agent of the corporation. *Wilson v. Southern Cal. Edison Co.*, 234
15 Cal. App. 4th 123, 164 (2015); Cal. Civ. Code § 3294(b). A “managing agent” includes “only
16 those corporate employees who exercise substantial independent authority and judgment in their
17 corporate decision-making so that their decisions ultimately determine corporate policy.” *Id.*

18 Finally, “[p]unitive damages . . . must be tied to oppression, fraud, or malice in the conduct
19 which gave rise to liability in the case” and caused plaintiff’s harm. See *Willis v. Buffalo Pumps*
20 *Inc.*, 2014 WL 1028437, *5 (S.D. Cal. Mar. 17, 2014); see also *Holdgrafer v. Unocal Corp.*, 160
21 Cal. App. 4th 907, 928 (2008) (“[W]e agree...that the...evidence should have been excluded from
22 trial because it involves deplorable conduct that had nothing to do with the conduct that harmed
23 Plaintiffs.”); *State Farm*, 538 U.S. at 422-423 (requiring the wrongful conduct to “have a nexus to
24 the specific harm suffered by the plaintiff”).

25 **B. There Is No Evidence Monsanto Willfully and Knowingly Disregarded Risk of**
26 **NHL or Intended to Cause Harm.**

27 There was no evidence, let alone clear and convincing evidence, that any Monsanto
28 employee or scientist intended to harm Plaintiff or other consumers, or willfully and knowingly

1 disregarded a cancer risk. Indeed, the documents and testimony relied upon by Plaintiff were
2 taken out of context, and the record establishes that they did not support Plaintiff's arguments or
3 the imposition of punitive damages.

4 **No Evidence Monsanto Willfully and Knowingly Disregarded a Risk that the**
5 **Formulation Could Cause NHL.** Plaintiff presented no evidence that any Monsanto employee
6 believed that exposure to the Formulation causes NHL. All testimony from Monsanto employees
7 was emphatically to the contrary. Dr. Farmer, for example, testified that there is no evidence to
8 support the statement that the Formulation causes cancer. Farmer Dep. at 52:20-22. She further
9 testified that the surfactants "are not carcinogenic" based on testing. *Id.* Dr. Goldstein testified
10 from a clinical view that "sound science supports the contention that glyphosate does not cause
11 cancer." Goldstein Dep. at 40:10-12.

12 Plaintiff relied on a sentence of one email in which Dr. Farmer correctly advised a public
13 relations employee that he should not say that animal carcinogenicity studies show that
14 "Roundup"—the Formulation—does not cause cancer, because glyphosate itself, not the
15 formulation, was the subject of these studies. Farmer Dep. at 52:3-7; 53:15-18; PX 305.
16 Everybody agreed that the U.S. EPA mandates long-term animal cancer bioassays on only
17 glyphosate, not the Formulation—because, as Dr. Foster explained, bioassays of the Formulation
18 are not feasible. Tr. at 4504:4-16. In the very same email, Dr. Farmer endorses the statement that
19 "Roundup did not cause cancer, birth defects, or adverse reproductive changes at dose levels far in
20 excess of likely exposure." PX 305. Plaintiff's misuse of Dr. Farmer's statement provides no
21 basis for a finding of intent to harm Plaintiff.

22 Plaintiff also asked the jury to consider a 2002 internal memorandum acknowledging "six
23 published studies that arguably associate glyphosate and other pesticides with lymphopietic
24 cancers or adverse reproductive outcomes." PX 282 (not admitted into evidence). But that was
25 not Monsanto's view, or the views of any of the regulators (or IARC) reviewing those studies.
26 Monsanto's view was stated in the very first sentence of the document, which makes explicit that
27 "[g]lyphosate has very favorable toxicologic properties. It is not carcinogenic, mutagenic or
28 neurotoxic and it is not a reproductive or developmental toxin." *Id.* With respect to the "six

1 published studies” the memo notes that “independent reviewers judge these studies to be poor
2 quality.” Further, the purpose of the memo is to address these “poor quality” studies with
3 additional research—a proposal to conduct a family farm study to better understand “applicator
4 pesticide exposure under ‘real world’ conditions.” *Id.* And that is what Monsanto did.

5 By the time Plaintiff was exposed to the Formulation, glyphosate had developed one of the
6 largest bodies of scientific data of any substance in the world. Portier Tr. at 2051:1-3; Martens
7 Dep. at 194:9-14. There was, and is, a global consensus of safety both before and after IARC.
8 Indeed, after IARC’s evaluation, all of the worldwide regulators continue to find that the
9 Formulation is safe and *not* carcinogenic—not only U.S. EPA, but also EFSA, ECHA, Australia,
10 New Zealand, and the German BfR authority. Farmer Dep. at 395:7-15, 400:16-24; Portier Tr. at
11 2014:6-14, 2110:23-2111:1; Goldstein Dep. at 340:7-341.3; *BMW of North America v. Gore*, 517
12 U.S. 559, 575 (1996); *see also Willis*, 2014 WL 1028437, at *5; (holding that knowledge by
13 defendant that “postdate[s] Plaintiff’s exposure to Defendant’s products . . . can lend no support to
14 Plaintiff’s claim that Defendant acted with malice” due to lack of nexus to specific harm). Even
15 IARC’s sister organization within WHO (JMPR) rejected a cancer link both before and after
16 IARC’s classification. Farmer Dep. at 396:5-20. Monsanto cannot be punished, consistent with
17 the “elementary notions of fairness” for a risk that no regulatory or scientific body, or other
18 manufacturer, had identified prior to Plaintiff’s exposure and MF diagnosis. *See also* Prosser and
19 Keeton on Torts § 36, at 233 n.41 (5th ed. 1984) (“In most contexts... compliance with a statutory
20 standard should bar liability for punitive damages.”). It is not consistent with Due Process to
21 “punish” Monsanto based on an honestly-held scientific conclusion that Monsanto shared with the
22 world’s regulatory scientists and Plaintiff’s own doctors.

23 Finally, Plaintiff presented no evidence that Monsanto had unique knowledge of risks
24 associated with exposure to the Formulation unknown in the scientific domain available to public
25 regulators.²³ Judge Nelson observed in the *Talcum* Order that, in light of “the fact that the

26 _____
27 ²³ Plaintiff alleges that Monsanto failed to properly respond to opinions expressed by Dr. Parry in
28 the late 1990s with respect to various published and regulatory genotoxicity studies. This
allegation is false for the reasons stated *infra* at 28-29, but in any event, the studies addressed by

1 scientific community was (and is) divided on the question of whether talc causes ovarian cancer,”
2 it was “significant” that plaintiff presented “no internal research or study by [defendant] that was
3 not in the public scientific domain” suggesting the defendant “‘knew or should have known’ of the
4 dangers of talc.” *Talcum* Order, at *15-16. She held that where science is divided a “showing [of
5 knowledge] beyond the publicly available literature” was required to support punitive damages.
6 *Id.* That reasoning applies with equal force here.

7 **No Evidence that Monsanto Intended to Cause Harm.** There likewise is no basis in the
8 record to conclude that anybody at Monsanto—at any time—intended to cause harm.

9 The Court cited two emails as a basis for denying Monsanto’s motion for directed verdict
10 on punitive damages—an email from Dr. Farmer discussing the McDuffie epidemiology study
11 (PX 313) and another email from Dr. Heydens discussing the issue of allegedly “ghostwriting” the
12 Williams (2000) paper (PX 362). 8/6/18 Tr. at 4908:22-4909:22. In the McDuffie email, Dr.
13 Farmer approves of the decision by the study authors not to identify glyphosate in the study
14 abstract. This email is in full accord with Dr. Farmer’s belief glyphosate does not cause cancer, a
15 conclusion with which regulators around the world agree. *See supra* at 21-22. Indeed, Plaintiff’s
16 own experts agree that there is no finding of any statistically significant association in that study
17 which is fully adjusted for other pesticides. *See supra* at 3-4. Further, Dr. Farmer’s reaction to the
18 McDuffie investigator’s drafting decision does not reflect *any* affirmative “act” much less the type
19 of “despicable” one required for imposition of punitive damages.

20 The other email the Court cited is by Dr. Heydens in late 2015 and uses the word
21 “ghostwrite” in reference to a review article by three other scientists published some 15 years
22 prior. As Dr. Heydens testified, however, Monsanto scientists did not “ghostwrite” the Williams
23 (2000) paper, unequivocally shown by the fact that both Dr. Heydens and Dr. Farmer, along with
24 other Monsanto scientists, specifically appear in the Williams (2000) acknowledgements as
25 making a “substantial contribution” to the paper. Heydens Dep. at 406:12-18. Nor was there any
26 claim that there was anything false or misleading about the Williams (2000) publication; no one

27

28 Dr. Parry were published in the peer-reviewed literature and/or known to U.S. EPA.

1 claimed the review of the science was wrong or fraudulent. And to the extent that Plaintiff relies
2 on purported ghostwriting of separate publications in late 2015, those allegations also ignore the
3 testimony of Drs. Heydens and Farmer, the explicit acknowledgment of Monsanto support in the
4 articles and, in any event, would have occurred *after* Mr. Johnson’s use of the Formulation. Thus,
5 there could be no conceivable nexus to any harm to him.

6 **Plaintiff’s Failure to Test Theory.** The other charge levied against Monsanto’s collective
7 mental state was that Monsanto failed to adequately test its product. Not only is that allegation
8 insufficient as a matter of law for punitive damages (it implies a negligence, not a malice
9 standard), the charge is emphatically contrary to the evidence that glyphosate is among the most
10 extensively studied chemicals of all time, backed by a massive regulatory body of studies over the
11 course of decades. Plaintiff presented no evidence that Monsanto did not comply with ongoing
12 regulatory requirements to support its product with GLP (Good Laboratory Practice)-quality
13 testing. Indeed, the very documents Plaintiff presented to the jury of “bad intent” show Monsanto
14 continuing to go above and beyond the regulatory requirements. For instance, PX 282 is an
15 internal memorandum deciding to conduct a biomonitoring study in Formulation-exposed farm
16 workers—above and beyond any regulatory requirements to do so—to determine whether there
17 could be any cancer concern from real world exposures to the Formulation. The evidence further
18 showed that *no regulatory agency* in the world requires the testing Plaintiff claims Monsanto
19 failed to conduct—long-term carcinogenicity testing on the Formulation (as opposed to separate
20 testing on the active or other ingredients)—and that no other glyphosate formulation manufacturer
21 in the world does such testing. There was thus no evidence that Monsanto failed to comply with
22 any applicable industry standard; in fact, evidence was presented (unrebutted) why such tests
23 *cannot be* and *are not* conducted. Foster Tr. at 4504:4-21, 4505:9-14.

24 Finally, the charge that Monsanto’s scientists “ignored” Dr. Parry’s advice to do more
25 genotoxicity tests (beyond the dozens already conducted and submitted to federal regulators) is
26 also factually baseless. The evidence was that Monsanto conducted the majority of additional
27 studies proposed by Dr. Parry and publicly presented the results. Martens Dep. at 127:11-129:3;
28 216:16-217:21, 218:18-25. Upon review the results of these studies, Dr. Parry agreed that the

1 Formulation was not genotoxic. *Id.* at 224:25-225:7, 227:20-228:5. Regulators around the world
2 agree. This evidence cannot, as a matter of law, support a finding of malice.

3 **The IARC Monograph does not support punitive damages.** The Monograph was
4 released *after* Plaintiff was diagnosed with MF, and thus cannot inform Monsanto’s mental state at
5 any relevant period of time. Nabhan Tr. at 2869:25-2861:25. Prior to that time (and since),
6 worldwide regulators have concluded that the Formulation is safe for use and need not contain a
7 cancer warning. Portier Tr. at 2010:4-25, 2098:21-23, 2106:12-15, 2110-2112, 2121-2122.
8 Moreover, Monsanto’s scientists testified that they all agreed with the EPA’s review of the science
9 (and EFSA, ECHA, BfR, etc.), which was indisputably more thorough than IARC’s. IARC
10 evaluated glyphosate (and the four other chemicals discussed in Monograph 112) during the
11 course of a single week. Blair Dep. at 117:16-24. Plaintiff’s expert Dr. Benbrook testified that
12 U.S. EPA’s ongoing review of glyphosate for its current reregistration has gone on for *several*
13 *years* and reflects the state of the science at that time. Benbrook Tr. at 3965:1-6. U.S. EPA, not
14 IARC, has access to all of the original study reports for each of the animal carcinogenicity studies.
15 IARC considered only a fraction of the data that U.S. EPA and other regulators considered,
16 including only a small fraction of the animal studies and genotoxicity studies that U.S. EPA
17 reviewed. IARC also did not consider the NCI Study, the unpublished data in its chairperson’s
18 possession underlying that publication, or the NAPP pooling data. And IARC did not perform a
19 real-world *risk* assessment. Portier Tr. at 1741:25-1742:15.

20 Thus, while Plaintiff argued that Monsanto had plans to “orchestrate outcry” with IARC’s
21 decision shortly before it issued, the evidence was that Monsanto legitimately disagreed with
22 IARC’s process. Goldstein Dep. at 209:7-15. First, anticipation of IARC’s classification is
23 entirely understandable, given the one-in-a-thousand historical odds that a chemical will be
24 classified as Group 4 (probably not carcinogenic), or the unlikely outcome that a product with 40
25 years of science and data would be classified as Group 3 (not classifiable). Neugut Tr. at 2598:4-
26 19; Nabhan Tr. at 3003:13-3005:25. Second, all of the Monsanto scientists who testified
27 explained that they disagreed with IARC’s classification after it was released.

28 At most, Plaintiff has shown that there is a bona fide scientific disagreement, with IARC’s

1 hazard assessment on one side and the fulsome assessment of every Monsanto scientist and every
2 regulatory body on the other. This Court, in considering the submissibility of punitive damages,
3 noted that “the science here is very much in dispute” and that the Court would “have to reconsider
4 [punitive damages], depending on what the jury does with that.” 7/30/18 Tr. at 4027:4-8; 8/6/18
5 Tr. at 4908:17-21. The Court should do so: a “bona fide disagreement” about a scientific dispute
6 does not demonstrate clear and convincing evidence of malice as a matter of law. *See Kendall*
7 *Yacht Corp. v. United California Bank*, 50 Cal. App. 3d 949, 959 (1975) (reversing punitive
8 damages award because it “remains purely speculative as to whether the Bank acted with such
9 malice rather than out of a bona fide disagreement over” plaintiff’s claims); *Satcher v. Honda*
10 *Motor Co.*, 52 F.3d 1311, 1316-17 (5th Cir. 1995) (debate concerning whether benefits of leg
11 guards outweighed their risks militated against punitive damages); *Berroyer v. Hertz*, 672 F.2d
12 334, 342 (3d Cir. 1982) (“difference of medical opinion on the degree of cancer risk” among
13 experts is “insufficient support” for punitive damages).

14 **C. No Clear and Convincing Evidence Monsanto Acted Despicably.**

15 Plaintiff also failed to present any evidence, let alone clear and convincing evidence, for
16 the second element of punitive damages—a “despicable act” by Monsanto. Plaintiff argued to the
17 jury that Monsanto’s supposed “ghostwriting” of scientific articles, attempts to influence EPA,
18 failure to return a phone call to Plaintiff, and failure to inform Plaintiff about IARC’s
19 classification in a later call was “despicable conduct.” The actual evidence falls far short of
20 “despicable conduct” under the JNOV standard.

21 **1. Monsanto’s Participation in Science Was Not Despicable.**

22 Plaintiff’s counsel repeatedly argued to the jury that Monsanto “made a choice to engage in
23 ghostwriting” and that in “document after document . . . Monsanto’s response to . . . legitimate
24 scientific concerns, is to make up science.” Closing Tr. at 5056:20-23. These assertions were
25 unfounded. No evidence was presented purporting to show that Monsanto fabricated any
26 scientific data. Plaintiff did not even try to present any such evidence; it appears the only reason
27 these allegations were presented was to allow Plaintiff’s counsel to make false assertions that
28 Monsanto “made up” science.

1 The evidence does not suggest anything despicable. Dr. Farmer explained, generally, how
2 Monsanto works with an independent author:

3 [I]’s a draft for them to include or exclude in their final publication. And we
4 provide input all the time because we have some more of the knowledge that they
5 do, but there’s nothing here that we’re trying to hide. We’re actually adding more
6 information for them to include in that review. Again, under the umbrella of
7 transparency, we’re trying to make sure that it’s a really thorough, complete
8 document. And then they can choose in that sense to either complete them, change
9 them, delete them, do whatever they want to do with them.

10 Farmer Dep. at 122:25-123:13. Dr. Farmer further explained that when Monsanto works with an
11 independent author “they talk about us in their credits” so “everyone knows” Monsanto made
12 contributions. *Id.* at 122:4-11. Again, the evidence bears this out:

- 13 • **Williams 2000:** Dr. Heydens explained repeatedly that Plaintiff’s allegation he
14 “ghostwrote” the Williams 2000 review is incorrect. (Heydens Dep. at 124:23-125:8).
15 The Williams 2000 paper unambiguously acknowledges Monsanto “made significant
16 contributions” to the paper, including specifically Dr. Heydens. (Portier Tr. at 1890:12-
17 1891:22). Monsanto was helping to make public the findings of regulatory safety studies;
18 it was not hiding its contribution from scientific or regulatory bodies, or the public at large.
- 19 • **Kier & Kirkland 2013:** Monsanto employee David Saltmiras did not and could not
20 participate in the preparation of this paper because it required review of proprietary
21 information of other glyphosate registrants. (PX 445). But what he did do—along with
22 several employees of other glyphosate registrants—is “thoughtfully review” the paper after
23 it was drafted, as well as coordinate the dissemination of information to Kier & Kirkland
24 from all the glyphosate registrants, all of which is noted on the face of the Kier & Kirkland
25 paper itself. (PX 799, p. 310-11).
- 26 • **Intertek Papers 2016:** These papers acknowledge on their face that they were sponsored
27 by Monsanto. At the request of the coordinator of the Intertek expert panels, Dr. Heydens
28 provided his “suggestions” as well as “minor edits” concerning an introductory summary
paper recounting the findings of the separately published independent panel papers, for the
coordinator to use “the way he saw fit.” (Heydens Dep. at 168:1-10). Dr. Heydens had
“no idea” what Intertek did with the edits before the summary paper was published, which,
in any event, long post-dated Mr. Johnson’s MF diagnosis and last exposure to the
Formulation. (*Id.* at 175:1-22).

29 Evidence showing Monsanto follows, understands, and participates in scientific study and
30 literature concerning glyphosate should be shocking to nobody.

31 Plaintiff’s unfounded ghostwriting accusations are also irrelevant to punitive damages
32 because they are entirely unrelated to the conduct that gave rise to liability. Plaintiff did not
33 introduce any evidence that tied alleged “ghostwriting” or responses to IARC to any effect on him.
34 He also did not present any evidence that such alleged conduct in any way altered the science or

1 was despicable. Although Plaintiff’s accusations allegations may have inflamed the jury, without
2 evidence that Monsanto’s participation in meaningful scientific debate had any connection to harm
3 to Plaintiff, they cannot support the award of punitive damages.

4 **2. Petitioning a Regulatory Agency Is Not Despicable Conduct.**

5 Plaintiff presented some testimony concerning Monsanto’s interactions with regulators,
6 including EPA, and argued during closing argument that this communication with EPA was
7 “creepy.” Tr. at 5067:21–5068:22. This is a straightforward attempt to punish First Amendment
8 protected activity. And it is unlawful. Punitive damages may not rest on discussions Monsanto
9 had with EPA, whether about glyphosate, the weaknesses of the IARC Monograph, or how the
10 science might affect regulatory standards. The First Amendment protects Monsanto’s right to
11 advocate its interest to U.S. EPA. *Stern v. United States Gypsum*, 547 F.2d 1329, 1342 (7th Cir.
12 1977) (right to petition government is “fundamental to the very idea of a republican form of
13 governance.”). Under the *Noerr-Pennington* doctrine, which is derived from the First
14 Amendment, civil liability may not rest on advocacy or lobbying efforts conducted before
15 governmental bodies. *See United Mine Workers of Am. v. Pennington*, 381 U.S. 657 (1965);
16 *E.R.R. Presidents Conference v. Noerr Motor Freight*, 365 U.S. 127 (1961); *accord Ludwig v.*
17 *Superior Court*, 37 Cal. App. 4th 8, 21 (1996) (“Those who petition the government are generally
18 immune from ... liability.”). The punitive damages award cannot rest on Monsanto’s lawful and
19 legitimate interactions with the EPA.

20 **3. Monsanto’s Failure to Return a Phone Call to Plaintiff Does Not**
21 **Support Punitive Damages.**

22 Plaintiff called Monsanto in November 2014 and the Missouri Regional Poison Control
23 (“MRPC”) in March 2015 to report that a rash appeared after an exposure to the Formulation. *See*
24 *PX 333*; *see also Johnson* Tr. at 3324:1-16. During the initial 30-45 minute call, Plaintiff spoke to
25 a Monsanto employee who “knows her product very well” and was accurately informed “we really
26 don’t have those symptoms along with this product.” Nabhan Tr. at 2983:2-2984:22. Plaintiff’s
27 counsel argued to the jury that Monsanto “made a choice when they didn’t call [Plaintiff] back.”
28 Closing Tr. at 5056:14-19; *PX 332*. Dr. Goldstein, a clinical physician employed by Monsanto,

1 was informed about the call, stated in internal correspondence that he intended to call him back,
2 testified under oath that he intended to call Plaintiff back, but did not recall doing so. Goldstein
3 Dep. at 37:13-17. Plaintiff made his second call in March 2015, when he spoke to MRPC at
4 length about his condition. Closing Tr. at 5103:10-20; PX 334.

5 With no evidentiary support, Plaintiff’s counsel attempted to insinuate nefarious conduct
6 from this circumstance. Arguing to the jury, Plaintiff’s counsel stated that Monsanto “made a
7 choice when they didn’t call [Plaintiff] back,” (Closing Tr. at 5056:14-19; PX 332) because
8 Monsanto was worried that calling Plaintiff back and warning about IARC would harm California
9 sales of Roundup. But Plaintiff offered no evidence that Monsanto made a “choice” not to call
10 Plaintiff, much less one motivated by greed or intent to deny Plaintiff any safety information. At
11 worst, this isolated episode amounted to “mere carelessness,” which “does not justify the
12 imposition of punitive damages.” *Lackner v. North*, 135 Cal. App. 4th 1188, 1210 (2006).²⁴

13 Further, there is no nexus between the phone calls and Plaintiff’s injury. At the time of the
14 calls, Plaintiff had already contracted his disease and been diagnosed with MF. There is no
15 evidence that disclosure of the IARC Monograph some 1-2 years after Plaintiff’s MF became
16 symptomatic could have had any impact on his existing cancer. As discussed above, the only
17 “evidence” of “tumor” promotion were the benign growths from the 2010 George study—deemed
18 by IARC itself to be an “inadequate study” with a “poor design.” PX 784 at 0034; Portier Tr. at
19 1863:21-25 (“[IARC] reviewed it. I don’t believe they used it.”); 2229:13-2230:6.²⁵

20 **D. No Clear and Convincing Evidence of Conduct by a Managing Agent.**

21 There was also no evidence in this case of conduct from an officer, director, or managing
22

23 ²⁴ The allegedly unreturned phone call involving Dr. Goldstein took place months before IARC’s
24 classification, and there is no evidence that Monsanto made a “choice” or directed MRPC to
25 suppress any information to Plaintiff during the second call, which occurred only days after IARC
publicly announced its classification of glyphosate.

26 ²⁵ EPA likewise declined to consider that study, on the basis that it was “inadequate in protocol,
27 conduct or reporting.” Portier Tr. at 2207:14-19; DX 2481 at 0070. Dr. Nabhan agreed that there
28 is no evidence that Plaintiff’s continued use of the Formulation contributed to his MF or somehow
made it worse—Dr. Nabhan just did not know (“I don’t think we know. You know, I mean, it’s
hard to tell.”). Tr. at 2865:1-5. Dr. Kuzel testified that he had never seen “any evidence” that
Plaintiff’s MF progressed because he continued using the Formulation. Tr. at 4777:16-22.

1 agent of Monsanto. See *Wilson*, 234 Cal. App. 4th at 164 (“managing agent” includes “only those
2 corporate employees who exercise substantial independent authority and judgment in their
3 corporate decisionmaking so that their decisions ultimately determine corporate policy.”).

4 At the jury instruction conference, the Court expressed the view that Kirk Azevedo and
5 Steven Gould were not managing agents. 8/6/18 Tr. at 4908:5-9. The Court was correct:
6 Azevedo, whose testimony was later stricken, was a former Monsanto sales representative located
7 in California who serviced distributors (Azevedo Dep. at 34:1-35:19) and Gould is a sales account
8 manager in California. PX 290. No evidence suggested either had any corporate responsibility
9 outside of making sales and managing accounts.

10 The Court also expressed its view that it was “not convinced” that either Dr. Farmer or
11 Dr. Goldstein were managing agents either. 8/6/18 Tr. at 4907:16-25. The evidence confirms that
12 they were not. Dr. Farmer is a “toxicologist in [Monsanto’s] product safety center” who has been
13 “one of the spokespersons for the safety of Roundup when it comes to the toxicology.” Farmer
14 Dep. at 14:23-15:7. Her specific job responsibility is to “make sure as a regulatory toxicologist . .
15 . [Monsanto] meets all the requirements by the regulators.” *Id.* at 19:14-17. A “spokesperson”
16 does not create the policies they are speaking on. Dr. Goldstein served as a “director” in medical
17 toxicology” and a “lead” in “medical sciences and outreach.” Goldstein Dep. at 151:8-13. He is a
18 physician and clinical toxicologist who reviews “complaints of human health” made to Monsanto.
19 *Id.* at 8:7-24. He is not formulating or directing corporate policy.

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CONCLUSION

The scientific evidence in this case falls far short of the sufficient and substantial evidence required to sustain this verdict. For all of the reasons stated above, Monsanto is entitled to judgment notwithstanding the verdict.

Dated: September 18, 2018

FARELLA BRAUN + MARTELLLP

By: 

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