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MONSANTO COMPANY

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:

ALL ACTIONS

Hearing Date: December 11, 2017

Time: 9:00 a.m.

**MONSANTO COMPANY'S NOTICE OF MOTION AND DAUBERT AND SUMMARY
JUDGMENT MOTION BASED ON FAILURE OF GENERAL CAUSATION PROOF**

TO ALL PLAINTIFFS AND THEIR ATTORNEYS OF RECORD:

PLEASE TAKE NOTICE that beginning on December 11, 2017, at 9:00 a.m., in Courtroom 4 of the United States District Court, Northern District of California, located at 450 Golden Gate Avenue, San Francisco, CA 94102, or as ordered by the Court, Defendant Monsanto Company ("Monsanto") will present its *Daubert* and Summary Judgment Motion Based on Failure of General Causation Proof.

Monsanto seeks an order excluding plaintiffs' general causation expert witnesses (retained and non-retained witnesses) under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), and granting summary judgment for Monsanto in all Roundup® lawsuits

1 pending before this Court based on plaintiffs' failure to present sufficient admissible evidence to
2 prove general causation.

3 DATED: October 6, 2017

Respectfully submitted,

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MONSANTO COMPANY

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS
LIABILITY LITIGATION

MDL No. 2741

Case No. 3:16-md-02741-VC

This document relates to:

ALL ACTIONS

Hearing Date: December 11, 2017
Time: 9:00 a.m.

**MEMORANDUM OF POINTS AND AUTHORITIES IN SUPPORT OF
MONSANTO COMPANY'S DAUBERT AND SUMMARY JUDGMENT
MOTION BASED ON FAILURE OF GENERAL CAUSATION PROOF**

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Cases

<i>In re Accutane Prod. Liab. Litig.</i> , No. 8:04-MD-2523-T-30, 2009 WL 2496444 (M.D. Fla. Aug. 11, 2009), <i>aff'd</i> , 378 F. App'x 929 (11th Cir. 2010)	36
<i>In re "Agent Orange" Prod. Liab. Litig.</i> , 611 F. Supp. 1223 (E.D.N.Y. 1985)	34
<i>Allen v. Pa. Eng'g Corp.</i> , 102 F.3d 194 (5th Cir. 1996)	9, 18, 33, 35
<i>Allison v. McGhan Med. Corp.</i> , 184 F.3d 1300 (11th Cir. 1999)	17
<i>Arias v. DynCorp</i> , 928 F. Supp. 2d 10 (D.D.C. 2013)	15, 40
<i>Avila v. Willitts Env'tl. Remediation Trust</i> , 633 F.3d 828 (9th Cir. 2011)	7, 11, 39
<i>Baker v. Sec'y of HHS</i> , No. 99-653V, 2003 WL 22416622 (Fed. Cl. Sept. 26, 2003)	25
<i>In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.</i> , 524 F. Supp. 2d 1166 (N.D. Cal. 2007)	<i>passim</i>
<i>Bldg. Indus. Ass'n v. Wash. State Bldg. Code Council</i> , 683 F.3d 1144 (9th Cir. 2012)	7
<i>Bourne v. E.I. DuPont De Nemours & Co.</i> , 189 F. Supp. 2d 482 (S.D. W. Va. 2002)	33
<i>Brock v. Merrell Dow Pharm., Inc.</i> , 874 F.2d 307 (5th Cir. 1989), <i>as modified</i> 884 F.2d 116 (5th Cir.1989)	33
<i>Burst v. Shell Oil Co.</i> , 650 F. App'x 170 (5th Cir. 2016)	8, 18
<i>Caraker v. Sandoz Pharm. Corp.</i> , 188 F. Supp. 2d 1026 (S.D. Ill. 2001)	7, 10, 15
<i>Castiac Lake Water Agency v. Whittaker Corp.</i> , No. CV 00-12613 AHM, 2002 WL 34700741 (C.D. Cal. Oct. 25, 2002)	32
<i>Celotex Corp. v. Catrett</i> , 477 U.S. 317 (1986)	39
<i>Chapman v. Proctor & Gamble Distrib., LLC</i> , 766 F.3d 1296 (11th Cir. 2014)	21

1	<i>Claar v. Burlington N. R. Co.,</i>	
	29 F.3d 499 (9th Cir. 1994)	26
2	<i>Conde v. Velsicol Chem. Corp.,</i>	
3	24 F.3d 809 (6th Cir. 1994)	21
4	<i>Daubert v. Merrell Dow Pharm., Inc.,</i>	
	43 F.3d 1311 (9th Cir. 1995)	<i>passim</i>
5	<i>Daubert v. Merrell Dow Pharm., Inc.,</i>	
6	509 U.S. 579 (1993).....	<i>passim</i>
7	<i>In re Denture Cream Prod. Liab. Litig.,</i>	
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8	<i>Dunn v. Sandoz Pharm. Corp.,</i>	
9	275 F. Supp. 2d 672 (M.D.N.C. 2003)	37
10	<i>Dura Auto Sys. of Ind., Inc. v. CTS Corp.,</i>	
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11	<i>Ellis v. Costco Wholesale Corp.,</i>	
12	657 F.3d 970 (9th Cir. 2011)	1
13	<i>FTC v. Wellness Support Net., Inc.,</i>	
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14	<i>Gen. Elec. Co. v. Joiner,</i>	
15	522 U.S. 136 (1997).....	<i>passim</i>
16	<i>Glastetter v. Novartis Pharm. Corp.,</i>	
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17	<i>Golden v. CH2M Hill Hanford Group, Inc.,</i>	
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19	<i>Good v. Fluor Daniel Corp.,</i>	
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20	<i>Haim v. HHS,</i>	
21	No. 90-1031V, 1993 WL 346392 (Fed. Cl. Aug. 27, 1993).....	34
22	<i>Hall v. Baxter Healthcare Corp.,</i>	
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23	<i>In re Hanford Nuclear Reservation Litig.,</i>	
24	292 F.3d 1124 (9th Cir. 2002)	9, 39
25	<i>Henricksen v. ConocoPhillips Co.,</i>	
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26	<i>Hollander v. Sandoz Pharm. Corp.,</i>	
27	289 F.3d 1193 (10th Cir. 2002)	15, 39
28	<i>Hollander v. Sandoz Pharm. Corp.,</i>	
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1	<i>Johnson v. Arkema, Inc.</i> , 685 F.3d 452 (5th Cir. 2012)	3, 4
2	<i>Karlo v. Pittsburgh Glass Works, LLC</i> , 849 F.3d 61 (3d Cir. 2017).....	25
3		
4	<i>Knight v. Kirby Inland Marine, Inc.</i> , 363 F. Supp. 2d 859 (N.D. Miss. 2005).....	20
5	<i>Kumho Tire Co. v. Carmichael</i> , 526 U.S. 137 (1999).....	1, 7
6		
7	<i>In re Lipitor (Atorvastatin Calcium) Mktg., Sales Practices & Prod. Liab. Litig.</i> , 227 F. Supp. 3d 452 (D.S.C. 2017).....	40
8	<i>In re Lipitor (Atorvastatin Calcium) Mktg., Sales Practices & Prod. Liab. Litig.</i> , 174 F. Supp. 3d 911 (D.S.C. 2016).....	37
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10	<i>Lopez .v Wyeth-Ayerst Lab.</i> , No. C 94–4054 CW, 1996 WL 784566 (N.D. Cal. Dec. 13, 1996), <i>aff’d</i> , 139 F.3d 905 (9th Cir. 1998).....	10
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12	<i>Lust v. Merrell Dow Pharm.</i> , 89 F.3d 594 (9th Cir. 1996)	24, 27, 28, 29
13	<i>Lynch v. Merrell-National Labs</i> , 830 F.2d 1190 (1st Cir. 1987).....	34
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15	<i>McClain v. Metabolife Int’l, Inc.</i> , 401 F.3d 1233 (11th Cir. 2005)	9, 35
16	<i>Merrell Dow Pharm., Inc. v. Havner</i> , 953 S.W.2d 706 (Tex. 1997)	33
17	<i>In re Mirena IUD Prod. Liab. Litig.</i> , 202 F. Supp. 3d 304 (S.D.N.Y. 2016), <i>appeal docketed</i> Nos. 16-2890 & 16-3012 (2d Cir. Aug. 19, 2016)	39, 40
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19	<i>Mitchell v. Gencorp Inc.</i> , 165 F.3d 778 (10th Cir. 1999)	8
20		
21	<i>Monroe v. Zimmer U.S., Inc.</i> , 766 F. Supp. 2d 1012 (E.D. Cal. 2011).....	33
22	<i>Myers v. U.S.</i> , No. 02CV1349-BEN, 2014 WL 6611398 (S.D. Cal. Nov. 20, 2014)	9
23		
24	<i>Nationwide Transp. Fin. v. Cass Info. Sys., Inc.</i> , 523 F.3d 1051 (9th Cir. 2008)	22
25	<i>Nelson v. Tenn. Gas Pipeline Co.</i> , 243 F.3d 244 (6th Cir. 2001)	10, 17, 18
26		
27	<i>Newkirk v. ConAgra Foods, Inc.</i> , 727 F. Supp. 2d 1006 (E.D. Wash. 2010), <i>aff’d</i> , 438 F. App’x 607 (9th Cir. 2011)	24, 26
28		

1	<i>Norris v. Baxter Healthcare Corp.</i> , 397 F.3d 878 (10th Cir. 2005)	15
2	<i>O’Hanlon v. Matrixx Initiatives</i> , No. CV 04-10391AHMJTLX, 2007 WL 2446496 (C.D. Cal. Jan. 3, 2007).....	24
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4	<i>Orr v. Bank of America, NT & SA</i> , 285 F.3d 764 (9th Cir. 2002)	39
5	<i>Orrell v. AstraZeneca Pharm. LP (In re Nexium Esomeprazole)</i> , 662 F. App’x 528 (9th Cir. 2016)	28
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7	<i>Pluck v. BP Oil Pipeline Co.</i> , 640 F.3d 671 (6th Cir. 2011)	9
8	<i>Redfoot v. B.F. Ascher & Co.</i> , No. C 05-2045 PJH, 2007 WL 1593239 (N.D. Cal. June 1, 2007)	24
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10	<i>In re REMEC Inc. Sec. Litig.</i> , 702 F. Supp. 2d 1202 (S.D. Cal. 2010).....	26
11	<i>In re Rezulin Prod. Liab. Litig.</i> , 309 F. Supp. 2d 531 (S.D.N.Y. 2004).....	31
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13	<i>Richardson v. Richardson–Merrell, Inc.</i> , 857 F.2d 823 (D.C. Cir.1988)	33
14	<i>Rider v. Sandoz Pharm. Corp.</i> , 295 F.3d 1194 (11th Cir. 2002)	1, 10
15		
16	<i>Schudel v. Gen. Elec. Co.</i> , 120 F.3d 991 (9th Cir. 1997), <i>abrogated on other grounds</i> <i>by Weisgram v. Marley Co.</i> , 528 U.S. 440 (2000).....	12
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18	<i>Siharath v. Sandoz Pharm. Corp.</i> , 131 F. Supp. 2d 1347 (N.D. Ga. 2001), <i>aff’d</i> , <i>Rider v. Sandoz Pharm. Corp.</i> , 295 F.3d 1194 (11th Cir. 2002)	11, 24
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20	<i>In re Silicone Gel Breast Implants Prod. Liab. Litig.</i> , 318 F. Supp. 2d 879 (C.D. Cal. 2004)	22, 24
21	<i>Soldo v. Sandoz Pharm. Corp.</i> , 244 F. Supp. 2d 434 (W.D. Pa. 2003).....	11, 37, 38
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23	<i>Valentine v. Pioneer Chlor Alkali Co., Inc.</i> , 921 F. Supp. 666 (D. Nev. 1996).....	19
24	<i>Wade-Greaux v. Whitehall Labs.</i> , 874 F. Supp. 1441 (D.V.I. 1994)	33, 35, 36
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26	<i>Washington v. Kellwood Co.</i> , 105 F. Supp. 3d 293 (S.D.N.Y. 2015).....	32
27	<i>Weisgram v. Marley Co.</i> , 528 U.S. 440 (2000).....	7
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1	<i>Wintz v. Northrop Corp.</i> , 110 F.3d 508 (7th Cir. 1997)	9
2	<i>Wright v. Williamette Indus., Inc.</i> , 91 F.3d 1105 (8th Cir. 1996)	9
3		
4	<i>Young v. Burton</i> , 567 F. Supp. 2d 121 (D.D.C. 2008)	9, 35
5	<i>In re Zicam Cold Remedy Mktg., Sales Practices & Prod. Liab. Litig.</i> , No. 09-md-2096-PHX-FJM, 2011 WL 798898 (D. Ariz. Feb. 24, 2011)	33
6		
7	<i>In re Zicam Cold Remedy Mktg., Sales Practices & Prod. Liab. Litig.</i> , 797 F. Supp. 2d 940 (D. Ariz. 2011)	9, 35
8	<i>In re Zolof Prods. Liab. Litig.</i> , 858 F.3d 787 (3d Cir. 2017).....	11, 40
9		
10	<i>Zwilling v. Garfield Slope Housing Corp.</i> , No. CV 94-4009(SMG), 1998 WL 623589 (E.D.N.Y. Aug. 17, 1998).....	13
11	<u>Rules</u>	
12	Fed. R. Civ. P. 26(a)(2)(B)	22
13	Fed. R. Civ. P. 56(a)	39
14	Fed. R. Evid. 104(a).....	7
15	Fed. R. Evid. 402	8
16	Fed. R. Evid. 702	1, 7, 8
17	<u>Other Authorities</u>	
18	Acquavella, J. et al., <i>Glyphosate Biomonitoring for Farmers and Their Families: Results from the Farm</i>	
19	<i>Family Exposure Study</i> , 112 <i>Envtl. Health Persp.</i> 321 (2004)	5
20	Alavanja, M. et al., <i>DRAFT-Lymphoma risk and pesticide use in the Agricultural Health Study</i> (Mar. 15,	
21	2013)	14
22	Alavanja, M. et al., <i>Non-Hodgkin Lymphoma Risk and Insecticide, Fungicide and Fumigant Use in the</i>	
23	<i>Agricultural Health Study</i> , 9 <i>PLoS One</i> (2014).....	6
24	Arnason, R., <i>Toxicologist Pans UN Glyphosate Report</i> , <i>The Western Producer</i> (Mar. 27, 2015),	
25	http://www.producer.com/daily/toxicologist-pans-un-glyphosate-report/	36
26	Berlin, J. et al., <i>The Use of Meta-Analysis in Pharmacoepidemiology</i> , <i>Pharmacoepidemiology</i> (5th ed.	
27	2012)	19
28		

1	Black, B. et al.,	
2	<i>Expert Evidence: A Practitioner's Guide to Law, Science, and the FJC Manual</i>	
	(1997).....	20
3	Blair, A. et al.,	
4	<i>Epidemiologic Studies of Cancer in Agric. Populations: Observations and Future</i>	
	<i>Directions</i> , 14 J. Agromedicine 125 (2009).....	6
5	Bolognesi, C. et al.,	
6	<i>Biomonitoring of Genotoxic Risk in Agricultural Workers from Five Columbian</i>	
	<i>Regions: Association to Occupational Exposure to Glyphosate</i> , 72 J. Toxicology	
	Envtl. Health, Part A 986 (2009)	35
7	Bolognesi, C. et al.,	
8	<i>Genotoxic Activity of Glyphosate and Its Technical Formulation Roundup</i> , 45 J.	
	Agric. & Food Chem. 1957 (1997).....	35
9	Brusick, D. et al.,	
10	<i>Genetic Toxicology in Hayes' Principles and Methods of Toxicology</i> (6th ed. 2014)	33
11	Cantor, K. et al.,	
12	<i>Pesticides and Other Agric. Risk Factors for Non-Hodgkin's Lymphoma Among Men</i>	
	<i>in Iowa and Minn.</i> , 52 Cancer Res. 2447 (1992).....	6
13	Chan, P. et al.,	
14	<i>NTP Tech. Report on Toxicity Studies of Glyphosate Administered in Dosed Feed to</i>	
	<i>F344/N Rats and B6C3F1 Mice</i> , 16 Toxicity Reports Series (1992),	
	https://ntp.niehs.nih.gov/ntp/htdocs/st_rpts/tox016.pdf	5
15	Chang, E. et al.,	
16	<i>Meta-Analysis of Glyphosate Use and Risk of Non-Hodgkin Lymphoma</i> , Exponent	
	(2017).....	21
17	Chang, E. et al.,	
18	<i>Systematic review and meta-analysis of glyphosate exposure and risk of</i>	
	<i>lymphohematopoietic cancers</i> , 51 J. Env'tl. Sci. & Health 402 (2016).....	19
19	De Roos, A. et al.,	
20	<i>Cancer Incidence among Glyphosate-Exposed Pesticide Applicators in the</i>	
	<i>Agricultural Health Study</i> , 113 Env'tl Health Perspectives 49 (2005)	12, 13, 14
21	Eaton, D.,	
22	<i>Scientific Judgment and Toxic Torts – A Primer in Toxicology for Judges and</i>	
	<i>Lawyers</i> , 12 J.L. & Pol'y 5 (2003).....	33
23	Eriksson, M. et al.,	
24	<i>Pesticide exposure as risk factor for non-Hodgkin lymphoma including</i>	
	<i>histopathological subgroup analysis</i> , 123 Int. J. Cancer 1657 (2008)	16, 17
25	EPA, Defining Pesticide Biomarkers: Biomarkers of Effect Categories,	
26	https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/defining-	
	pesticide-biomarkers	31
27	EPA Office of Pesticide Programs,	
28	<i>Glyphosate Issue Paper: Evaluation of Carcinogenic Potential</i> (Sept. 12, 2016),	
	https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0385-0094	passim

1	EPA Reregistration Eligibility Decision (RED) Glyphosate (Sept. 1993), https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/red_PC-417300_1-Sep-93.pdf	5, 25
2		
3	European Chemicals Agency, <i>Opinion Proposing Harmonized Classification and Labelling at EU Level of</i> <i>glyphosate (ISO); N-(phosphonomethyl) glycine</i> (Mar. 15, 2017), https://echa.europa.eu/documents/10162/2d3a87cc-5ca1-31d6-8967-9f124f1ab7ae	2
4		
5	European Food Safety Authority, <i>Conclusion on the peer review of the pesticide risk assessment of the active substance</i> <i>glyphosate</i> , 13(11) EFSA J. 4302 (2015), http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2015.4302/epdf	2, 5, 31
6		
7		
8	Green, M. et al., <i>Reference Guide on Epidemiology, in Reference Manual on Scientific Evidence</i> (3d ed. 2011), https://www.fjc.gov/sites/default/files/2015/SciMan3D01.pdf	passim
9		
10	Hill, A. Bradford, <i>The Environment and Disease: Association or Causation?</i> , 58 Proc. R. Soc. Med. 295 (1965).....	37
11		
12	Huff, J., Jameson, C. et al., <i>Carcinogenesis Studies: Results of 398 Experiments on 104 Chemicals from the U.S.</i> <i>National Toxicology Program</i> , Nat'l Inst. Env'tl. Health Sci., Nat'l Toxicology Program (1988)	30, 31
13		
14	IARC, <i>Agents Classified by the IARC Monographs, Volumes 1-119</i> (June 28, 2017), https://monographs.iarc.fr/ENG/Classification/ClassificationsAlphaOrder.pdf	3
15		
16	IARC, <i>IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Preamble</i> (Jan. 2006), http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf	3
17		
18	IARC, <i>Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate,</i> <i>Malathion, Parathion, and Tetrachlorvinphos</i> , Monograph Vol. 112 on the <i>Evaluation of Carcinogenic Risks to Humans</i> (2015), http://monographs.iarc.fr/ENG/Monographs/vol112/mono112-10.pdf	3, 19
19		
20	Joint Management of Pesticide Residues, <i>Pesticide residues in food – 2004, Joint FAO/WHO Meeting on Pesticide Residues</i> (2006), http://apps.who.int/iris/bitstream/10665/43624/1/9241665203_eng.pdf	2, 31
21		
22	Klaunig, J. et al., <i>Oxidative Stress and Oxidative Damage in Chemical Carcinogenesis</i> , 254 Toxicology and Applied Pharmacology 86 (2011)	32
23		
24	Leukemia & Lymphoma Society, NHL, https://www.lls.org/lymphoma/non-hodgkinlymphoma?src1=20045&src2=	6
25		
26	Ltr. from Bernhard Url, Exec. Director, EFSA, to Prof. Christopher J. Portier, Senior Consulting Scientist, Env'tl. Def. Fund (Jan. 13, 2016), https://www.efsa.europa.eu/sites/default/files/EFSA_response_Prof_Portier.pdf	29
27		
28		

1	Morse, H. et al.,	
2	<i>B Lymphoid Neoplasms of Mice: Characteristics of Naturally Occurring and</i>	
	<i>Engineered Diseases and Relationships to Human Disorders</i> , 81 <i>Advances in</i>	
	<i>Immunol.</i> 97 (2003)	30
3	New Zealand EPA,	
4	<i>Review of the Evidence Relating to Glyphosate and Carcinogenicity</i> (Aug. 11, 2016),	
	http://www.epa.govt.nz/Publications/EPA_glyphosate_review.pdf	2
5	NIH, <i>Cancer Stat Facts: Non-Hodgkin Lymphoma</i> ,	
6	https://seer.cancer.gov/statfacts/html/nhl.html	6
7	NIH, <i>Adult NHL Treatment</i> ,	
	https://www.cancer.gov/types/lymphoma/patient/adult-nhl-treatment-pdq#section/all	6
8	Nuzzo, R.,	
9	<i>Statistical Errors</i> , 506 <i>Nature</i> 150 (2014)	26
10	Orsi, L. et al.,	
11	<i>Occupational exposure to pesticides and lymphoid neoplasms among men: results of</i>	
	<i>a French case-control study</i> , 66 <i>Occup. Environ. Med.</i> 291 (2009)	16
12	Pahwa, M. et al.,	
13	<i>An Evaluation of Glyphosate Use and the Risk of Non-Hodgkin Lymphoma Major</i>	
	<i>Histological Sub-Types in the North American Pooled Project</i> (Aug. 31, 2015).....	16
14	Paz-y-Mino, C. et al.,	
15	<i>Baseline determination in social, health, and genetic areas in communities affected</i>	
	<i>by glyphosate aerial spraying on the northeastern Ecuadorian border</i> , 26 <i>Rev. Env'tl.</i>	
	<i>Health</i> 45 (2011)	36
16	Schinasi, L. et al.,	
17	<i>Non-Hodgkin Lymphoma and Occupational Exposure to Agricultural Pesticide</i>	
18	<i>Chemical Groups and Active Ingredients: A Systematic Review and Meta-Analysis</i> ,	
	11 <i>Int'l J. Env'tl. Res. & Public Health</i> 4449 (2014).....	19
19	Wasserstein, R. et al.,	
20	<i>Statement on p-values: Context, Process, and Purpose</i> , 70 <i>Amer. Statistician</i> 129	
	(2016).....	28
21	Weisenburger, D.,	
22	<i>An Epidemic of Non-Hodgkin's Lymphoma: Comments on Time Trends, Possible</i>	
	<i>Etiologies, and the Role of Pathology</i> , 5 <i>Mod. Pathol.</i> 481 (1992)	6

ISSUES TO BE DECIDED

- I. Whether plaintiffs have satisfied their burden to present expert testimony that is scientifically reliable and relevant within the meaning of *Daubert* and that is sufficient to prove general causation, *i.e.*, “whether there is sufficient admissible evidence that glyphosate and/or Roundup is capable of causing cancer (specifically, Non-Hodgkin’s Lymphoma [“NHL”]) in humans.” Pretrial Order 15 (filed Mar. 3, 2017), ECF No. 186.
- II. Whether plaintiffs’ failure to present sufficient admissible expert testimony to prove general causation entitles Monsanto Company (“Monsanto”) to summary judgment in all Roundup[®] lawsuits pending before this Court.

INTRODUCTION

As Justice Breyer stated, the essential gatekeeping role bestowed on district courts in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), ensures that “the powerful engine of tort liability . . . points toward the right substances and does not destroy the wrong ones.” *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 148-49 (1997) (Breyer, J., concurring). The *Daubert* trilogy – *Daubert*, *Joiner*, and *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999) – “shift[ed] the focus to the kind of empirically supported, rationally explained reasoning required in science, [which] has greatly improved the quality of the evidence upon which juries base their verdicts.” *Rider v. Sandoz Pharm. Corp.*, 295 F.3d 1194, 1197 (11th Cir. 2002). “Under *Daubert*, the trial court must act as a ‘gatekeeper’ to exclude junk science that does not meet Federal Rule of Evidence 702’s reliability standards by making a preliminary determination that the expert’s testimony is reliable.” *Ellis v. Costco Wholesale Corp.*, 657 F.3d 970, 982 (9th Cir. 2011).

In this case, plaintiffs’ expert witnesses present speculation and self-selected bits of data, offer subjective opinions, and selectively parrot third-party “conclusions” in a transparent effort to side-step their obligations to demonstrate reliable methodologies of their own. Plaintiffs designated six retained experts, with specialties designated by plaintiffs’ counsel as follows: Dr. Alfred Neugut (“Epidemiology”), Dr. Beate Ritz (“Epidemiology”), Dr. Christopher Portier (“Toxicology”), Dr. Charles Jameson (“Toxicology”), Dr. Chadi Nabhan (“Oncology and

NHL”), and Dr. Dennis Weisenburger (“Pathology and NHL”). Letter from Robin Greenwald to Heather Pigman (May 16, 2017) (Hollingsworth Decl., Ex. 1) (“5/16/17 Letter”). Plaintiffs also designated Dr. Aaron Blair (epidemiology) and Dr. Matthew Ross (mechanistic data) as non-retained experts. These experts collectively piece together an evidence trail which they say supports their opinion that glyphosate indeed causes NHL in humans.

Plaintiffs’ experts’ opinions stand in stark contrast to decisions repeatedly and consistently reached over a period of 40 years by regulatory agencies worldwide – including the United States Environmental Protection Agency (“EPA”) and those similarly tasked with human health and environmental protection in Canada, Australia, New Zealand, Korea, Japan, and the European Union. **Every** major regulatory agency charged with answering the question has, with the benefit of all the available primary data, concluded that glyphosate is **not** likely to pose risks of carcinogenicity, including NHL, in humans.¹ Non-regulatory bodies concerned with public health have reached the same conclusions.² Plaintiffs’ experts endorse the sole outlying assessment reported by a working group of the self-governing International Agency for Research on Cancer (“IARC”).³ This working group: (1) was chaired by Dr. Blair, who admits

¹ See, e.g., EPA Office of Pesticide Programs, *Glyphosate Issue Paper: Evaluation of Carcinogenic Potential* at 140 (Sept. 12, 2016), <https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0385-0094> (“EPA OPP”) (“strongest support is for ‘not likely to be carcinogenic to humans’ at the doses relevant to human health risk assessment”); European Food Safety Authority, *Conclusion on the peer review of the pesticide risk assessment of the active substance glyphosate*, 13(11) EFSA J. 4302 at 11 (2015), <http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2015.4302/epdf> (“EFSA 2015”) (“glyphosate is unlikely to pose a carcinogenic hazard to humans”); European Chemicals Agency, *Opinion Proposing Harmonized Classification and Labelling at EU Level of glyphosate (ISO); N-(phosphonomethyl) glycine* at 31 (Mar. 15, 2017), <https://echa.europa.eu/documents/10162/2d3a87cc-5ca1-31d6-8967-9f124f1ab7ae> (“based on the epidemiological data as well as on data from long-term studies in rats and mice ... no hazard classification for carcinogenicity is warranted for glyphosate”); New Zealand EPA, *Review of the Evidence Relating to Glyphosate and Carcinogenicity* at 16 (Aug. 11, 2016), http://www.epa.govt.nz/Publications/EPA_glyphosate_review.pdf (“The overall conclusion is that ... glyphosate is unlikely to be genotoxic or carcinogenic to humans and does not require classification ... as a carcinogen or mutagen.”).

² E.g., Joint Management of Pesticide Residues, *Pesticide residues in food – 2004, Joint FAO/WHO Meeting on Pesticide Residues* at 158 (2006), http://apps.who.int/iris/bitstream/10665/43624/1/9241665203_eng.pdf, (“JMPR”) (“In view of the absence of a carcinogenic potential in animals and the lack of genotoxicity in standard tests, the Meeting concluded that glyphosate is unlikely to pose a carcinogenic risk to humans.”).

³ IARC, *Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos, Monograph Vol. 112 on the Evaluation of Carcinogenic Risks*

1 to hiding epidemiology data that shows no increased risk of NHL attributable to glyphosate; (2)
 2 included Dr. Portier, who at the time worked for an environmental activist group opposed to the
 3 use of pesticides, Dep. of Christopher Portier 26:9-18 (Sept. 5, 2017) (Hollingsworth Decl., Ex.
 4 2) (“Portier Dep.”), and already was engaged by Plaintiffs’ counsel in other litigation connected
 5 to an IARC review, *id.* 75:14-77:2; and (3) reached its conclusion during a week-long meeting
 6 that considered five total compounds, without its members reviewing published primary long-
 7 term rodent bioassay data or many of the valid regulatory mechanistic studies.⁴

8 Monsanto recognizes that neither the extraordinary catalog of regulatory agencies’
 9 decisions nor IARC’s surprising conclusions can or should substitute for the analysis required
 10 by *Daubert*. However, it is clear that in bootstrapping IARC’s methodology and then
 11 embracing it as their own, each of plaintiffs’ experts employ a hazard assessment methodology
 12 (which is a “first step” that does not take into account a variety of important scientific factors,
 13 such as human relevance, in hypothesizing about causation);⁵ this fails *Daubert* because they
 14 have applied a “*threshold of proof*” that is “*lower than that appropriate in tort law.*” *Johnson v.*
 15 *Arkema, Inc.*, 685 F.3d 452, 464 (5th Cir. 2012) (emphasis in original) (quoting *Allen v. Pa.*
 16 *Eng’g Corp.*, 102 F.3d 194, 198 (5th Cir. 1996)). Indeed, three of plaintiffs’ experts (Drs.
 17 Jameson, Neugut, and Nabhan) admit they **only** performed hazard assessments;⁶ their opinions

18
 19 to Humans (2015), <http://monographs.iarc.fr/ENG/Monographs/vol112/mono112-10.pdf> (“IARC
 Monograph 112”).

20 ⁴ See *infra* at n.21 (Dr. Blair’s decision to hide data); Portier Dep. 29:23-30:17 (work for
 Environmental Defense Fund); *infra* at 29 (limitations of IARC review).

21 ⁵ IARC intends its hazard assessment methodology to be only a “first step” in assessing the
 22 carcinogenic potential of a compound. IARC, *IARC Monographs on the Evaluation of*
 23 *Carcinogenic Risks to Humans Preamble* at 2 (Jan. 2006), [http://monographs.iarc.fr/ENG/](http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf)
 24 [Preamble/CurrentPreamble.pdf](http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf) (“IARC Preamble”). A hazard assessment finding that a
 25 compound is “probably carcinogenic” in fact has “no quantitative significance.” IARC Preamble
 26 at 22. Thus, IARC classifies a wide variety of commonly-used substances and exposures as
 27 “carcinogenic” or “probably carcinogenic” to humans, including bacon, hot dogs, and red meat;
 alcoholic beverages; salted fish; shiftwork; frying food; and certain hot beverages. IARC,
 28 *Agents Classified by the IARC Monographs, Volumes 1-119*, 1, 16, 29, 30, 31, 35 (June 28,
 2017), <https://monographs.iarc.fr/ENG/Classification/ClassificationsAlphaOrder.pdf>; see
 Monsanto Company’s Brief Regarding the Relevance of IARC and EPA to General Causation
 (Feb. 8, 2017), ECF No. 134.

⁶ See, e.g., Dep. of Alfred Neugut 254:22-256:14 (Aug. 7, 2017) (Hollingsworth Decl., Ex. 3)
 (“Neugut Dep.”) (“[I] tried to adhere to their [IARC’s] criteria and methodologies for
 establishing, I guess what I would consider to be public policy, as well as judgments with regard

1 should be excluded on this basis alone. *Id.*

2 Plaintiffs’ experts relied on three categories of evidence (epidemiology studies, rodent
3 carcinogenicity data, and data from mechanism studies), but the general causation opinions that
4 they cobbled together fail to satisfy *Daubert* – no matter what methodology they claim to apply
5 – because the opinions are not the product of reliable science and do not have a valid scientific
6 connection to the pertinent inquiry at issue here. The examples of these flaws are many:

- 7
- 8 • they rely on epidemiological data that lacks statistical significance and/or is not properly
9 controlled for known confounding variables, which violates bedrock principles of both
10 epidemiology and *Daubert*, *infra* at 15-21;
 - 11 • they rely on untested statistical methodologies to interpret animal carcinogenicity data,
12 changing such methodologies over time in a blatant results-driven approach, *infra* at 24-29;
 - 13 • they selectively chose to include or exclude data in any given statistical analysis, thereby
14 ultimately applying different tests in different situations, again solely to achieve a pre-
15 conceived result that supports their opinions, *infra* at 26-27;
 - 16 • they improperly extrapolate data from animal tests to human risk, without any citation to any
17 literature or support for said extrapolation, *infra* at 21-23, 29-30;
 - 18 • they completely ignore relevant human exposure levels in their analyses despite recognizing
19 their importance in any risk or causation assessment, *infra* at 5, 29-30, 34-35; and
 - 20 • their opinions have never been published, tested, peer-reviewed or generally accepted outside
21 the confines of this litigation, confirming they were created solely for litigation, *infra* at 23,
22 26-28, 35, 38-39.

23 Any one of these flaws is sufficient to exclude plaintiffs’ experts; collectively they
24 demand exclusion. Since plaintiffs’ experts’ opinions “are no more than educated guesses
25 dressed up in evening clothes,” they fail to satisfy *Daubert*. *Hall v. Baxter Healthcare Corp.*,
26 947 F. Supp. 1387, 1407 (D. Or. 1996).

27 **BACKGROUND OF GLYPHOSATE AND GLYPHOSATE-BASED HERBICIDES**

28 Glyphosate-based herbicides (“GBHs”) became commercially available in 1974 when
Monsanto introduced Roundup[®], a mixture of glyphosate and surfactants (chemical compounds
commonly found in products such as soaps that allow glyphosate to travel on the surface of the
weed to growing areas). In the forty years since its initial registration, a robust scientific

to this issue”); Dep. of Chadi Nabhan 257:7-258:22 (Aug. 23, 2017) (Hollingsworth Decl., Ex. 4) (“Nabhan Dep.”) (describing application of hazard assessment methodology); Expert Report of Charles Jameson *passim* (Hollingsworth Decl., Ex. 5) (“Jameson Report”) (stating that he conducted a hazard assessment at least 15 times); Dep. of Charles Jameson *passim* (Sept. 21, 2017) (Hollingsworth Decl., Ex. 6) (“Jameson Expert Dep.”) (describing his methodology as hazard assessment at least 43 times).

1 database has developed for glyphosate and GBHs, including multiple published human
 2 population studies (epidemiology), over a dozen long-term rodent carcinogenicity bioassays,
 3 and hundreds of mechanistic studies.⁷

4 It is undisputed that the bioavailability of glyphosate is extremely low, meaning that
 5 very little of the chemical is absorbed and circulated in the human system.⁸ Even the heaviest
 6 users of GBHs absorb relatively small systemic doses from all possible routes of exposure. For
 7 example, the Farm Family Exposure Study evaluated urinary concentrations for farmers and
 8 their families, with urine samples taken the day before, the day of, and for three days after a
 9 GBH application.⁹ The authors found that “the highest estimated systemic dose was 0.004
 10 mg/kg.” *Id.* No comparable dose is considered clinically meaningful to human health by any
 11 regulatory entity; for example, “[n]one of the systemic doses estimated in this study approached
 12 the [EPA] reference dose [which includes a hundred-fold safety factor] for glyphosate of 2
 13 mg/kg/day.” *Id.*

14 Plaintiffs’ allegations are based only on dermal exposure. See Transcript of Proceedings
 15 at 10 (Feb. 24, 2017) (Hollingsworth Decl., Ex. 7). Yet GBHs are “poorly absorbed dermally”
 16 and contribute minimally (if at all) to a hypothetical systemic dose. EPA Reregistration
 17 Eligibility Decision (RED) Glyphosate at 21 (Sept. 1993), [https://www3.epa.gov/pesticides/](https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/red_PC-417300_1-Sep-93.pdf)
 18 [chem_search/reg_actions/reregistration/red_PC-417300_1-Sep-93.pdf](https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/red_PC-417300_1-Sep-93.pdf) (“EPA RED”). “Dermal
 19 penetration has also been shown to be relatively low for human skin (<1%), indicating dermal
 20 exposure will only contribute slightly to a systemic biological dose.” EPA OPP at 15.
 21 Approximately 90% of what remains is eliminated in urine within 6 hours.¹⁰

22 ⁷ See EPA OPP at 130 (“A large database is available for evaluating the carcinogenicity potential
 23 of glyphosate.”); EFSA 2015 at 10 (“The glyphosate dossier consists of an exceptionally large
 24 database, therefore the toxicological evaluation ... rel[ies] on a magnitude of valid studies rather
 than on one ‘key study’ for each endpoint.”). Many of these studies were conducted by
 independent investigators neither working with nor funded by Monsanto.

25 ⁸ See EPA OPP at 15 (glyphosate’s oral, inhalation, and dermal exposure profile “suggests that
 there is low potential for a sustainable biological dose following glyphosate exposure.”).

26 ⁹ See J. Acquavella et al., *Glyphosate Biomonitoring for Farmers and Their Families: Results*
from the Farm Family Exposure Study, 112 *Envtl. Health Persp.* 321, 321 (2004).

27 ¹⁰ P. Chan et al., *NTP Tech. Report on Toxicity Studies of Glyphosate Administered in Dosed*
Feed to F344/N Rats and B6C3F1 Mice, 16 *Toxicity Reports Series* at 18 (1992),
 28 https://ntp.niehs.nih.gov/ntp/htdocs/st_rpts/tox016.pdf (describing rapid elimination).

BACKGROUND REGARDING NON-HODGKIN'S LYMPHOMA

NHL is a highly diverse group of blood cancers classified into more than 60 distinct subtypes. Collectively, NHL is the seventh most common cancer and adults have approximately a 2.1% chance of developing NHL during their lifetimes. The cause of most NHL cases is not known. There are, however, several established risk factors that may increase a person's likelihood of developing the disease. Aging is an important risk factor. People with autoimmune disease, acquired immunodeficiencies (HIV/AIDS), and organ transplant recipients have an elevated risk for NHL. External factors that suppress the immune system (*i.e.*, chemotherapy or treatments for autoimmune diseases) may contribute to NHL's development. Viral infections, such as Hepatitis C, also play a role.¹¹

Farming also has long been considered a potential risk factor for NHL, with several epidemiology studies showing a small but statistically significant increased risk. *See* M. Alavanja et al., *Non-Hodgkin Lymphoma Risk and Insecticide, Fungicide and Fumigant Use in the Agricultural Health Study*, 9 PLoS One 1 (2014). Scientists identified this potential risk factor, and many of the studies were undertaken, **before** the introduction of GBHs. *See, e.g.*, K. Cantor et al., *Pesticides and Other Agric. Risk Factors for Non-Hodgkin's Lymphoma Among Men in Iowa and Minn.*, 52 Cancer Res. 2447, 2448 Table 2 (1992) (farmers who ceased farming between 1950-69 had a statistically significant increased risk of NHL). Multiple hypotheses, including by some of plaintiffs' experts, have been raised to explain this positive association, including exposure to diesel fumes from farming equipment, farm animals, UV rays, and a range of other pesticides, herbicides, and insecticides.¹²

¹¹ *See generally* NIH, *Cancer Stat Facts: Non-Hodgkin Lymphoma*, <https://seer.cancer.gov/statfacts/html/nhl.html>; NIH, *Adult NHL Treatment*, <https://www.cancer.gov/types/lymphoma/patient/adult-nhl-treatment-pdq#section/all>; Leukemia & Lymphoma Society, *NHL*, <https://www.lls.org/lymphoma/non-hodgkinlymphoma?src1=20045&src2=>.

¹² *See* A. Blair et al., *Epidemiologic Studies of Cancer in Agric. Populations: Observations and Future Directions*, 14 J. Agromedicine 125, 128 (2009). These hypotheses implicate exposure to a plethora of agricultural chemicals other than GBHs, including atrazine, carbaryl, lindane, 2,4-D, and chlorophenols. *See id.* at 127-28; D. Weisenburger, *An Epidemic of Non-Hodgkin's Lymphoma: Comments on Time Trends, Possible Etiologies, and the Role of Pathology*, 5 Mod. Pathol. 481 (1992).

1 **STANDARDS GOVERNING ADMISSIBILITY OF EXPERT TESTIMONY**

2 In *Daubert*, the Supreme Court addressed the admissibility of expert testimony and
 3 established “the exacting standards of reliability such evidence must meet.” *Weisgram v.*
 4 *Marley Co.*, 528 U.S. 440, 455 (2000). Even where a party retains an apparently well-
 5 credentialed witness, “the trial court’s gatekeeping function requires more than simply taking
 6 the expert’s word for it.” Fed. R. Evid. 702 advisory committee’s note (2000 amendment).
 7 Stated differently, “nothing in either *Daubert* or the Federal Rules of Evidence requires a
 8 district court to admit opinion evidence that is connected to existing data only by the *ipse dixit*
 9 of the expert.” *Joiner*, 522 U.S. at 146 (rejecting causation opinion based on non-statistically
 10 significant epidemiology and animal studies that plaintiffs’ experts could not reliably
 11 extrapolate to humans). Instead, courts are required to ensure that an expert “employs in the
 12 courtroom the same level of intellectual rigor that characterizes the practice of an expert in the
 13 relevant field.” *Kumho Tire*, 526 U.S. at 152. A court resolves a *Daubert* challenge as a
 14 “preliminary” admissibility question under Federal Rule of Evidence 104(a), *see Daubert*, 509
 15 U.S. at 592 & n.10 (quoting Rule 104(a)); thus the proponent of the testimony does not benefit
 16 from any inferences in its favor. Moreover, the burden is affirmative and cannot be carried by
 17 mere attacks on the opposing side. *See, e.g., Caraker v. Sandoz Pharm. Corp.*, 188 F. Supp. 2d
 18 1026, 1034 (S.D. Ill. 2001) (“Plaintiffs’ experts’ broad criticisms of the existing
 19 epidemiological evidence do[] not help them meet their burden,” as “plaintiffs’ burden is an
 20 affirmative one, not served by such attacks.”).

21 As the proponents of the expert testimony at issue here, plaintiffs have the burden of
 22 proving that it is admissible under *Daubert* and its progeny. *See, e.g., Daubert*, 509 U.S. at 592
 23 n.10; *Bldg. Indus. Ass’n v. Wash. State Bldg. Code Council*, 683 F.3d 1144, 1154 (9th Cir.
 24 2012). This burden requires plaintiffs to make three kinds of showings.

25 **First**, plaintiffs must establish that the witness has the “knowledge, skill, experience,
 26 training, or education,” Fed. R. Evid. 702, to render an opinion on the specific issue addressed
 27 by his testimony. *See Avila v. Willitts Env’tl. Remediation Trust*, 633 F.3d 828, 839 (9th Cir.
 28 2011) (affirming exclusion of toxicology opinions of doctor with degrees in chemistry).

1 **Second**, plaintiffs must establish scientific reliability – *i.e.*, that the expert’s testimony is
 2 “ground[ed] in the methods and procedures of science,” not “subjective belief or unsupported
 3 speculation.” *Daubert*, 509 U.S. at 589-90 (quotation marks omitted); *see* Fed. R. Evid. 702
 4 (requiring that expert testimony be “based on sufficient facts or data” and “the product of
 5 reliable principles and methods” and that the expert “has reliably applied the principles and
 6 methods to the facts of the case”). The *Daubert* Court identified four non-exhaustive factors for
 7 courts to consider when evaluating scientific reliability: (1) whether the expert’s theory can be
 8 and has been tested, because “[s]cientific methodology today is based on generating hypotheses
 9 and testing them to see if they can be falsified;” (2) whether the theory “has been subjected to
 10 peer review and publication” because “submission to the scrutiny of the scientific community is
 11 a component of ‘good science;” (3) the known or potential error rate of the expert’s technique;
 12 and (4) whether the theory has attained “general acceptance” in the scientific community.
 13 *Daubert*, 509 U.S. at 593-94. As the Ninth Circuit stated, “we must determine nothing less than
 14 whether the experts’ testimony reflects ‘scientific knowledge,’” *Daubert v. Merrell Dow*
 15 *Pharm., Inc.*, 43 F.3d 1311, 1315 (9th Cir. 1995) (“*Daubert II*”) (quoting *Daubert*, 509 U.S. at
 16 590), and “something doesn’t become ‘scientific knowledge’ just because it’s uttered by a
 17 scientist,” *id.* at 1315-16. “Under *Daubert*, any step that renders the analysis unreliable . . .
 18 renders the expert’s testimony inadmissible. This is true whether the step completely changes a
 19 reliable methodology or merely misapplies that methodology.” *Mitchell v. Gencorp Inc.*, 165
 20 F.3d 778, 782 (10th Cir. 1999) (internal quotation omitted) (ellipsis in original); *Burst v. Shell*
 21 *Oil Co.*, 650 F. App’x 170, 174 (5th Cir. 2016) (same) (quotation omitted).

22 **Third**, plaintiffs must satisfy the “fit” requirement by establishing that the expert’s
 23 testimony assists the trier of fact by having “a valid scientific connection to the pertinent
 24 inquiry.” *Daubert*, 509 U.S. at 591-92. “*Daubert* stressed the importance of the ‘fit’ between
 25 the testimony and an issue in the case.” *Daubert II*, 43 F.3d at 1320. The “fit” requirement is
 26 directed to **scientific** relevance and “is more stringent than the relevancy requirement of [FRE
 27 402], reflecting the special dangers inherent in scientific expert testimony.” *FTC v. Wellness*
 28 *Support Net., Inc.*, Case No. 10-cv-04879-JCS, 2013 WL 5513332, at *9 (N.D. Cal. Oct. 4,

2013) (internal quotation omitted); see *Daubert II*, 43 F.3d at 1321 n.17 (same).

A crucial component of “fit” in product liability litigation is that general causation must be assessed at human-relevant doses. As Judge Breyer has stated, “dose matters” in *Daubert* assessments, particularly where, as here, “all of plaintiffs’ experts ... agree that there is a dose effect.”¹³ *In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1174 (N.D. Cal. 2007).¹⁴ In *In re Hanford Nuclear Reservation Litigation*, the Ninth Circuit explained fit in the context of the general causation inquiry as “whether exposure to a substance for which a defendant is responsible, such as radiation **at the level alleged by the plaintiffs**, is capable of causing a particular injury or condition in the general population.” 292 F.3d 1124, 1133 (9th Cir. 2002) (emphasis added). Thus, only real-world exposure levels are “relevant to the task at hand,” *Daubert*, 509 U.S. at 597 – here, assessing whether exposure to GBHs can cause NHL.

¹³ *E.g.*, Amended Expert Report of Christopher Portier at 22-51 (Hollingsworth Decl., Ex. 8) (“Portier Amended Report”) (identifying various “dose-related trends”); Jameson Report at 21-29 (same); Expert Report of Beate Ritz at 23 (Hollingsworth Decl., Ex. 9) (“Ritz Report”) (dose response observed in epidemiology studies); Expert Report of Chadi Nabhan at 21 (Hollingsworth Decl., Ex. 10) (“Nabhan Report”) (“Dose response effect is seen in some case-control studies”); Expert Report of Alfred Neugut at 22 (Hollingsworth Decl., Ex. 11) (“Neugut Report”) (epidemiology studies “suggest that there is a dose-response relationship”); Expert Report of Dennis Weisenburger at 4 (Hollingsworth Decl., Ex. 12) (“Weisenburger Report”) (“dose-response effect was evaluated” in epidemiology studies).

¹⁴ See also *McClain v. Metabolife Int’l, Inc.*, 401 F.3d 1233, 1242 (11th Cir. 2005) (“Often low dose exposures – even for many years – will have no consequence at all, since the body is often able to completely detoxify low doses before they do any damage.”) (quotation marks omitted); *Myers v. U.S.*, No. 02CV1349-BEN, 2014 WL 6611398 at *46 (S.D. Cal. Nov. 20, 2014) (The fact that thallium was present in plaintiff’s urine “is a *non sequitur*. It is not the presence of thallium, it is the dose that matters.”); *In re Zicam Cold Remedy Mktg., Sales Practices & Prod. Liab. Litig.*, 797 F. Supp. 2d 940, 945-46 (D. Ariz. 2011) (“In order to explain how a pervasive substance is harmful, one must show that at a particular level of exposure, the substance becomes toxic. Without requiring this kind of evidence, the door is open to meritless claims based on generally harmless levels of exposure.”); *Young v. Burton*, 567 F. Supp. 2d 121, 128-29 (D.D.C. 2008) (under *Daubert*, “scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff’s burden in a toxic tort case”); *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671, 679 (6th Cir. 2011) (same); *Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997) (same); *Allen*, 102 F.3d at 199 (same); *Wright v. Williamette Indus., Inc.*, 91 F.3d 1105, 1106 (8th Cir. 1996) (same).

ARGUMENT

I. PLAINTIFFS HAVE FAILED TO SATISFY THEIR BURDEN OF PRESENTING ADMISSIBLE EXPERT TESTIMONY TO PROVE GENERAL CAUSATION.

To admit plaintiffs’ general causation experts’ opinions here, the Court “would have to make several scientifically unsupported ‘leaps of faith’ . . . [but] [t]he *Daubert* rule requires more.” *Rider*, 295 F.3d at 1202. Therefore, and as discussed below, the Court should exclude plaintiffs’ experts’ general causation opinions in each of the three relevant scientific disciplines.

A. Plaintiffs’ Experts’ Results-Driven Methodologies Do Not And Cannot Reliably Account For The Enormous, Consistent, And Important Body Of Negative Epidemiology That Does Not Support Their General Causation Opinions.

“Epidemiology . . . is generally considered to be the best evidence of causation in toxic tort actions.” *Id.* at 1198; *Lopez v. Wyeth-Ayerst Lab.*, No. C 94–4054 CW, 1996 WL 784566, at *3 (N.D. Cal. Dec. 13, 1996) (citing *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307, 313 (5th Cir. 1989), *as modified* 884 F.2d 116 (5th Cir.1989) (“While we do not hold that epidemiologic proof is a necessary element in all toxic tort cases, it is certainly a very important element. This is especially true when the only other evidence is in the form of animal studies of questionable applicability to humans.”)), *aff’d*, 139 F.3d 905 (9th Cir. 1998). Epidemiologic studies measure “the strength of an association between exposure and disease” by calculating ratios (“relative risks” or “odds ratios”) comparing individuals with or without an exposure in relation to a disease outcome. *Caraker*, 188 F. Supp. 2d at 1031. A risk ratio of 1.0 “suggests that there is no association between a product and the disease.” *In re Bextra*, 524 F. Supp. 2d at 1173; *see also Caraker*, 188 F. Supp. 2d at 1031-32. Ratios above 1.0 can suggest a positive association (and below 1.0 can suggest a negative association) but “[b]efore any inferences are drawn about causation, the possibility of other reasons for the association must be examined, including chance, biases . . . , and confounding causes.” *Nelson v. Tenn. Gas Pipeline Co.*, 243 F.3d 244, 253 (6th Cir. 2001). In other words, statistical significance and proper controls are key to any epidemiological analysis.

Regarding statistical significance, epidemiologists account for chance by calculating a “confidence interval” around a point estimate of relative risk. As Judge Breyer has explained:

[I]f a given study show[s] a relative risk of 1.40 (a 40[%] increased risk of adverse events), but the 95[%] confidence interval is .8 to 1.9, we would say that we are 95 [%] confident that the true value, that is, the actual relative risk, is between .8 and 1.9. Because the confidence interval includes results which do not show any increased risk, and indeed, show a decreased risk, that is, it includes values less than 1.0, we would say the study does not demonstrate a “statistically significant” increased risk of an adverse outcome.

In re Bextra, 524 F. Supp. 2d at 1174; *In re Zolof Prods. Liab. Litig.*, 858 F.3d 787, 793 (3d Cir. 2017) (statistical significance is “an important metric to distinguish between results supporting a true association and those resulting from mere chance”). Regarding proper controls, an epidemiologic study cannot provide evidence of general causation unless “[it] properly accounts for potential confounding factors,” i.e., other exposures that might be the true explanation for a reported association. *In re Bextra*, 524 F. Supp. 2d at 1172.¹⁵

A plaintiff seeking to prove general causation absent statistically significant associations proven through epidemiology, as here, faces “a high bar . . . with respect to the [*Daubert*] reliability requirement.” *Siharath v. Sandoz Pharm. Corp.*, 131 F. Supp. 2d 1347, 1358 (N.D. Ga. 2001) (excluding epidemiology opinions lacking statistically significant support), *aff’d*, *Rider*, 295 F.3d 1194. Plaintiffs’ experts cannot overcome that bar.¹⁶ Indeed, as plaintiffs’ expert Dr. Neugut concedes, there is no pesticide-adjusted odds ratio ***anywhere in the***

¹⁵ See M. Green et al., *Reference Guide on Epidemiology*, in *Reference Manual on Scientific Evidence* 549, 591-93 (3d ed. 2011), <https://www.fjc.gov/sites/default/files/2015/SciMan3D01.pdf> (“Reference Manual”). Epidemiologists can attempt to control for confounding by conducting statistical analyses. Dep. of Beate Ritz 163:2-17 (Sept. 18, 2017) (Hollingsworth Decl., Ex. 13) (“Ritz Dep.”).

¹⁶ Three of plaintiffs’ retained experts – Dr. Jameson (chemist/toxicologist), Dr. Portier (toxicologist), and Dr. Nabhan (oncologist) – are not qualified to render epidemiology-based opinions because they do not have the requisite specialized knowledge or experience, see 5/16/17 Letter, as a review of their CVs confirms. Dr. Jameson was involved in the IARC Working Group animal subgroup, Fact Dep. of Charles Jameson 139:4-14 (May 3, 2017) (Hollingsworth Decl., Ex. 14) (“Jameson Fact Dep.”), but a different subgroup addressed epidemiology, *id.* 138:19-24, and he did not attend those discussions, *id.* 301-21:22. Dr. Nabhan readily admits that he has never been an epidemiologist. Nabhan Dep. 20:25-21:2. Plaintiff’s non-retained expert Dr. Ross (chemist and molecular toxicologist) also lacks epidemiology expertise. He does primarily “bench research” on animals – “[i]n test tubes [and] Petri dishes” – which “is not epidemiological research.” Dep. of Matthew Ross 12:13-14:19 (May 3, 2017) (Hollingsworth Decl., Ex. 15) (“Ross Dep.”). The Court should preclude these four witnesses from presenting epidemiology opinions. See *Avila*, 633 F.3d at 839 (witness excluded because he lacked expertise relevant to opinion that he sought to present); *Soldo v. Sandoz Pharm. Corp.*, 244 F. Supp. 2d 434, 571 (W.D. Pa. 2003) (same).

1 **epidemiologic literature** that reports a statistically significant positive association between
 2 glyphosate and NHL. Neugut Dep. 158:23-159:6; *see also id.* 45:14-18 (agreeing that one
 3 “would not label an exposure as being associated with an outcome unless there is a finding of an
 4 increased risk that is statistically significant”).¹⁷

5 **1. The most reliable epidemiology evidence – a major, ongoing prospective cohort**
 6 **study controlling for exposures to other pesticides – does not find any**
 7 **association between exposure to GBHs and NHL.**

8 The glyphosate epidemiologic literature is comprised of cohort (prospective) and case-
 9 control (retrospective) studies. As Dr. Neugut explains, cohort studies are “generally
 10 preferred,” as they are “more naturalistic,” “because the people are unbiased at the beginning of
 11 the study when you get your data.” Neugut Dep. 72:1-10, 73:17-74:9.

12 Here, powerful prospective cohort epidemiology, the “Agricultural Health Study”
 13 (“AHS”), exists – and finds no association between GBHs and NHL. AHS is funded by the U.S.
 14 government. Neugut Dep. 121:8-14. A 2005 report presented data for over 50,000 pesticide
 15 applicators, including 92 who developed NHL. *See De Roos 2005* at 49, 51 (Table 2).¹⁸ The
 16 authors reported that their data “provided **evidence of no association between glyphosate**
 17 **exposure and NHL incidence.**” *Id.* at 53 (emphasis added); *see id.* at 51 (Table 2) (multi-
 18 variable RR for NHL and ever/never use of glyphosate = 1.1 (95% CI 0.7-1.9) (not statistically
 19 significant)). Plaintiffs’ experts agree that the AHS study shows no association.¹⁹

19 ¹⁷ The Ninth Circuit has imposed an even higher bar, explaining that general causation cannot
 20 be established unless the epidemiologic data shows a statistically significant doubling of the
 21 risk. *Daubert II*, 43 F.3d at 1321 (under *Daubert*’s “fit” requirement, for epidemiology studies
 22 to support any general causation opinion, studies must show statistically significant RR in
 23 excess of 2.0 because a RR of less than 2.0 “may suggest [an adverse effect], but it actually
 24 tends to disprove legal causation, as it shows that [the chemical] does not double the likelihood
 25 of [the adverse effect].”); *see Schudel v. Gen. Elec. Co.*, 120 F.3d 991, 996 (9th Cir. 1997)
 26 (same), *abrogated on other grounds by Weisgram*, 528 U.S. 440.

24 ¹⁸ A. De Roos et al., *Cancer Incidence among Glyphosate-Exposed Pesticide Applicators in the*
 25 *Agricultural Health Study*, 113 *Envtl Health Perspectives* 49 (2005) (“De Roos 2005”).
 26 Although this article uses the term “glyphosate,” the study involved formulated products
 27 (GBHs), which are what pesticide applicators use. The epidemiology studies at issue in this
 28 litigation involve GBHs. Ritz Dep. 52:2-4.

26 ¹⁹ Neugut Dep. 128:1-7 (admitting lack of association in AHS analysis that controlled for other
 27 pesticides or other potential confounders); Dep. of Dennis Weisenburger 191:4-15 (Sept. 11,
 28 2017) (Hollingsworth Decl., Ex. 16) (“Weisenburger Dep.”) (admitting that De Roos 2005
 results “were negative” and that “there was no association found between glyphosate exposure
 and [NHL]”).

1 This study also reported data from various exposure analyses to account for the most
 2 highly exposed populations – “cumulative exposure days” and “intensity-weighted cumulative
 3 exposure days” – none of which showed a dose-response relationship between glyphosate
 4 exposure and NHL. See De Roos 2005 at 52 (“[t]here was ***no association between glyphosate***
 5 ***exposure and . . . NHL***, whether the exposure metric was ‘ever used,’ ‘cumulative exposure
 6 days,’ or ‘intensity-weighted cumulative exposure days’” (emphasis added)). Again, plaintiffs’
 7 experts acknowledge this is what the AHS data shows. Neugut Dep. 131:2-132:15, 133:4-8
 8 (admitting that AHS reported that subjects with higher durations and higher intensity of
 9 glyphosate exposure had lower incidence of NHL than subjects with lower durations and lower
 10 intensity of exposure); Dep. of Aaron Blair 155:25-157:21 (Mar. 20, 2017) (Hollingsworth Decl.,
 11 Ex. 17) (“Blair Dep.”) (same).

12 Plaintiffs’ experts also validate the AHS. According to Dr. Blair (one of the AHS co-
 13 authors), the AHS was initiated to address some of the limitations of earlier retrospective case-
 14 control studies regarding risks of pesticides or other exposures in farmers. See Blair Dep. 94:6-
 15 95:1, 95:23-96:1; see also Neugut Dep. 124:1-4. Unlike the case-control studies discussed
 16 below, the AHS results are not skewed by recall bias because information about exposures was
 17 collected from the participants before they were diagnosed with cancer.²⁰ See Neugut Dep.
 18 124:1-4 (AHS “was initiated to avoid the problem of recall bias in case-control studies”). In
 19 addition, the AHS methodology “appropriately controlled for lifestyle factors and multiple
 20 pesticide exposures [*i.e.*, exposures to chemicals other than glyphosate] in the statistical models,
 21 reducing the potential for confounding by other farm exposures associated with NHL.” Expert
 22 Report of Lorelei Mucci at 32 (Hollingsworth Decl., Ex. 18) (“Mucci Report”). Moreover, “the
 23 number of NHL cases [in the AHS] was sufficiently large to provide reasonable statistical
 24

25 ²⁰ In retrospective case-control studies, the quality of the data is determined to a large extent by
 26 the participants’ ability to accurately recall past exposures. The “recall bias” that occurs when
 27 the information provided on exposure differs between the study groups can severely undermine
 28 the reliability of the study results. See *Zwillinger v. Garfield Slope Housing Corp.*, No. CV 94-
 4009(SMG), 1998 WL 623589, at *18 (E.D.N.Y. Aug. 17, 1998). “Research has shown that
 individuals with disease (cases) tend to recall past exposures more readily than individuals with
 no disease (controls)” *Reference Manual* at 585 (footnotes omitted).

1 power,” and the study “had virtually complete follow-up of the cohort for cancer incidence and
2 mortality . . . which reduces selection bias.” *Id.* at 32-33.

3 Discovery in this litigation has brought to light an even more robust and more recent
4 independent analysis of AHS data. This analysis was prepared in 2013. See M. Alavanja et al.,
5 *DRAFT-Lymphoma risk and pesticide use in the Agricultural Health Study* (Mar. 15, 2013)
6 (unpublished study on file with authors) (“Alavanja 2013”). The analysis was subsequently
7 published as a study in the peer-reviewed literature, but only after the authors – one of whom is
8 Dr. Blair – removed the findings for glyphosate (and other herbicides) and substituted findings
9 for a different category of pesticides. See Blair Dep. 259:23-260:15; Mucci Report at 33.

10 The Alavanja 2013 cohort study likewise finds “***no evidence of association between***
11 ***exposure to glyphosate and NHL.***” Blair Dep. 172:11-15. Alavanja 2013 provides an
12 additional seven years of follow-up for NHL cases in the AHS cohort, Blair Dep. 167:21-
13 168:16, and is more than four times larger than De Roos 2005, *id.* 171:21-172:1. Dr. Blair
14 testified that the RR for an “ever/never” analysis of glyphosate and NHL based on the Alavanja
15 2013 data was below 1.0 – about 0.9. *Id.* 173:6-11. Moreover, “the applicators in highest levels
16 of exposure to glyphosate, both by lifetime days and intensity-weighted lifetime days, had the
17 exact same incidence of [NHL] as applicators with no exposure to glyphosate,” so a “***completely***
18 ***null result.***” *Id.* 173:12-23.²¹

19 Of course, despite recognizing certain advantages of the AHS over case-control studies
20 and despite agreeing that the report showed no association, plaintiffs’ experts still criticize the
21 AHS data in various ways. But attacking the results of the AHS does not satisfy plaintiffs’

22
23 ²¹ Dr. Blair admitted that the updated findings from the AHS for glyphosate and NHL were not
24 considered by the IARC working group that evaluated glyphosate in March 2015, even though
25 he was the chair of the working group – and even though he had reviewed the data and co-
26 authored a report on it in March 2013. Blair Dep. 176:5-177:25. Dr. Blair did not disclose even
27 the existence of the new information to any of his fellow working group members at IARC. *Id.*
28 177:13-178:7. Dr. Blair’s decision to withhold the finding astonishingly and unfortunately left
IARC with a skewed, incomplete set of available data for its glyphosate assessment. See Blair
Dep. 182:16-183:17 (admitting that 2013 AHS data would have reduced IARC’s reported meta-
relative risk and probably made it not statistically significant). Thus, to the extent that any of
plaintiffs’ experts bolster their general causation opinions with the conclusions in the IARC
monograph, those opinions are unreliable because the monograph is based on incomplete data
and totally undermined by the complete, most recent epidemiological data.

burden of presenting scientifically reliable expert testimony to prove general causation. *See, e.g., Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 886 (10th Cir. 2005) (“Mere criticism of epidemiology cannot establish causation.”); *Hollander v. Sandoz Pharm. Corp.*, 289 F.3d 1193, 1213 (10th Cir. 2002) (“[P]laintiffs have the burden of demonstrating the harmful effect of [the drug]. Accordingly, it was not unreasonable for the district court to conclude that [plaintiffs’ expert’s] attack on the [epidemiology] study did not constitute reliable [general causation] evidence . . .”); *see also Caraker*, 188 F. Supp. 2d at 1034 (same).

2. When properly adjusted for confounding, the case-control studies upon which plaintiffs’ experts rely likewise do not find any association between exposures to GBHs and NHL.

With independent, undisputed prospective data failing to lend any support to their hypotheses, plaintiffs’ experts accordingly rely instead on cherry-picked, unadjusted epidemiologic data from retrospective case-control studies that previously have been found insufficient to satisfy their *Daubert* burden. *Arias v. DynCorp*, 928 F. Supp. 2d 10, 24-25 (D.D.C. 2013) (excluding expert NHL causation opinion based upon studies at issue here). Case-control studies are generally considered less reliable than cohort studies because they are more prone to biases that can lead to spurious associations. *See* Neugut Dep. 77:12-79:10. But most importantly here, when properly adjusted to avoid confounding by other pesticides, even these studies do not find any association between GBHs and NHL, with non-statistically significant findings and ORs closely surrounding the null value of 1.0.

The case-control studies at issue here can be grouped into three main geographical regions: North America, France, and Sweden. Certain studies overlap with other studies due to pooling of data from the studies. *See* Mucci Report at 37 & Figure 3.

The North American Pooled Project (“NAPP”) is a pooled analysis combining original data from three previously-published case-control studies from the United States and Canada. *See id.* 45. In 2015, the NAPP analysis was presented to the International Society for Environmental Epidemiology. *Id.* The NAPP reported non-statistically significant adjusted ORs for GBHs and NHL of 1.13 (95% CI=0.84-1.51) for a group consisting of self-respondents and proxy respondents and an even lower OR of 0.95 (95% CI=0.69-1.32) for what Dr. Neugut

1 agreed was a more reliable group limited to self-respondents. M. Pahwa et al., *An Evaluation of*
 2 *Glyphosate Use and the Risk of Non-Hodgkin Lymphoma Major Histological Sub-Types in the*
 3 *North American Pooled Project* at Slide 26 (Aug. 31, 2015) (on file with Dr. Blair) (“Pahwa
 4 2015”); Neugut Dep. 263:24-264:17; *see also* Weisenburger Dep. 136:24-137:15 (proxy
 5 respondents “are always a concern” and are more likely to give “less reliable” answers
 6 regarding pesticide exposure than self respondents); Blair Dep. 140:11-23 (same).

7 A case-control study from France addresses whether occupational exposure to various
 8 pesticides (including glyphosate) is associated with NHL and other lymphoid cancers.²² The
 9 study reported an OR for GBHs of exactly 1.0 (95% CI=0.5-2.2), Orsi 2009 at 295 (Table 3) –
 10 *i.e., a null finding.*

11 To the extent relied upon by plaintiffs’ experts as a GBH study, a third case-control
 12 study from Sweden is undermined by a major methodological flaw because it compares a group
 13 of individuals exposed to **GBHs and other pesticides** to a group of individuals not exposed to
 14 **GBHs or any other pesticides**.²³ This methodology is “[i]n contrast with a standard
 15 epidemiological approach” because it improperly measures the association between a group of
 16 pesticides and NHL rather than any association specific to GBHs. *See* Mucci Report at 53;
 17 Neugut Dep. 227:6-17 (admitting this “would be a methodological flaw in the study” and
 18 “would make it impossible to actually adjust for the potential impact of other exposures”). The
 19 results of the study also clearly show the impact of this confounding. The multivariable
 20 analyses that attempted to adjust for other pesticides generated a **non-statistically-significant**
 21 OR (1.5, 95% CI=0.77-2.94) for glyphosate, whereas the analysis that did not adjust for other
 22 pesticides (and instead included them in the comparison) had a higher, statistically significant
 23 OR (2.02, 95% CI=1.10-3.71) for glyphosate. *See* Eriksson 2008 at 1661 (Table VII). Further,
 24 this study reported elevated odds ratios for **all** 20 or so different pesticides examined, a finding
 25 that Dr. Neugut agrees suggests “systemic bias.” Neugut Dep. 283:14-284:24.

26 ²² L. Orsi et al., *Occupational exposure to pesticides and lymphoid neoplasms among men:*
 27 *results of a French case-control study*, 66 *Occup. Environ. Med.* 291 (2009) (“Orsi 2009”).

28 ²³ M. Eriksson et al., *Pesticide exposure as risk factor for non-Hodgkin lymphoma including*
histopathological subgroup analysis, 123 *Int. J. Cancer* 1657, 1658 (2008) (“Eriksson 2008”).

1 Plaintiffs’ experts disregard the properly adjusted odds ratios in all case-control studies
 2 and instead rely conveniently on odds ratios that are **not** adjusted for the confounding effect of
 3 other pesticides and that are subject to recall and selection biases. This is litigation advocacy,
 4 not sound science generated by a reliable methodology as required by *Daubert*.²⁴ Whatever
 5 their contribution to the public policy debate, the limited scientific value of these data to the
 6 general causation question here is summarized by Dr. Blair. He admits that in each case-control
 7 study that reported elevated odds ratios between glyphosate and cancer, “**chance, bias and**
 8 **confounding could not be excluded as explanations for the finding.**” Blair Dep. 119:18-25.²⁵
 9 Plaintiffs’ experts choose to ignore this chance, bias, and confounding for their litigation
 10 opinions – but because they have no “good grounds” to do so, their opinions are the antithesis of
 11 reliable science, as explained below.²⁶

12
 13 ²⁴ See, e.g., *In re Bextra*, 524 F. Supp. 2d at 1176 (excluding expert who reached general
 14 causation conclusion by “cherry-picking observational studies that support his conclusion,”
 15 stating that this “is not ‘good science’”). For example, Dr. Ritz originally relied upon the NAPP
 16 findings in her expert report based upon an abstract that only reported confounded odds ratios.
 17 Ritz Report at 15-16. After becoming aware of the data from the same study that was adjusted
 18 for other pesticide exposures – and showed no evidence of an association – she sought to
 19 distance herself from the study results. See Ritz Dep. 305:10-306:17; see also *id.* at 292:11-
 20 293:1. Regarding Eriksson 2008, Dr. Weisenburger admits that: (a) the study includes a
 21 multivariate analysis that controls for other pesticide exposures and generated an OR that is not
 22 statistically significant; (b) the study reports other ORs that were not adjusted for exposure to
 23 other pesticides; (c) he does not know whether any of the unadjusted ORs would be statistically
 24 significant if they were controlled for other pesticides; (d) like Dr. Neugut, the fact that almost
 25 every unadjusted OR for various substances was above 1.0 suggests some kind of bias in the
 26 study; and (e) the study does not show a statistically significant association between glyphosate
 27 and NHL (or any NHL sub-type) controlled for other pesticides. Weisenburger Dep. 181:4-
 28 184:2, 184:24-185:20. Nevertheless, Dr. Weisenburger incredulously claimed that the study
 showed a statistically significant response. *Id.* 181:20-22; Weisenburger Report at 4-5.

²⁵ Because chance, bias, and confounding cannot be excluded as explanations for any positive
 result in the retrospective case-control data, the epidemiology sub-group and the full IARC
 working group determined that the epidemiology evidence for glyphosate and NHL was
 “limited,” in their hazard assessment parlance. See Blair Dep. 118:11-119:17; see also
 Weisenburger Dep. 61:14-62:2 (admitting that a perceived association in an epidemiology study
 “might be due to a causal association” or due to “confounding or bias or the play of chance”).

²⁶ See *Nelson*, 243 F.3d at 253 (“Before any inferences are drawn about causation, the
 possibility of other reasons for the association **must be examined**, including chance, biases . . . ,
 and confounding causes.” (emphasis added)); *In re Bextra*, 524 F. Supp. 2d at 1173 (“[t]he
 downside to observational studies is that . . . it is more difficult to control for confounding
 factors”). More generally, “[s]howing association is **far removed** from proving causation.”
Allison v. McGhan Med. Corp., 184 F.3d 1300, 1315 n.16 (11th Cir. 1999) (emphasis added);
 see *Nelson*, 243 F.3d at 253 (“an association does not mean that there is a cause and effect
 relationship”); see also *Reference Manual* at 555.

1 **First**, plaintiffs’ experts rely on epidemiology findings that lack statistical significance.
 2 But ruling out chance is a bedrock principle of epidemiology. *See Joiner*, 522 U.S. at 145-47
 3 (affirming *Daubert* exclusion because, *inter alia*, experts relied on epidemiology study that was
 4 not statistically significant); *Burst*, 650 F. App’x at 174-75 (same); *Allen*, 102 F.3d at 197
 5 (same); *see also* Neugut Dep. 45:14-18 (requiring statistically significant increased risk before
 6 he would conclude that an exposure is associated with an outcome).

7 **Second**, plaintiffs’ experts rely on epidemiologic findings that fail to adjust for
 8 pesticides and other farming exposures that confound the analyses. But as Dr. Blair testified,
 9 something is “going on with farmers that appears to be associated with an increased risk of
 10 [NHL] that predated glyphosate being on the scene,” Blair Dep. 90:15-20, “**that we know for a**
 11 **fact can’t be glyphosate.**” *Id.* 90:15-91:3. This is classic confounding. *See id.* 91:23-92:4 (to
 12 implicate glyphosate exposure in farmers, one wants to ensure that one “can control for those
 13 other possible confounders to be sure that [one is] actually studying glyphosate”); *see also*
 14 Weisenburger Dep. 179:24-180:5 (admitting that increase of NHL cases in 1950’s could not
 15 have been caused by glyphosate); *id.* 93:16-23 (admitting other pesticide exposures could be
 16 “major confounder for the issue of whether glyphosate can cause [NHL]”).

17 **Third**, plaintiffs’ experts fail to account for recall bias, which artificially increases the
 18 odds ratios in case-control studies where, as would be expected, people who have cancer recall
 19 more exposures than people who do not have cancer and have not been thinking about their
 20 prior exposures. *See* Blair Dep. 95:2-22.

21 In light of these serious flaws – and Dr. Blair’s (correct) admission that the flaws cannot
 22 be eradicated when one evaluates the glyphosate-NHL retrospective case-control literature –
 23 that literature does not and cannot provide scientifically reliable support for plaintiffs’ experts’
 24 general causation opinions. *See Joiner*, 522 U.S. at 145-46 (epidemiology cannot provide a
 25 scientifically reliable basis for an affirmative causation opinion if it is statistically insignificant
 26 or inadequately controlled for bias and other confounders); *Nelson*, 243 F.3d at 252-53 (expert
 27 was properly excluded because he failed to account for confounding factors); *In re Bextra*, 524
 28 F. Supp. 2d at 1179 (not scientifically reliable for expert to rely on study that failed to account

for critical confounding factors); *Valentine v. Pioneer Chlor Alkali Co., Inc.*, 921 F. Supp. 666, 678 (D. Nev. 1996) (excluding expert’s opinions that failed to account for selection bias, recall bias, and confounding); *see also* Neugut Dep. 39:19-40:1 (conceding that epidemiology evidence alone is not sufficient to show causal relationship between glyphosate and NHL); *id.* 38:4-39:10 (accepting IARC’s classification of epidemiology as “limited” and admitting chance, bias, and confounding could not be ruled out); *id.* 61:16-20 (making dispositive concession under *Daubert* that, “looking just at the epidemiological data, bias and confounding cannot be excluded as an explanation for the findings in those studies”).²⁷

3. Meta-analysis studies also do not provide scientifically reliable support for plaintiffs’ experts’ epidemiology opinions.

Plaintiffs’ experts also rely on two meta-analyses that combine results from some of the glyphosate epidemiological studies.²⁸ However, these meta-analyses do not provide scientifically reliable support for plaintiffs’ experts’ general causation opinions.

Combining results of observational studies (as opposed to results from randomized, controlled, experimental trials) into a meta-analysis amplifies and exaggerates the flaws in the underlying studies, such as bias and confounding. This is the “garbage in = garbage out” problem with meta-analyses: “Combining a group of poorly done studies can produce a precise summary result built on a very weak foundation.” J. Berlin et al., *The Use of Meta-Analysis in Pharmacoepidemiology*, in *Pharmacoepidemiology* at 726 (5th ed. 2012). Thus, it is not reasonably disputed that meta-analysis is “of limited value in combining the results of

²⁷ Drs. Nabhan, Portier, and Jameson agreed that the epidemiology evidence is “limited” as that term was used in the IARC glyphosate review – *i.e.*, that chance, bias, or confounding could not be ruled out with reasonable confidence. Nabhan Dep. 101:16-102:7; Portier Dep. 140:4-141:15; Jameson Report at 9-10, 19. The epidemiology data is “not sufficient by itself to demonstrate causality” between NHL and glyphosate exposure. Portier Dep. 140:4-141:15.

²⁸ *See* L. Schinasi et al., *Non-Hodgkin Lymphoma and Occupational Exposure to Agricultural Pesticide Chemical Groups and Active Ingredients: A Systematic Review and Meta-Analysis*, 11 Int’l J. Env’tl. Res. & Public Health 4449 (2014); *see also* IARC Monograph 112 at 30; E. Chang et al., *Systematic review and meta-analysis of glyphosate exposure and risk of lymphohematopoietic cancers*, 51 J. Env’tl. Sci. & Health 402 (2016). A meta-analysis combines the final results of multiple studies into a combined relative risk and is not dependent upon having access to original study data while a pooled analysis combines the individual data from multiple studies in order to calculate a single risk ratio. Both methods are subject to the various biases inherent to the underlying studies they are based upon.

1 epidemiologic studies based on observation.” *Knight v. Kirby Inland Marine, Inc.*, 363 F. Supp.
 2 2d 859, 866 n.13 (N.D. Miss. 2005); *see In re Bextra*, 524 F. Supp. 2d at 1174 (“[O]ne problem
 3 with meta-analysis, **particularly in meta-analysis of observational studies**, is that the
 4 [underlying] studies often use disparate methodologies.” (emphasis added)); *Reference Manual*
 5 at 607 (“[W]hen meta-analysis is applied to observational studies—either case-control or
 6 cohort—it becomes more controversial.”).²⁹

7 Dr. Neugut summarized the *Daubert* failings of plaintiffs’ experts’ reliance on meta-
 8 analyses when he acknowledged several of their more well-established problems. He admitted
 9 that “meta-analysis is not appropriate” in some cases and that “the results can be misleading.”
 10 Neugut Dep. 94:5-15. He also conceded that a meta-analysis does not fix “an underlying recall
 11 bias,” “an underlying selection bias,” or “a problem with confounding in any of the underlying
 12 studies.” *Id.* 99:11-100:4. He acknowledged the misleading results can be further skewed by
 13 “publication bias” – where “positive findings may be published and null findings may not be
 14 published.” *Id.* 104:11-19; *see also* Blair Dep. 202:7-21 (discussing publication bias; admitting
 15 that “it’s more difficult to get negative findings published” and that “as a result, sometimes
 16 negative findings . . . are not published”). And finally, Dr. Neugut testified that “if you haven’t
 17 included every study, then you . . . have to be concerned that you are biasing the results
 18 upward.” Neugut Dep. 106:2-8.

19 Each of these problems exists here, which means that the meta-analyses upon which
 20 plaintiffs’ experts rely are not scientifically valid support for their opinions. These meta-
 21 analyses combine observational studies that used different methodologies (cohort studies versus
 22 case-control studies) and that took different approaches towards confounding (controlling
 23 versus not controlling for exposures to other pesticides). Moreover, the meta-analyses do not
 24

25 ²⁹ *See also Reference Manual* at 608 (“The appeal of a meta-analysis is that it generates a single
 26 estimate of risk . . . but this strength can also be a weakness, and may lead to a false sense of
 27 security regarding the certainty of the estimate.”); B. Black et al., *Expert Evidence: A*
 28 *Practitioner’s Guide to Law, Science, and the FJC Manual* at 98 (1997) (“[A]ggregation of
 nonrandomized observational studies [for a meta-analysis] is especially fraught with peril. Such
 studies often do not have a common research design, and **controlling for biases is particularly**
problematic.” (emphasis added)).

1 fix flaws in the underlying case-control studies like recall bias, selection bias, or confounding.
 2 *See supra* at 15-19. “At the heart of meta-analyses . . . is that their validity is completely
 3 dependent on the validity in the design and conduct of the original studies,” Mucci Report at 58,
 4 but – as recognized by IARC and most of plaintiffs’ experts – bias and confounding cannot be
 5 excluded as explanations for the results of the original studies at issue here. In turn, they cannot
 6 be excluded as explanations in any meta-analyses.

7 Publication bias and the failure to include every study are especially acute and obvious
 8 problems here. Neither of the meta-analyses upon which plaintiffs’ experts rely included the
 9 findings of the 2013 AHS analysis or the NAPP pooling of the North American case-control
 10 studies. As Dr. Blair acknowledged in his deposition, if these two studies are included, the
 11 meta-relative risk for glyphosate and NHL ***becomes lower and does not show any statistically***
 12 ***significant increased risk***. Blair Dep. 182:16-183:17; 189:4-8. Indeed, when the authors of
 13 one of the original meta-analyses (Chang & Delzell) updated their meta-analysis using the same
 14 methodology and the more current glyphosate/NHL epidemiology data, they calculated a meta-
 15 RR of 1.0 (0.86 – 1.2), a completely null finding of no association. Mucci Report at 59-60; *see*
 16 *also* E. Chang et al., *Meta-Analysis of Glyphosate Use and Risk of Non-Hodgkin Lymphoma*,
 17 *Exponent 1*, 5 (2017); Ritz Dep. 284:9-285:9 (acknowledging that meta-analysis methodology
 18 uses most recent data adjusted for exposure to other pesticides). In short, all of the most reliable
 19 epidemiology data fail to support an association between GBHs and NHL, and therefore
 20 plaintiffs have failed to meet their burden under *Daubert*.

21 **B. Plaintiffs’ Experts Do Not Employ Reliable Scientific Methodologies And Make**
 22 **Unsupported Scientific Leaps In Their Opinions Regarding The Glyphosate Rodent**
Carcinogenicity Data.

23 As discussed above, where epidemiology data exists (as it does here), it is the best
 24 method to assess causation in humans at the relevant inquiry involving real-world exposure
 25 levels. Plaintiffs’ toxicology experts – Drs. Portier and Jameson – nevertheless purport to rely
 26 on “secondary methodologies, including . . . animal studies [that] are insufficient proof of
 27 general causation.” *Chapman v. Proctor & Gamble Distrib., LLC*, 766 F.3d 1296, 1308 (11th
 28

1 Cir. 2014).³⁰ Regardless, glyphosate is not even a rodent carcinogen, as regulatory agencies
 2 around the world that have reviewed at least 14 long-term studies have concluded. Plaintiffs’
 3 experts disagree, yet in doing so, they rely on unreliable methodologies, blatant untested
 4 statistical manipulations, and a series of *ipse dixit* conclusions that contradict each other and the
 5 actual data.³¹ As such, these opinions must be excluded.³²

6 As a general matter, plaintiffs’ experts admit “the purpose of doing an animal bioassay
 7 study is to determine if the chemical can cause cancer *in the experimental animals*,” not
 8 humans. Jameson Expert Dep. 28:10-12; 28:12-15 (“[an animal bioassay is] not -- not looking
 9 to investigate does it form a specific kind of tumor that is the same as found in humans.”);
 10 Portier Dep. 163:7-23 (rodent models “are not developed for the purpose of identifying tumors

11 ³⁰ See also *Conde v. Velsicol Chem. Corp.*, 24 F.3d 809, 813-14 (6th Cir. 1994) (rejecting
 12 reliance on animal studies where epidemiology does not show risk of alleged injury); *In re*
 13 *Silicone Gel Breast Implants Prod. Liab. Litig.*, 318 F. Supp. 2d 879, 891 (C.D. Cal. 2004)
 14 (“Animal studies are not generally admissible where contrary epidemiological evidence in
 humans exists.” (citing *Richardson v. Richardson–Merrell, Inc.*, 857 F.2d 823, 830 (D.C.
 Cir.1988)).

15 ³¹ Although they share some opinions regarding glyphosate (including about the lack of
 16 relevance of animal data to human NHL), see *infra*, the methodologies that Drs. Portier and
 17 Jameson apply to interpretation of the animal data are so arbitrary and pliable that they reach
 18 divergent conclusions with regard to multiple tumor types, one finding statistically significant
 trends and evidence of carcinogenicity where the other finds none. See, e.g., Jameson Expert
 19 Dep. 119:17-19; 123:4-124:6 (statistically significant trend in hemangiosarcomas in male mice
 according to Jameson, non-statistically significant trend according to Portier); *id.* 142:4-11
 20 (statistically significant increase in lung adenocarcinomas due to glyphosate according to
 Jameson; lung adenocarcinoma due to chance according to Portier). Dr. Jameson could not
 21 explain these discrepancies. See *id.* 124:1-9 (“So if he has a number in his expert report that is
 different than this, it’s probably due to the fact that he did additional analysis or subsequent
 22 analysis of the data because being a statistician, they always evaluate and reevaluate the data, so
 that ... But I don’t know.”).

23 ³² Six of plaintiffs’ experts (Drs. Nabhan, Weisenburger, Neugut, Ritz, Blair, and Ross) are not
 24 qualified to render opinions on rodent bioassay data. As plaintiffs’ counsel admit, those experts
 have no specialty experience in toxicology, the branch of science under which analysis of rodent
 25 bioassay data falls. See 5/16/17 Letter. These admissions are borne out by the experts’ CVs,
 none of which recites any experience in the conduct or interpretation of rodent carcinogenicity
 26 bioassay data. Further, mere membership in the IARC working group does not bestow the
 requisite expertise. See Blair Dep. 49:11-13 (“Different subgroups [of the IARC working
 group] evaluate different components. I’m really familiar with the epidemiology, not so much
 27 the other.”); Ross Dep. 58:22-59:11 (explaining he did not review the genotoxicity data). Dr.
 Ritz revealed for the first time at her deposition that she intends to offer opinions on the rodent
 28 carcinogenicity bioassays. Ritz Dep. 79:2-16. In addition to her complete lack of qualifications
 to do so, those opinions must be excluded under Federal Rule of Civil Procedure 26(a)(2)(B)
 because neither the opinions nor her methodologies are disclosed in her expert report. See Ritz
 Report. Undisclosed opinions must be excluded. See *Nationwide Transp. Fin. v. Cass Info.*
Sys., Inc., 523 F.3d 1051, 1062 (9th Cir. 2008).

1 that arise in humans from exposure to chemicals.”).³³ They further admit “it has always been a
 2 challenge to extrapolate from effects observed in experimental animal bioassays to potential
 3 effects in humans in order to protect humans from potentially harmful chemical exposures.”
 4 Portier Dep. 158:14-159:16; Jameson Expert Dep. 9:3-6 (“[T]he fact that something causes a
 5 kidney tumor in a mouse, I don’t know what that says about causing non-Hodgkin’s lymphoma
 6 in humans.”). Yet they give no reason why extrapolating generally from animals to humans is
 7 appropriate in this case notwithstanding: (1) the overwhelmingly negative epidemiology data,
 8 (2) the fact that animal dosing is orders of magnitude higher than the plausible human exposure,
 9 *see infra* at 29-30, (3) they rely on animal tumors *other than* lymphoma, further improperly
 10 skewing their extrapolation of animal data to human risk of NHL, and (4) even with respect to
 11 lymphomas in rodents, they cannot explain why extrapolating is appropriate when they agree
 12 that the immune systems of rodents and humans have important differences. Portier Dep.
 13 167:8-15; Jameson Expert Dep. 169:16-19.

14 They cannot cite one article in the published literature that suggests that CD-1 or Swiss
 15 Albino mice (the mouse models at issue in this case) are appropriate for assessing the potential
 16 for a substance to cause NHL in humans. Portier Dep. 171:21-172:3; Jameson Expert Dep.
 17 27:18-24. Dr. Jameson concedes that both strains have a “high spontaneous incidence” of
 18 malignant lymphoma and that actually he is aware of scientific literature *objecting* to the use of
 19 mice as a model for evaluating whether a chemical can cause lymphoma precisely “*because of*
 20 *the high background level . . .*” Jameson Expert Dep. 29:13-30:5, 133:17-134:8. They
 21 certainly cannot cite any literature to support the opinion that any of the other tumors at issue in
 22 the rodent studies are associated with NHL in people. *See, e.g.*, Portier Dep. 174:24-175:7.

23
 24
 25 ³³ Rodent (mice or rat) carcinogenicity studies start from the hypothesis that the test compound
 26 does not cause cancer (a “null hypothesis”). Prior to the study, investigators select which
 27 statistical tests they will apply to the data and at what probability level (or “p-value”) between 0
 28 and 1 that the data will be considered significant. If the p-value is below the selected level of
 significance, then further evaluation is needed to assess whether the tumors seen are related to
 the test compound. If the p-value exceeds the selected level, the null hypothesis holds (*i.e.*, the
 study is considered to show no compound-related effect).

1 Plaintiffs' experts' failure to explain how data regarding tumors allegedly induced in
 2 rodent models upon which they rely are relevant to NHL in humans is a fatal *Daubert* flaw;
 3 without such an explanation, the rodent data cannot "fit" the allegations in this case and must be
 4 excluded on that basis alone. *See, e.g., Joiner*, 522 U.S. at 144 (approving exclusion of expert
 5 testimony based on "seemingly far-removed" animal studies where expert failed to explain why
 6 the extrapolation was scientifically proper).³⁴ Other factors, such as shifting methodologies and
 7 made-for-litigation opinions, amply support that outcome as well.

8 **1. Dr. Portier's result-oriented opinions ignore established scientific principles,**
 9 **violate his own purported methodology, and have not been subjected to**
 10 **scientific scrutiny.**

11 Beyond the broad *Daubert* flaws in his general mouse-to-man methodology, Dr. Portier
 12 plays fast and loose with basic statistical principles in a desperate effort to find any calculation
 13 to support his predetermined conclusion that glyphosate is carcinogenic in rodents.³⁵ He has no
 14 defined methodology and will discard any data – no matter how credible or whether it appears
 15 in his own non-litigation opinions – in an effort to create "proof" of carcinogenicity. He ignores
 16 the authors' pre-study protocols for data analysis and their conclusions about that data in favor

17 ³⁴ *See also O'Hanlon v. Matrixx Initiatives*, No. CV 04-10391AHMJTLX, 2007 WL 2446496,
 18 at *2 (C.D. Cal. Jan. 3, 2007) ("[W]hen extrapolating from studies concerning one substance,
 19 one species, one dose level or one manner of exposure, it is incumbent upon the expert to
 20 explain and demonstrate why the extrapolation is scientifically proper. ... [P]ositive results in
 21 other animal studies, standing alone, cannot establish positive results for the human claiming
 22 the same impact from the drug or chemical element."); *Newkirk v. ConAgra Foods, Inc.*, 727 F.
 23 Supp. 2d 1006, 1026 (E.D. Wash. 2010), *aff'd*, 438 F. App'x 607 (9th Cir. 2011) (excluding
 24 expert who "offers no explanation for how and why the results of [rat] studies can be
 25 extrapolated to humans); *Redfoot v. B.F. Ascher & Co.*, No. C 05-2045 PJH, 2007 WL
 26 1593239, at *11, n.18 (N.D. Cal. June 1, 2007) ("In general, [e]xtrapolations of animal studies
 27 to human beings are generally not considered reliable in the absence of a scientific explanation
 28 of why such extrapolation is warranted." (internal quotation omitted)); *Siharath*, 131 F. Supp.
 2d at 1366-67 ("[E]xtrapolating from animals to humans is difficult because differences in
 absorption, metabolism, and other factors may result in interspecies variation in responses.")
 (internal citation omitted); *In re Silicone Gel Breast Implants*, 318 F. Supp. 2d at 891 (same).

³⁵ Plaintiffs signed Dr. Portier up to assist them in the litigation no later than March 29, 2015,
 just nineteen days after the IARC Working Group concluded and nine days after their findings
 became public. (At the time he signed the retainer, he was already working with the same
 counsel in another litigation connected with a different IARC review.) *See* Portier Dep. 75:14-
 77:2. Therefore, all of Dr. Portier's post-IARC activities (at least) are those of a "professional
 plaintiff's witness" and "[i]t is not unreasonable to presume that [his opinion on glyphosate] was
 influenced by a litigation-driven financial incentive." *Lust v. Merrell Dow Pharm.*, 89 F.3d 594,
 597 (9th Cir. 1996) (excluding similar opinion as litigation-driven).

1 of his *post hoc* made-for-litigation reassessment in which he uses whatever method of
 2 calculation best supports the opinion he already reached.³⁶

3 All of these failings in methodology are exemplified in his analysis of renal tumors in
 4 the Monsanto 1983 mouse bioassay. Before initiating the study, the original investigators
 5 specified which two statistical tests would be used to interpret the data and set a .01 level of
 6 significance. *See supra* at n.33 (describing bioassay methodology). When applied upon
 7 conclusion of the study, they found no statistically significant increase in incidence in renal
 8 tumors in mice exposed to glyphosate. *See* EPA RED at 14. In 1985, a group of independent
 9 pathologists reviewed the study and reached the same conclusion using a .05 level of
 10 significance and updated tumor data. *Id.* Thirty years later, Dr. Portier embarked on a multi-
 11 year quest to find a statistical test that would yield a different result. First, in March 2015 for
 12 the IARC Monograph, Dr. Portier applied an “approximate trend” test that achieved a
 13 statistically significant result (p-value = .034 (below the .05 level of significance that he used
 14 for all of his calculations). Portier Dep. 47:1-17. Some months later, Dr. Portier applied a
 15 different test that also achieved a modestly more statistically significant result (p-value = .03).³⁷
 16 After another well-known biostatistician publicly criticized Dr. Portier’s calculation by noting
 17 that the test Dr. Portier selected is known to have a large bias toward finding a statistically
 18 significant effect,³⁸ Dr. Portier again changed his statistical method, this time using the more
 19 appropriate “exact trend” test and reaching a result (p-value = .063) that was not statistically
 20 significant.³⁹ Presumably unhappy with that turn of events, Dr. Portier unveiled two more novel
 21 calculations (the fourth and fifth in his changing methodologies) in his 2017 expert report here,

22 _____
 23 ³⁶ Federal courts distrust this type of results-seeking “analysis.” *See, e.g., Baker v. Sec’y of HHS*,
 24 No. 99-653V, 2003 WL 22416622 at *30 (Fed. Cl. Sept. 26, 2003) (“[t]he validity of this type of
 ‘post-hoc’ statistical testing” is “highly questionable”); *Karlo v. Pittsburgh Glass Works, LLC*,
 849 F.3d 61, 82 (3d Cir. 2017) (“researcher who searches for statistical significance in multiple
 attempts raises the probability of discovering it purely by chance, committing Type I error”).

25 ³⁷ *See* Expert Report of Christopher Portier, Attachments 4 and 5 at Table 4 (Hollingsworth
 26 Decl., Ex. 19) (“Portier Report”).

27 ³⁸ *See* Portier Report, Attachment 6 at 1 (commenter noting that Dr. Portier’s test statistics “are
 extremely skewed” due to his use of the approximate trend test).

28 ³⁹ *See* Portier Report, Attachment 7 at 2 (admitting that switching from approximate to exact test
 results in a p-value greater than .05).

1 with one once again finding statistical significance (p-values = .065, .011).⁴⁰ This is blatant “p-
 2 hacking,” an oft-rejected and unscientific process also known as data dredging, in which
 3 numerical data is manipulated to generate a statistically significant result and is then used *post*
 4 *hoc* to support a pre-existing scientific conclusion.⁴¹

5 Dr. Portier’s “willingness to change the pre-specified statistical endpoint – with the effect
 6 of turning a ‘not significant’ study result into a [significant one] – again, demonstrates a lack of
 7 objectivity and reliability.” *In re Denture Cream Prod. Liab. Litig.*, No. 09-2051-MD, 2015 WL
 8 392021, at *10 (S.D. Fla. Jan. 28, 2015). Stated differently, “[c]oming to a firm conclusion first
 9 and then doing research to support it is the antithesis of [the scientific] method.” *Claar v.*
 10 *Burlington N. R. Co.*, 29 F.3d 499, 502-03 (9th Cir. 1994).⁴²

11 In another “opinion first, data later” made-for-litigation supposition, Dr. Portier
 12 developed a novel technique that, he says, permits him to pool, or combine, data from studies
 13 done at different laboratories in different animals by different investigators years apart despite
 14 his own admission that “there is considerable genetic variability across animal strains both over
 15 time and space.” Portier Amended Report at 51.⁴³ Perhaps even worse, Dr. Portier applied his
 16 personally developed, untested, non-peer reviewed pooling methodology inconsistently to
 17 ensure that he generated a result that supports his predetermined opinions regarding the

18 ⁴⁰ The second p-value is based on a novel statistical test called a “p-hist” test, which itself is
 19 unreliable. *See infra*.

20 ⁴¹ *See, e.g.,* R. Nuzzo, *Statistical Errors*, 506 *Nature* 150, 150-52 (2014).

21 ⁴² *See In re REMEC Inc. Sec. Litig.*, 702 F. Supp. 2d 1202, 1273 (S.D. Cal. 2010) (excluding
 22 expert who predetermined the “results of his analysis” and applied methodology suited to reach
 23 his predetermined result); *Newkirk*, 727 F. Supp. 2d at 1021 (same; citing *Claar*, 29 F.3d at 502-
 24 03); *In re Denture Cream*, 2015 WL 392021, at *10 (“[a] scientist who has a formed opinion as
 25 to the answer he is going to find before he even begins his research may be less objective than
 26 he needs to be in order to produce reliable scientific results.” (quoting *Perry v. United States*,
 27 755 F.2d 888, 892 (11th Cir. 1985)); *id.* (excluding under *Daubert* plaintiffs’ MDL experts’
 28 testimony based on methodologies that are “contrived to reach a particular result”) (citing *Rink*
v. Cheminova, Inc., 400 F.3d 1286, 1293 n.7 (11th Cir. 2005)).

⁴³ Dr. Portier developed this novel pooling analysis because “[m]ethods for the combined
 analysis of multiple animal cancer bioassays are not available in the scientific literature.” Portier
 Amended Report at 21. At deposition, he claimed that pooling disparate data is a peer-reviewed
 methodology, but could not provide a citation. Plaintiffs failed to respond to a post-deposition
 request that Dr. Portier identify this source. *See* Ltr. from H. Pigman to R. Greenwald (Sept. 14,
 2017) (Hollingsworth Decl., Ex. 20). Any attempt to provide one now must be rejected. *Supra*
 at n.32.

1 outcome. For instance, he chose to include three studies in rats (Brammer, Suresh, and Wood)
 2 in his pooled analysis for skin keratocanthomas, but then chose to exclude one of those studies
 3 (Suresh) for his pooling of hepatocellular adenomas and mammary gland tumors. Portier Dep.
 4 210:6-212:25. He applied a similarly selective pooling technique in assessing tumors in
 5 mice. *Id.* 236:17-238:3 (when initial pooled calculations were unhelpful, he recalculated
 6 statistics *after* excluding animals in one high-dose group from analysis). When asked how he
 7 justified such random, self-serving inclusions and exclusions of data, Dr. Portier testified that **he**
 8 ***included all data when it helped increase his statistically significant result and removed data***
 9 ***that defeated statistical significance.*** See *id.* 236:24-240:1.⁴⁴

10 Electing to selectively pool the data ensured that Dr. Portier conjured up statistically
 11 significant results, otherwise absent, for a variety of tumors. Such tactics are unscientific p-
 12 hacking and must be excluded. See *Lust*, 89 F.3d at 597-98 (excluding expert whose testimony
 13 is based on methodology that is unscientific and not accepted). When confronted with the
 14 doubtful scientific validity of his methods, Dr. Portier claimed that his pooled data were not a
 15 part of his causation assessment. Portier Dep. 238:15-22 (“This is a – this is the pooling
 16 evaluation here. There is reason – that’s just simply an observation on my part. That is all it is.
 17 This is not used as part of my overall evaluation.”). However, his expert report and other
 18 portions of deposition testimony clearly state otherwise. See, e.g., Portier Amended Report at
 19 33 (“The analysis of the pooled studies yields $p_{\text{trend}}=0.013$ **supporting the conclusion that**
 20 ***glyphosate causes hepatocellular adenomas in Wistar rats*** with similar background
 21 responses.”) (emphasis added); Portier Dep. 188:19-22 (“Q. You reached your rat causation
 22 opinions through the application of a pooling methodology, correct? A. Yes, I did.”).

23 Beyond selective pooling and *post hoc* trend analyses, for certain “rare” rodent tumors,
 24 Dr. Portier purported to compare historical control values (as opposed to study-generated
 25 control values) to tumor incidences to generate self-described “p-hist” values. Dr. Portier does
 26

27 ⁴⁴ Dr. Portier engaged in similar p-hacking by excluding the Lankas rat study from his pooled
 28 analysis of adrenal cortical tumors, but then including the Lankas study in his evaluation of
 other types of tumors, such as testicular tumors. Portier Dep. 214:3-219:23.

1 not disclose any formal methodology for this work. There is a reason for that: his “p-hist”
 2 analysis is novel, untested, unpublished, and created for litigation. Therefore, opinions which
 3 rest on it are inadmissible.⁴⁵ See *Orrell v. AstraZeneca Pharm. LP (In re Nexium*
 4 *Esomeprazole)*, 662 F. App’x 528, 530 (9th Cir. 2016) (affirming district court’s decision to
 5 exclude expert opinion on general causation formed for purposes of litigation and that was not
 6 subject to peer review) (citing *Daubert II*, 43 F.3d at 1318).

7 Additionally, as even Dr. Portier recognizes, a potential for a multiple comparison error
 8 arises in any of the rodent bioassays because, when making the hundreds of statistical
 9 comparisons required in these studies, some statistically significant results will occur by chance
 10 alone and are not representative of compound-related effects. Portier Amended Report at 50.
 11 Despite peer-reviewed methodology available to do so, Dr. Portier makes no effort to control
 12 for these statistical errors. Instead, he decided to draw conclusions based on his self-concocted
 13 statistically significant p-values, a methodology that has been rejected by the American
 14 Statistical Association (“ASA”), of which Dr. Portier is an elected fellow. See R. Wasserstein et
 15 al., *Statement on p-values: Context, Process, and Purpose*, 70 Amer. Statistician 129, 132
 16 (2016).

17 As one would expect, Dr. Portier’s Hail Mary attempt at aggregating his inconsistent
 18 and disparate methodologies into a single analysis (*e.g.*, Table 15 in his revised expert report) is
 19 also irredeemably flawed because the underlying statistics result from inconsistently applied
 20 and non-validated methodologies. For example, Dr. Portier claimed to have derived his number
 21 of “expected” statistically significant tumor incidences from an estimate prepared by another
 22

23 ⁴⁵ Even if the “p-hist” methodology had any indicia of reliability other than Dr. Portier’s
 24 inadmissible *ipse dixit*, he applies his creation in an inconsistent way that renders opinions based
 25 on it inadmissible. *Lust*, 89 F.3d at 598 (courts “should be wary that the [expert’s] method has
 26 not been faithfully applied”). For instance, Dr. Portier conducted a p-hist evaluation using a
 27 historical control database for systemic hemangiosarcomas, but admitted at deposition that he
 28 was not sure which hemangiosarcomas to combine for his evaluation and that a pathologist
 would be more qualified to answer that question. Portier Dep. 246:11-248:20. So he chose the
 lowest historical control rate possible for hemangiosarcomas in order to generate the most
 significant possible test statistic. *Compare, e.g.*, Portier Dep. 247:14-17 (admitting there were 29
 hemangiosarcomas in historical control data of similar length) with 242:24-243:8 (agreeing that
 he applied a historical control rate based on 0/1424 hemangiosarcomas).

1 statistician, Portier Dep. 299:17-301:5, but admitted that he had not verified that information.
 2 *Id.* 316:23-317:9. He also admitted that in his “observed” statistically significant tumor sites, he
 3 included results derived from his novel p-hist test that may or may not have been statistically
 4 significant. *Id.* 308:7-311:12; 321:9-323:24. Such methodological hijinks have no basis in the
 5 scientific literature, no calculable error rate, and no acceptance in the scientific community, and
 6 therefore opinions based on them should be excluded. *See, e.g., Lust*, 89 F.3d at 597-98
 7 (excluding testimony by expert that selectively chose his support from the scientific landscape).

8
 9 **2. Dr. Jameson conducts the wrong scientific assessment and concedes that his
 opinions add little to the analysis of the risks of NHL in humans.**

10 Like Dr. Portier, Dr. Jameson opines that a variety of tumors seen in long-term rodent
 11 bioassays were related to the animals’ exposure to glyphosate, notwithstanding contrary
 12 opinions offered by regulatory agencies and others. In reaching his opinions, Dr. Jameson used
 13 a hazard assessment methodology. *See supra* at 3-4.⁴⁶ A hazard assessment is at most a
 14 “screening assessment,” and more must be done to assess causation under *Daubert*.⁴⁷ For
 15 example, Dr. Jameson purposely did not take into account whether any carcinogenic effects he
 16 allegedly observed would be seen at human relevant doses. Dr. Jameson admits that hazard
 17 assessments examine animal carcinogenicity “under the most extreme conditions,” and if the
 18 compound is an animal carcinogen, “you do additional studies. You do the risk analysis” –

19
 20 ⁴⁶ The precise hazard assessment methodology he employed is unclear, even to Dr. Jameson. In
 21 his report, Dr. Jameson asserted that his hazard assessment methodology “is the same as defined
 22 and characterized by IARC.” Jameson Report at 9. At his deposition, Dr. Jameson claimed that
 23 his hazard assessment criteria are “very similar” to IARC’s, but that he developed his criteria
 24 “specifically for this – for my expert report.” Jameson Expert Dep. 266:3-16. Whichever
 25 version he used, Jameson hides behind it to explain his disagreements with regulators like EPA
 26 and EFSA (whom he acknowledges reviewed more data than he did). *See, e.g.,* Jameson Fact
 27 Dep. 166:21-167:8 (agencies reviewed more data); Jameson Expert Dep. 207:3-10 (“Again, the
 28 EPA was doing their risk assessment, and evidently ... these particular tumors did not meet their
 criteria for inclusion in their risk assessment ... [F]or the purpose of the hazard identification I
 did, these liver tumors -- I consider these liver tumors to be associated with exposure to
 glyphosate and, therefore, I included them in my report.”); *see also id.* 111:4-19; 144:5-145:1;
 152:4-20, 180:6-25, 206:5-209:1, 248:21-249:12 (same regarding other tumor types).

⁴⁷ *See* Monsanto Co.’s Br. Regarding the Relevance of IARC and EPA to Gen. Causation, ECF
 No. 134.; *see also* Ltr. from Bernhard Url, Exec. Director, EFSA, to Prof. Christopher J. Portier,
 Senior Consulting Scientist, Env’tl. Def. Fund (Jan. 13, 2016), https://www.efsa.europa.eu/sites/default/files/EFSA_response_Prof_Portier.pdf.

1 which he did not do – “to see what happens at the human relevant doses.” Jameson Expert Dep.
 2 248:6-249:20; *compare, e.g.* EPA OPP at 85 (high dose males in CD-1 mouse study received
 3 4945 mg/kg/day glyphosate) *with supra* at 5 (noting highest estimated systemic dose in farmers
 4 of 0.004 mg/kg and EPA reference dose of 2 mg/kg/day). Dr. Jameson also cannot point to any
 5 published study or article suggesting that the various tumor findings he identifies in his hazard
 6 assessment are associated with or predictive of NHL in humans.⁴⁸ Therefore, his opinions do
 7 not fit the general causation question here; they do not demonstrate “a valid scientific
 8 connection to the pertinent inquiry” in the lawsuit. *Daubert*, 509 U.S. at 591-92.

9 Dr. Jameson’s methodology for evaluating the glyphosate data also differs from
 10 methodology he published *prior* to being retained as an expert witness in this litigation. While
 11 at the National Toxicology Program, Dr. Jameson co-authored a publication that describes a set
 12 of factors that should be considered when interpreting tumor findings in long-term
 13 carcinogenicity bioassays.⁴⁹ The first of his pre-litigation factors requires assessing whether the
 14 results of one study are replicated by “results within the same chemical in other experiments (or
 15 elsewhere in the same experiment, if animals of a different sex, strain or species were also
 16 studied).” *Id.* For glyphosate, results from at least 12 different experiments by seven different
 17 sponsors are available for comparison, which Dr. Jameson concedes is “more than you usually
 18 see for a particular compound.” Jameson Expert Dep. 32:23-24. Yet for purposes of his expert
 19 opinion in this case, Dr. Jameson claims – as he must, in order to advance the opinion he does –

20
 21 ⁴⁸ See, e.g., Jameson Expert Dep. 10:16-25 (not aware of any publications or any research that
 22 has been done regarding whether renal tumors in mice are predictive of NHL in humans),
 23 27:12-17 (not aware of any research or published papers investigating the association between
 24 mouse lymphomas and NHL in humans), 59:24-60:3 (not aware of any studies or published
 25 papers investigating the association between lung adenocarcinomas and NHL in humans),
 203:11-204:2 (not aware of any data or articles regarding the association between hepatocellular
 26 adenomas in rats and NHL in humans); see generally H. Morse et al., *B Lymphoid Neoplasms of*
 27 *Mice: Characteristics of Naturally Occurring and Engineered Diseases and Relationships to*
 28 *Human Disorders*, 81 *Advances in Immunol.* 97, 99 (2003) (“Fundamental differences between
 mice and men may preclude the exact modeling of any human disease.”).

⁴⁹ See J. Huff & C. Jameson et al., *Carcinogenesis Studies: Results of 398 Experiments on 104*
Chemicals from the U.S. National Toxicology Program, Nat’l Inst. Env’tl. Health Sci., Nat’l
 Toxicology Program at 7 (1988) (“Huff & Jameson”) (“[s]cientific judgment must entail full
 consideration of all the available relevant information together with the statistical findings in an
 attempt to assess the truth”).

1 that it does not matter whether the tumor findings he identifies are replicated across species,
 2 strain, or sex, or even in other experiments with the same strain.⁵⁰ Had he attempted the
 3 comparison for each of the four tumor types that he claims are replicated, *see* Jameson Report at
 4 29, he would have observed no replication across species, strain, or sex across or within any
 5 study.⁵¹

6 Where an expert abandons his peer-reviewed pre-litigation methodology in favor of
 7 developing new ones just for the litigation, his opinions must be excluded. *In re Rezulin Prod.*
 8 *Liab. Litig.*, 309 F. Supp. 2d 531, 562 (S.D.N.Y. 2004) (where expert “eschew[ed]” his prior
 9 published, peer-reviewed opinion, his opinion was “not based on scientific method but on the
 10 expediencies of this particular litigation” and was therefore inadmissible).

11 **C. Plaintiffs’ Experts’ Opinions Based On The Mechanistic Data Do Not Withstand**
 12 ***Daubert* Scrutiny Because The Data Fails The “Fit” Requirement And Is Not**
Scientifically Reliable For Such Purposes.

13 EPA, EFSA, and every other major international regulatory agency, as well as the JMPR
 14 of the WHO, have found that glyphosate and GBHs are **not** genotoxic based on both the review
 15 of the published literature and hundreds of regulatory studies containing primary data.⁵² These
 16 agencies did not evaluate oxidative stress because “explicit relationships” between oxidative
 17 stress and adverse outcomes in the human body “have yet to be defined.” *See* EPA, Defining
 18 Pesticide Biomarkers: Biomarkers of Effect Categories, <https://www.epa.gov/pesticide-science->
 19

20 ⁵⁰ *See e.g.*, Jameson Expert Dep. 34:12-35:2 (“not necessary” to see results replicated across
 21 species), 48:17-21, 50:25-51:3 (disregarding that kidney tumors and hemangiosarcomas in male
 22 mice not replicated in females), 53:25-54:12; 62:7-18 (pancreatic islet cell tumors and interstitial
 cell tumors in male rats not replicated in any other rat study, in any mouse study, or in female
 rats in the same study).

23 ⁵¹ Dr. Jameson’s pre-litigation publication also emphasized the limitations of statistics for
 24 interpreting bioassay results, noting that “[a]lthough *p* values may be helpful in deciding whether
 25 or not a substance is carcinogenic, they must not be used inflexibly or given undue weight.”
 26 Huff & Jameson at 7. Nevertheless, in his evaluation of the tumor data here, Dr. Jameson does
 27 just that, giving statistical significance total weight in order to infer causation of otherwise
 random and disparate findings. *See* Jameson Expert Dep. 23:20-23 (“[T]here was a significant
 28 increase in [hemangiosarcomas in male mice], so ... it can be said that glyphosate caused the
 hemangiosarcomas in that particular study.”), 59:19-22 (“since [glyphosate] caused a significant
 increase of lung adenocarcinomas, in this particular study, it’s an animal carcinogen”).

⁵² *See* EPA OPP at 131 (“The genotoxicity studies demonstrate that glyphosate is not directly
 mutagenic or genotoxic *in vivo*.”); EFSA 2015 at 10 (“Glyphosate did not present genotoxic
 potential ...”); JMPR at 132 (“glyphosate ... is devoid of a relevant genotoxic potential.”).

1 [and-assessing-pesticide-risks/defining-pesticide-biomarkers](#). Nevertheless, plaintiffs' experts,
 2 most of whom are not qualified to opine on this topic,⁵³ claim that genotoxicity and the
 3 induction of oxidative stress are each mechanisms by which exposure to glyphosate and GBHs
 4 can increase the risk of NHL in humans.⁵⁴ In support of their assertion, they either rely on
 5 IARC or repeat the conclusions of select articles rather than reviewing all of the relevant data as
 6 required by *Daubert*. Experts cannot simply parrot the conclusions of others as the basis for
 7 their opinions.⁵⁵ Further, plaintiffs' experts do not offer a methodology that allows them to leap

8 ⁵³ Seven of plaintiffs' experts who offer opinions in this area have no qualifications to do so.
 9 Drs. Nabhan, Jameson, and Neugut admitted as much. See Nabhan Dep. 24:14-25:21 ("Well,
 10 I'm not a toxicologist ... a toxicologist is able to look at the -- at the evidence when the product
 11 or compound is going through the process of being approved through toxicology assays,
 12 through animal studies, et cetera. I don't do that. I just look at the literature and review the
 13 literature."); Neugut Dep. 305:5-10 (has not conducted an expert analysis of the data and will
 14 defer to other experts); Jameson Fact Dep. 90:5-7, 19-20 ("I am not a genetic toxicologist"); see
 15 also *id.* 89:5-6, 99:17-19. Two others wrongly seek to appropriate qualifications by imputation
 16 of the knowledge of colleagues. Dr. Ritz claims she is in an "interdisciplinary department" and
 17 "teaches toxicologists." Ritz Dep. 54:22-55:10. She does not, however, teach them
 18 genotoxicology; instead, all of the courses she teaches involve epidemiology. See Ritz Report
 19 at 14 (CV listing current teaching positions). Similarly, Dr. Weisenburger claims the ability to
 20 critically assess mechanistic data by virtue of the fact that his interdisciplinary specialty is
 21 human pathology and he has trained himself to do so. Weisenburger Dep. 18:22-19:3. He is
 22 mistaken. See *Dura Auto Sys. of Ind., Inc. v. CTS Corp.*, 285 F.3d 609, 614 (7th Cir. 2002) ("A
 23 scientist, however well credentialed he may be, is not permitted to be the mouthpiece of a
 24 scientist in a different specialty. That would not be responsible science."); *Washington v.*
 25 *Kellwood Co.*, 105 F. Supp. 3d 293, 311 (S.D.N.Y. 2015) (deeming plaintiffs' expert qualified
 26 in one area "does not provide him with carte blanche (*sic*) to opine on every issue in the case.");
 27 *Castiac Lake Water Agency v. Whittaker Corp.*, No. CV 00-12613 AHM, 2002 WL 34700741,
 28 at *5 (C.D. Cal. Oct. 25, 2002) (same). Drs. Blair and Ross are not qualified to opine on the
 mechanistic data or IARC's conclusions regarding it given their failure to review or analyze it.
 See *supra* at n. 32.

⁵⁴ Cell change due to both genotoxicity (damage to the genetic material of a cell) and oxidative
 stress (in which oxygen-depleted cells release free radicals into the body) occurs naturally on a
 daily basis. See Weisenburger Dep. 239:11-15; Portier Dep. 345:19-23. All cells are equipped
 with various protective and repair mechanisms designed to stabilize the cell in the event
 genotoxicity or oxidative stress occurs. Portier Dep. 344:25-345:6; J. Klaunig et al., *Oxidative
 Stress and Oxidative Damage in Chemical Carcinogenesis*, 254 *Toxicology and Applied
 Pharmacology* 86, 93 (2011). The goal of mechanistic experiments conducted *in vivo* (in cells
 extracted from exposed living animals or humans) and *in vitro* (cells are exposed in a petri dish)
 is to identify whether cell change is of a type, amount, or severity that increases the risk that
 cellular mutations will occur, which in turn may increase the risk of cancer by overriding the
 body's capacity for cellular repair.

⁵⁵ See, e.g., Jameson Report at 30-31 (summarizing genotoxic and oxidative stress studies "[a]s
 noted in Monograph 112"); Neugut Report at 18-19 (same); Weisenburger Report at 8 (same);
 Portier Amended Report at 55-62 (same); Nabhan Dep. 149:19-150:16 (IARC is "authoritative"
 in genotoxicity studies "so I do rely heavily on what the IARC says"); Ritz Report at 24-25
 (listing positive findings reported in five mechanistic studies included in IARC as
 "confirm[ation]" that oxidative stress and genotoxicity are the mechanisms by which glyphosate

1 in some scientifically valid way from the disparate mechanistic data to NHL in humans.

2 As an initial matter, this Court need not consider mechanistic data here because it cannot
 3 reliably help answer the general causation question. “That [a compound] may have [] effects on
 4 living cells or genes is the beginning, not the end of the scientific inquiry and proves nothing
 5” *Allen*, 102 F.3d at 198; *see also Richardson*, 857 F.2d at 830 (“*in vivo* and *in vitro* studies
 6 may provide a clue signaling the need for further research”); *Merrell Dow Pharm., Inc. v.*
 7 *Havner*, 953 S.W.2d 706, 730 (Tex. 1997) (rejecting expert’s reliance on *in vitro* studies to
 8 support his general causation opinion; fact that a substance may adversely affect cells *in vitro*
 9 proves nothing about causation).⁵⁶

10 Genotoxicity and carcinogenicity are not synonymous. Some genotoxic compounds are
 11 not carcinogens, and some carcinogens are not genotoxic. The same lack of association is true
 12 of oxidative stress. Thus, any finding that a chemical has mechanistic capabilities is not even
 13 reliably demonstrative of the chemical’s carcinogenic potential generally, much less the
 14 chemical’s potential to cause any specific malignancy, such as NHL.⁵⁷ *Allen*, 102 F.3d at 198.
 15 To the limited extent it has been considered as a part of the *Daubert* inquiry, courts have found
 16 mechanistic data is only relevant in situations where scientifically sound human data is not
 17 available.⁵⁸ That of course is not the case here.

18 cause NHL).

19 ⁵⁶ *See also Brock*, 874 F.2d 307 (concluding that *in vitro* studies are speculative and too difficult
 20 to extrapolate to whole humans because of the complexities of metabolism and uncertainties of
 21 dose equivalents); *Wade-Greaux v. Whitehall Labs.*, 874 F. Supp. 1441 (D.V.I. 1994) (finding
 22 that *in vitro* tests are of limited value in understanding the effects of human exposure); *Bourne*
 23 *v. E.I. DuPont De Nemours & Co.*, 189 F. Supp. 2d 482 (S.D. W. Va. 2002) (when experts use
 24 *in vitro* tests to show extrapolation to humans, the expert’s reliability is viewed with suspicion);
 25 *see also D. Brusick et al., Genetic Toxicology* in Hayes’ *Principles and Methods of Toxicology*
 26 at 1186 (6th ed. 2014) (explaining that *in vitro* experiments are “susceptible to false-positive
 27 responses generated by nonphysiological treatment conditions”).

24 ⁵⁷ *See Portier Dep.* 350:23-351:12 (conceding that the fact that a substance causes genotoxicity
 25 or oxidative stress does not establish that it is carcinogenic); D. Eaton, *Scientific Judgment and*
 26 *Toxic Torts – A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & Pol’y 5, 17 (2003)
 27 (stating that not all carcinogens increase cancer risk by causing mutation).

26 ⁵⁸ *See Monroe v. Zimmer U.S., Inc.*, 766 F. Supp. 2d 1012, 1029-30 (E.D. Cal. 2011)
 27 (considering *in vitro* study by plaintiffs’ expert because conducting epidemiological research in
 28 humans was not feasible); *In re Zicam Cold Remedy Mktg., Sales Practices & Prod. Liab. Litig.*,
 No. 09-md-2096-PHX-FJM, 2011 WL 798898, at *9-10 (D. Ariz. Feb. 24, 2011) (admitting
 specific *in vitro* study because of absence of human data).

1 In addition, mechanistic data is too speculative to extrapolate to disease in humans in a
 2 scientifically reliable way because mechanistic experiments do not use routes of exposure that
 3 replicate those experienced by humans. For example, many mechanistic studies upon which
 4 plaintiffs' experts rely expose rodents to glyphosate or GBHs by intraperitoneal ("IP")
 5 injections (injections directly into the body cavity, similar to an intravenous injection).⁵⁹
 6 Similarly, plaintiffs' experts rely on studies in which cells sitting in a petri dish were exposed
 7 directly to glyphosate or GBHs.⁶⁰ No plaintiff in this litigation alleges either route (or even a
 8 comparable route) of exposure. Causation opinions based on studies addressing artificial
 9 scenarios of exposure are inadmissible, where, as here, the experts offer no methodology for
 10 extrapolating from the resulting data to the actual circumstances of the case.⁶¹ Such opinions
 11 are particularly untrustworthy where, as here, epidemiology studies utilizing exposure routes
 12 that are the same as those alleged by plaintiffs here are available – and show no increased risk.⁶²

13 Similarly, as plaintiffs' experts necessarily concede, the "level of exposure is an
 14 important consideration in the formation of NHL from exposure to glyphosate." Jameson
 15 Report at 15. But the mechanistic studies test doses far in excess of even the most

17 ⁵⁹ *E.g.*, Portier Amended Report at 58-59, 72 (citing at least 10 IP administration studies);
 18 Jameson Report at 30 (citing multiple IP administration studies); Ritz Report at 25 (citing one
 19 IP administration study out of the five she discussed).

19 ⁶⁰ *E.g.*, Portier Amended Report at 56-61, 70-71 (citing at least 25 *in vitro* petri dish studies);
 20 Jameson Report at 30-31 (citing seven); Ritz Report at 25 (citing two); Neugut Report at 19
 21 (citing seven); Weisenburger Report at 9 (citing nine).

21 ⁶¹ *See, e.g., Joiner*, 522 U.S. at 144-45 (holding that district court did not abuse its discretion
 22 when it found that animal studies involving mice injected with large doses of alleged toxin were
 23 dissimilar to the plaintiffs' situation and thus were unreliable as a basis for expert's opinion as
 24 to causation); *Good v. Fluor Daniel Corp.*, 222 F. Supp. 2d 1236, 1244-46 (E.D. Wash. 2002)
 25 (finding an expert's methodology unreliable because his *in vitro* testing did not properly account
 26 for the different means of absorption in the laboratory instead of the body); *Haim v. HHS*, No.
 27 90-1031V, 1993 WL 346392, at *15 (Fed. Cl. Aug. 27, 1993) (holding expert's testing of
 28 chemical on rodents was not analogous to human exposure because he did not explain
 difference in effect when chemical was injected numerous times directly into brain of rodents as
 opposed to how chemical affected humans when humans were exposed only to one injection,
 and not directly into brain).

⁶² *See supra* at 10-21; *Lynch v. Merrell-National Labs*, 830 F.2d 1190, 1194 (1st Cir. 1987)
 (excluding lower-level scientific studies where they stood in the face of significant contrary
 epidemiological data); *In re "Agent Orange" Prod. Liab. Litig.*, 611 F. Supp. 1223, 1241
 (E.D.N.Y. 1985) (excluding lower-level studies of Agent Orange based partly on the court's
 earlier conclusion that there was significant contrary epidemiological data).

precautionary estimates of human exposure, rendering plaintiffs' experts' use of them invalid.⁶³ For example, multiple plaintiffs' experts rely on a Bolognesi, et al. 1997 study in rodents. Yet the experimental doses in that rodent study (300 mg/kg (glyphosate) or 900 mg/kg (GBH)) are thousands of times higher than real-world exposures. Compare C. Bolognesi et al., *Genotoxic Activity of Glyphosate and Its Technical Formulation Roundup*, 45 J. Agric. & Food Chem. 1957, 1958 (1997) with *supra* at 5 (highest farm worker exposure measured was 0.04 mg/kg and protective EPA reference dose is 2 mg/kg/day). Compounding the lack of scientific fit is the fact that at such high doses, cytotoxicity occurs, meaning that any effect seen is not the result of the compound itself, but of a secondary condition in which the cell is overwhelmed and damaged or destroyed. See Guidance Document on Revisions to OECD Genetic Toxicology Test Guidelines, Section 4.1.2 (cytotoxicity can lead to "biologically irrelevant positive results"); *Wade-Greaux*, 874 F. Supp. at 1154 (recognizing principle that at some dosage level, virtually any substance, even sugar or salt, can cause malformations).

Certain of the experts' mechanistic opinions are inadmissible for other reasons. Dr. Portier's opinions are derived at least in part by adding up the positive studies and performing more novel statistics instead of actually evaluating the methods and reliability of the studies themselves. See Portier Amended Report at 66 (summarizing genotoxicity studies by "Number Positive" and "Number Negative"); *id* at 69-70 (statistical analysis to determine "expected" number of positive studies). An amalgam opinion that lacks verification, general acceptance, or scientific reasoning or standards, is not derived by the scientific method and is inadmissible.⁶⁴

Plaintiffs' experts also rely on two *in vivo* studies (Bolognesi 2009 and Paz-y-Mino 2007) whose methodologies are so flawed that any opinions based on them must be excluded.⁶⁵

⁶³ *McClain*, 401 F.3d at 1242 (assessing general causation includes assessment of exposure at human-relevant levels); *In re Zicam*, 797 F. Supp. 2d at 945-46 (same); *Young*, 567 F. Supp. 2d at 133 (excluding expert testimony, noting that without information linking levels of plaintiffs' exposures with known hazardous levels of exposure, the expert's "testimony about the health effects of any such 'exposure' cannot possibly be anything other than conjecture").

⁶⁴ See *Young*, 567 F. Supp. 2d at 133; *Allen*, 102 F.3d at 198.

⁶⁵ For example, among other methodological flaws, Bolognesi et al. failed to control for exposures to confounding factors that may result in genotoxicity and failed to blind the investigators as to the exposure status (exposed vs. unexposed) of study participants to reduce the risk of bias. C. Bolognesi et al., *Biomonitoring of Genotoxic Risk in Agricultural Workers*

Moreover, the Bolognesi 2009 authors concluded that their study “indicates that the genotoxic risk potentially associated with exposure to [GBH] ... is low.” *Id.* Dr. Keith Solomon, one of the co-authors, explained that “[w]hen we looked at the differences in the micronuclei between those two groups [exposed and not exposed], we found no difference.” See R. Arnason, *Toxicologist Pans UN Glyphosate Report*, The Western Producer (Mar. 27, 2015), <http://www.producer.com/daily/toxicologist-pans-un-glyphosate-report/>. Similarly, the Paz-y-Mino authors have distanced themselves from their 2007 study cited by plaintiffs’ experts in a subsequent study⁶⁶ that found no evidence of genotoxicity in the same population.⁶⁷

Not surprisingly, both Bolognesi 2009 and Paz-y-Mino 2007 have been deemed low quality by EPA. EPA OPP at 196. Yet plaintiffs’ experts reach to interpret data in sweeping ways not supported by the studies and once again disagree with the study authors,⁶⁸ lacking any support for doing so other than their own impermissible speculation and *ipse dixit*. Their opinions must be excluded.⁶⁹

from *Five Columbian Regions: Association to Occupational Exposure to Glyphosate*, 72 J. Toxicology Env'tl. Health, Part A 986 (2009).

⁶⁶ Paz-y-Mino and others published another study of the same communities four years later. C. Paz-y-Mino et al., *Baseline determination in social, health, and genetic areas in communities affected by glyphosate aerial spraying on the northeastern Ecuadorian border*, 26 Rev Env'tl. Health 45 (2011). The 2011 study reported “no chromosomal alterations in the analyzed individuals” as a result of exposure to GBHs and identified a variety of factors that alone could cause any genotoxicity results discussed in the earlier report. *Id.* at 50.

⁶⁷ Flaws in the 2007 study include: (1) a lag time of two weeks to two months between the study subjects’ purported aerial exposure to glyphosate and when blood samples were collected, during which the subjects could have encountered any number of chemicals or exposures that confounded the results; (2) a study population that complained of various acute illnesses which could have confounded the results as viral and bacterial infections have been shown to cause DNA damage; (3) a poorly defined control population; (4) freezing of blood samples prior to testing and thus decreasing the reliability of the results; and (5) no indication that a single investigator reviewed the slides in a blinded manner to prevent differences in interpretation or bias in the analysis.

⁶⁸ *E.g.*, Portier Dep. 360:17-23 (when questioned regarding the Bolognesi 2009 study investigators’ conclusion of “low” genotoxic risk Dr. Portier stated “I don’t know how they could possibly come to that conclusion. I can’t imagine where they got that from this data.”).

⁶⁹ See *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1169 (E.D. Wash. 2009) (“Nothing in *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert.”) (citation omitted); *In re Accutane Prod. Liab. Litig.*, No. 8:04-MD-2523-T-30, 2009 WL 2496444, at *2 (M.D. Fla. Aug. 11, 2009) (“When an expert relies on the studies of others, he must not exceed the limitations the authors themselves place on the study.”), *aff’d*, 378 F. App’x 929 (11th Cir.

D. Plaintiffs' Experts' Opinions Are Otherwise Inadmissible To Prove General Causation.

Plaintiffs' experts purport to apply the guidelines of epidemiologist Sir Bradford Hill to the relevant glyphosate data as a sum-up of their opinions.⁷⁰ First published in 1965, the Bradford Hill guidelines are an oft-cited set of nine "viewpoints" or factors considered by epidemiologists when evaluating whether "the most likely interpretation" of "an association between two variables" is causation. See A. Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. R. Soc. Med. 295, 295 (1965) ("Bradford Hill").⁷¹ It is well-settled, however, that a necessary *predicate* to application of the guidelines is the existence of epidemiology that demonstrates a specific, clear-cut association between the two variables under examination (here, exposure to GBHs and NHL), and any such epidemiological association must be determined by scientifically valid reasoning and methodology. See, e.g., *Daubert*, 509 U.S. at 592-93; Bradford Hill at 295 (criteria applied only where observations "reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance").⁷²

No such association exists here. For there to be one warranting application of the Bradford Hill guidelines, epidemiology must eliminate confounding factors and differences between the compared subjects. See, e.g., *Daubert II*, 43 F.3d at 1321 ("an epidemiologist would

2010).

⁷⁰ Whether non-epidemiology experts such as Drs. Portier, Jameson, and Nabhan have the qualifications to apply Bradford Hill is "questionable." *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Practices & Prod. Liab. Litig.*, 174 F. Supp. 3d 911, 933 (D.S.C. 2016).

⁷¹ The nine factors are strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment, and analogy. Bradford Hill at 295-99. "None of [the] nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*." *Id.* at 299.

⁷² See *Soldo v. Sandoz Pharm. Corp.*, 244 F. Supp. 2d 434, 569 (W.D. Pa. Jan. 13, 2003) ("The Bradford Hill criteria start with an association demonstrated by epidemiology and then apply such criteria as the temporal sequence of events, the strength of the association, the consistency of the observed association, the dose-response relationship, and the biologic plausibility of the observed association.") (quotation omitted); *Dunn v. Sandoz Pharm. Corp.*, 275 F. Supp. 2d 672, 679 (M.D.N.C. 2003) (noting the "first step in the causation analysis pursuant to Bradford Hill is an epidemiological study that has identified an association between two variables") (citation omitted).

1 take a sample of the population and compare the frequency of birth defects in children whose
 2 mothers took Bendectin with the frequency of defects in children whose mothers did not ...”);
 3 *Hollander v. Sandoz Pharm. Corp.*, 95 F. Supp. 2d 1230, 1237 (W.D. Okla. 2000) (association
 4 cannot be established by studies that are “not controlled ... and do not eliminate confounding
 5 variables”), *aff’d*, 289 F.3d 1193 (10th Cir. 2002); *Soldo*, 244 F. Supp. 2d at 569 (same).
 6 Plaintiffs’ experts fail to meet this predicate.

7 Nor do plaintiffs’ experts faithfully apply the guidelines in any event. For example,
 8 retroactive case-control studies cannot reliably evaluate *temporality* because the cases were
 9 diagnosed with cancer before the study began. *Reference Manual* at 560-561 (where “both
 10 exposure and disease are determined in an individual at the same point in time, it is not possible
 11 to establish the temporal relationship between exposure and disease ... which would be
 12 necessary for drawing any casual inference.”) Point estimates for associations below a RR of 2.0
 13 would not satisfy the *strength* criterion. *See supra* at 12-21; *Reference Manual* at 612 n.193
 14 (“[S]tudies that find a relative risk less than 2.0 should not be sufficient for causation.”) The
 15 robust AHS cohort study found no evidence of a *dose response*. *See supra* at 12-14. No
 16 *consistent* positive data specific to glyphosate compounds across studies exists. *See* Neugut Dep.
 17 158:23-159:6. And *specificity* is not particularly useful for determining causality in cancer,
 18 because there are exposures associated with many cancer types. Fundamentally, plaintiffs’
 19 experts’ failures cannot be saved with an incantation of purportedly magic words.

20 Whatever wrap-up methodology they offer, plaintiffs’ experts also fail to meet the core
 21 *Daubert* factors. For example, as discussed above, many of plaintiffs’ experts rely on novel or
 22 untested theories – such as Dr. Portier’s ever-changing statistical techniques – with unknown
 23 error rates. *See supra* at 24-29. In other instances – such as Dr. Jameson’s hypothesized
 24 extrapolation from rodent data to human NHL – the error rate is simply impermissibly high.
 25 *See, e.g.*, Jameson Expert Dep. 304:20-25 (Q. “By the use of the term ‘potential,’ you mean that
 26 if an experimental animal study shows cancer, it has a more than 50 percent likelihood of being
 27 a human carcinogen, true? A. I don’t know that you can put a percentage on it.”). Likewise,
 28 many of plaintiffs’ experts – again such as Dr. Portier – advance their methodology specifically

1 for this litigation, meaning that those methods have never been subject to peer review and lack
 2 the general acceptance of the scientific community. *See supra* at 24-29. Plaintiffs’ experts –
 3 such as Drs. Ritz and Weisenburger – also rely on cherry-picked, biased, and confounded data
 4 in ways that are far from gaining general acceptance, and in fact have been rejected by the
 5 scientific community. *See supra* at n.24. They all miss in every particular instance their
 6 obligation to establish “fit.” And the proposition that hazard assessments – such as that
 7 conducted by Drs. Jameson, Neugut and Nabhan – can be used as surrogates for opinions of a
 8 causal relationship also is not accepted in the scientific community. *See supra* at 3 & n.6.

9 In sum, the methodologies that led to plaintiffs’ experts’ general causation conclusions
 10 have not been published, peer reviewed, generally accepted, or tested and the potential error is
 11 unknown or too high to satisfy *Daubert’s* exacting standards of reliability.

12 **II. MONSANTO IS ENTITLED TO SUMMARY JUDGMENT BECAUSE**
 13 **PLAINTIFFS HAVE FAILED TO PRESENT ADMISSIBLE EXPERT**
 14 **TESTIMONY TO SATISFY THEIR BURDEN OF PROVING GENERAL**
CAUSATION.

15 A district court “shall grant summary judgment if the movant shows that there is no
 16 genuine dispute as to any material fact and the movant is entitled to judgment as a matter of
 17 law.” Fed. R. Civ. P. 56(a). A “complete failure of proof concerning an essential element of
 18 the nonmoving party’s case necessarily renders all other facts immaterial.” *Celotex Corp. v.*
 19 *Catrett*, 477 U.S. 317, 323 (1986). When ruling on a summary judgment motion, a district court
 20 is required to consider only admissible evidence. *See, e.g., Orr v. Bank of America, NT & SA*,
 21 285 F.3d 764, 773 (9th Cir. 2002).

22 All claims asserted in this toxic tort litigation require plaintiffs to prove general
 23 causation. *See Golden v. CH2M Hill Hanford Group, Inc.*, 528 F.3d 681, 683 (9th Cir. 2008);
 24 *In re Hanford*, 292 F.3d at 1134; *Daubert II*, 43 F.3d at 1315. Here, general causation is a
 25 complex medical and scientific issue that is beyond the knowledge of lay jurors. Accordingly,
 26 plaintiffs must present expert testimony to prove general causation. *See Avila*, 633 F.3d at 836;
 27 *Hollander*, 289 F.3d at 1214; *In re Mirena IUD Prod. Liab. Litig.*, 202 F. Supp. 3d 304, 310-12
 28 (S.D.N.Y. 2016), *appeal docketed* Nos. 16-2890 & 16-3012 (2d Cir. Aug. 19, 2016).

Plaintiffs have no admissible expert testimony to prove general causation because the opinions of their experts (retained and non-retained) do not satisfy *Daubert*. Thus, Monsanto is entitled to summary judgment as a matter of law. *See, e.g., In re Zolof*, 858 F.3d at 800 (affirming MDL court’s exclusion of general causation opinions and entry of summary judgment in numerous personal injury cases); *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Practices & Prod. Liab. Litig.*, 227 F. Supp. 3d 452, 485, 491 (D.S.C. 2017) (MDL court granting summary judgment in numerous personal injury cases, based on exclusion of general causation opinions); *In re Mirena*, 202 F. Supp. 3d at 327-28 (same); *Arias*, 928 F. Supp. 2d at 24-26 (granting summary judgment in numerous personal injury cases, based on exclusion of general causation opinions regarding glyphosate and NHL).

CONCLUSION

The *Daubert* gatekeeping role requires the Court to “separate[] expert opinion evidence based on good grounds from subjective speculation that masquerades as scientific knowledge.” *Glastetter v. Novartis Pharm. Corp.*, 252 F.3d 986, 989 (8th Cir. 2001) (quotation omitted). In this case, plaintiffs’ experts have presented the latter – not the former. This Court should end the masquerade by excluding all of plaintiffs’ experts’ general causation opinions and entering summary judgment for Monsanto in all Roundup[®] lawsuits pending in this Court.

DATED: October 6, 2017

Respectfully submitted,

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