IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF ILLINOIS

IN RE: PARAQUAT PRODUCTS LIABILITY LITIGATION	
This document relates to: *Richter, et al., v. Syngenta AG, et al.*, No. 3:21-cv-571 *Fuller, et al., v. Syngenta AG, et al.*, No. 3:21-pq-836 *Burgener v. Syngenta AG, et al.*, No. 3:21-pq-1218 *Coward v. Syngenta AG, et al.*, No. 3:21-pq-1560	Case No. 3:21-md-3004-NJR MDL No. 3004 Hon. Judge Nancy J. Rosenstengel

DEFENDANTS' ROADMAP BRIEF IN SUPPORT OF SUMMARY JUDGMENT AND RULE 702 MOTIONS

INTRODUCTION

Parkinson's disease ("PD") has existed for thousands of years—since humans began living long enough to develop it. As one of the most common age-related neurological diseases in the world, it has been exhaustively studied since it was first given a name 200 years ago. Paraquat, for its part, has been on the U.S. market for nearly 60 years and has been sold all over the world. Thousands of studies—by Defendants and independent scientists alike—have evaluated its safety. And the hypothesis that a connection exists between paraquat and PD has been tested extensively since it was first raised decades ago. But after all that research, ***no study in the peer-reviewed literature concludes that paraquat causes PD.*** To the contrary, the independent, peer-reviewed literature has reported "a consensus in the scientific community that the available evidence ***does not*** warrant a claim that paraquat causes [PD]." Ex. 1 (Weed 2021) at 183 (emphasis added). That was the state of the science when this MDL was formed; it remains so today.

Nevertheless, Plaintiffs offer a single expert willing to opine that paraquat causes PD; others adopt and rely on that assumption. But in their scientific work, Plaintiffs' experts have said the opposite, or failed to address the question at all. Their opinions in this litigation are not based on some new truth that the entire scientific community has failed to discover; rather, Plaintiffs' experts abandoned rigorous scientific methodology to manufacture unreliable, novel opinions for purposes of this litigation. But as the Seventh Circuit has cautioned: "Law lags science; it does not lead it." *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996); *see also In re Zantac (Ranitidine) Prod. Liab. Litig.*, 2022 WL 17480906, at *4 (S.D. Fla. Dec. 6, 2022) (excluding Plaintiffs' causation experts because "there is no scientist outside this litigation who concluded that ranitidine causes cancer"). That principle, if applied faithfully here, should resolve the remaining bellwether cases in Defendants' favor.

I. OVERVIEW OF THE SCIENTIFIC CONSENSUS: PARAQUAT DOES NOT CAUSE PD.

PD is a naturally occurring neurodegenerative disease that affects about one million people in North America and more than six million worldwide. *See* Ex. 2 (Elbaz 2002) at 29. First identified by Dr. James Parkinson in 1817, PD is the second most common age-related neurogenerative disease in the world. *See* Ex. 3 (Olanow 2022) at 3122. But PD afflicted humans long before Dr. Parkinson's day, and centuries before paraquat or other synthetic chemicals existed; descriptions of its clinical features were recorded as far back as Ancient Egypt. *See generally* Ex. 4 (Raudino 2012). Despite centuries of study, PD's cause remains unknown in 85-90% of cases, *see* Ex. 3 (Olanow 2022) at 3130, with genetic mutations accounting for the remaining 10-15%, *see* Ex. 5 (Blauwendraat 2020) at 170; Ex. 6 (Lang 2020) at 2417.

Paraquat was first sold in the U.S. in 1964, but scientists did not even hypothesize a potential connection to PD until the mid-1980s. At that time, doctors discovered a chemical byproduct of synthetic heroin production (called MPTP) that could cause rapid-onset PD-like motor symptoms ("parkinsonism")—but not PD—in drug users who injected MPTP-contaminated heroin. *See* Ex. 7 (Langston 1983) at 979-80. This led scientists to hypothesize that environmental factors might cause PD. Because paraquat bears some superficial molecular similarity to MPTP, it was one of many chemicals that scientists began to investigate. *See* Ex. 8 (Langston 2017) at S16. Research revealed important differences between MPTP and paraquat, however, including that paraquat does not cause rapid-onset movement disorders in humans—even at high doses. Ex. 9 (A. Lang Dep. Tr.) at 202:19-205:13.

In the decades since, hundreds of studies have been conducted to investigate the hypothesis that paraquat might cause PD. These studies fall into two broad categories: studies of human populations exposed to paraquat, and studies of animals (mostly rodents) exposed to high doses of paraquat (mostly by injection). This overall body of evidence has repeatedly been analyzed in

review articles, where scientists draw conclusions about the current state of scientific evidence. Ex. 9 (A. Lang Dep. Tr.) at 215:19-216:5. Not a single review article has concluded that paraquat causes PD, as even Plaintiffs' own expert in PD etiology (causation), Dr. Anthony Lang, admitted at his deposition. Ex. 9 (A. Lang Dep. Tr.) at 214:22-215:24; 47:25-49:2.

Instead, the consensus in the peer reviewed literature is that the available evidence does ***not*** show that paraquat causes PD. Recently, the journal *NeuroToxicology* published a peer-reviewed "review of reviews" that assessed the published review articles "where epidemiological studies on paraquat and [PD] were collected, described, and interpreted along with discussions of biological plausibility, that is, the mechanism by which paraquat could induce [PD] at the molecular level." Ex. 1 (Weed 2021) at 180. That study reviewed the work of 46 scientists across 12 peer-reviewed articles, spanning from 2006 to 2019. *Id*. at 181. After surveying that field, it reported that no review concluded paraquat causes PD (regardless of methods used or funding source), and instead found "a consensus in the scientific community that the available evidence ***does not warrant a claim that paraquat causes Parkinson's disease***." *Id.* at 183 (emphasis added).

That 2021 article is not the first to reach this opinion after surveying the literature. Independent scientists have done so time and again when assessing the paraquat-PD hypothesis. For example, in 2009, a multidisciplinary group of experts (including toxicologists, epidemiologists, geneticists, neuroscientists, and medical practitioners) met "to assess what is known about the contribution of environmental factors to PD" and "[t]o identify conclusions that could be drawn with confidence from existing data," among other things. Ex. 10 (Bronstein 2009) at 118. That group evaluated dozens of environmental factors hypothesized as potential causes of PD, including dairy intake, lack of exercise, genetics, vitamin intake, farming, fat consumption, well water, increased BMI, higher educational attainment, and paraquat, among many others. The scientists placed each factor

in one of four categories according to the strength of evidence linking them to PD. *See id.* at 118-19. After extensive review, this group of scientists placed paraquat in the ***lowest possible category***: insufficient evidence to determine whether there is any association between paraquat and PD (let alone a causal relationship). *Id.* at 119.

The Agricultural Health Study ("AHS")—the longest-term prospective epidemiology study ever conducted of pesticide exposure and PD in U.S. agricultural workers—recently reached the same conclusion. Ex. 11 (Shrestha 2020).[1] The AHS was conducted in the U.S. by independent scientists working for various public health agencies and followed more than 38,000 chemical applicators plus over 27,000 of their spouses (66,110 participants in total) for 25 years. *See id.* at 2. In 2020, AHS scientists published their long-awaited findings and reported ***no statistically significant increased risk***. In fact, those who used paraquat the most had the ***lowest*** PD risk—25% lower than those who never used paraquat at all. *See id.* at Table 4.

The EPA agrees too: There is insufficient evidence to support a claim that paraquat causes PD. Since the paraquat-PD hypothesis emerged in the 1980s, the EPA has twice comprehensively reevaluated paraquat's safety; each time, the agency reaffirmed that it is safe for its intended use as an herbicide. Ex. 13 (EPA July 2021) at 17-18. An additional review in 2021 specifically evaluated the paraquat-PD hypothesis, determining that there was insufficient evidence of a clear associative or causal relationship paraquat exposure and PD. *Id.* at 17. As part of the EPA's most recent review process, a panel of EPA scientists, in conjunction with the U.S. National Toxicology Program, completed an eight-year study (spanning three separate administrations) analyzing all of

[1] The AHS study update by Shreshta et al. was a follow-up to an older study that previously reported a statistically significant increased risk. *See* Ex. 14 (Tanner 2011). After considering a larger and more updated data set, the AHS failed to replicate the findings of the earlier study. *See* Ex. 12 (EPA June 22, 2021) at 77 ("Notably, Shrestha et al. (2020) did not replicate the earlier AHS findings reported by Tanner et al. (2011)….").

the evidence in the existing literature on paraquat and "PD-like" pathologies—including 28 epidemiology studies, 217 animal studies, and 244 lab studies. *See* Ex. 15 (EPA June 2019) at 29. The EPA, like the broader scientific community, determined that "the weight of evidence was insufficient to link paraquat exposure from pesticidal use of US registered products to [PD] in humans." *Id*. at 4, 6, 7; Ex. 13 (EPA July 2021) at 18. What is more, the EPA's assessment specifically rejected comments like those advanced by Plaintiffs here, including that "laboratory animal and mechanistic data" might provide "evidence that there is an association between paraquat exposure and PD." Ex. 16 (EPA Sept. 2020) at 2 (explaining that high-dose injections of animals with paraquat were not useful in assessing risk to farmers, and reiterating "conclusion that the weight of evidence was insufficient to link paraquat exposure … to PD in humans").

Thus, over the past five years, there have been three independent, exhaustive scientific analyses evaluating the paraquat-PD hypothesis: (1) the *NeuroToxicology* review of the scientific literature; (2) updated results from the AHS epidemiology study of U.S. farmers; and (3) the EPA's latest safety assessment of paraquat. Those scientists all agree: No evidence establishes that paraquat causes PD. These studies contradict and undermine the handful of smaller, older studies Plaintiffs cite that had suggested further study of a potential association between paraquat and PD.

Tellingly, outside this litigation, Plaintiffs' own experts have consistently ***denied*** that paraquat is a proven cause of PD. For example, Dr. Peter Spencer has repeatedly published texts concluding "[t]here is ***no evidence*** that paraquat induces parkinsonism." Ex. 17 (Spencer 1995) at 328 (emphasis added); *see also* Ex. 18 (Spencer 2000) at 943 (calling it "unlikely that paraquat is neurotoxic to humans …. Epidemiological studies of rural and urban populations have ***failed to demonstrate a robust correlation*** between historical deployment of paraquat and clusters of [PD] cases." (emphasis added)). Similarly, Plaintiffs' specific-causation expert, Dr. Lang, wrote a book

on PD in which he discussed the scientific evidence of associations between pesticides (including paraquat) and PD, and concluded that it does ***not*** show any consistent association—let alone causation. Ex. 19 (Lang 2013) at 19-21, 28-30.

At bottom, Plaintiffs' experts offer causation opinions that they have never submitted to the rigors of peer review and publication, that run counter to the scientific consensus, and that contradict their own out-of-court publications. In doing so, they fail to employ the same level of scientific rigor they use outside of court and instead rest on unreliable analyses, speculative extrapolation from flawed evidence, and blind adoption of lawyer-fed assumptions. This is precisely the kind of unreliable litigation-science the Court must guard against as gatekeeper.

II. OVERVIEW OF RULE 702 MOTIONS: PLAINTIFFS' EXPERTS SHOULD BE EXCLUDED.

Plaintiffs found a single expert, ***Dr. Martin Wells***, willing to opine that a causal association exists between paraquat and PD. Dr. Wells is an oft-excluded statistician who—until now—has ***disclaimed*** his ability to opine on general causation. But here, Dr. Wells claims to have the expertise to offer a sweeping general causation opinion that the scientific community has never reached. As in prior cases, Dr. Wells employs "ramshackle," "results-driven" methodologies (if they can be called methodologies at all) that fail to "objectively" examine the data. *See* Defs.' Mot. To Exclude Wells at 1. Indeed, Dr. Wells reaches his opinions only by cherry-picking data and disregarding contrary evidence like the AHS study. After his deposition, Dr. Wells attempted to backfill—belatedly inventing a brand new (and previously undisclosed) "methodology" that, not coincidentally, resulted in the exact same conclusions he had already reached. Neither of his reports comes anywhere close to meeting Rule 702 or *Daubert*'s standards.

For specific causation, Plaintiffs claim to rely on ***Dr. Anthony Lang***, a neurologist. In reality, however, Dr. Lang's role is merely to serve as a mouthpiece for Dr. Wells, lending an air of credibility to opinions that Dr. Lang has *rejected* outside this litigation. Dr. Lang is the only of

Plaintiffs' experts who is an expert in PD etiology, and has published extensively on the potential causes of PD. Yet rather than *independently analyze* those issues (or any other issue in the case), Dr. Lang accepts two critical *assumptions* provided to him by Plaintiffs' counsel: (1) that Dr. Wells is correct that paraquat causes a 2.8x increase in the risk of PD; and (2) that the increased risk Dr. Wells estimated can be applied to each Plaintiff. Relying exclusively on these assumptions—and without assessing or pressure-testing them in any way—Dr. Lang opines that those assumptions mean paraquat caused each bellwether Plaintiff's PD, without any additional analysis. Dr. Lang's parroting of Dr. Wells's opinions is not expert testimony. Moreover, Dr. Lang's wholesale reliance on Dr. Wells's conclusions means no specific causation expert has attempted to adjust for individual dose, family history, or any other established PD risk factors. Any reliable specific causation opinion must account for those issues—but Dr. Lang's does not.[2]

Both causation opinions also rest, in part, on toxicologist ***Dr. Vanessa Fitsanakis***, who says it is biologically plausible that paraquat causes PD. Dr. Fitsanakis reaches this conclusion based on experimental studies in which scientists *injected animals* (usually rodents) with doses of paraquat *thousands of times larger* than the doses human workers absorb in real-world occupational use. To justify that leap, Dr. Fitsanakis invents a "theoretical exposure" model to argue that a worker could "hypothetically" be exposed to high doses equivalent to those injected into animals in labs. Among other extraordinary assumptions, that model assumes a paraquat user would spray paraquat for seven consecutive days while covered from head to toe in tens of thousands of open scratches and abrasions. Unsurprisingly, no Plaintiff alleges any facts even approaching those assumptions. Dr. Fitsanakis's testimony is so untethered from both science and the record as to be inadmissible.

[2] The testimony of Plaintiff Coward's specific causation expert—***Dr. Binit Shah***—suffers from the same problems and should be excluded for the same reasons.

Similar problems infect the opinions of ***Mr. Stephen Petty***. Real-world scientific studies have shown that human applicators receive tiny doses of paraquat (if any dose at all) thousands of times less than those used in the animal studies Plaintiffs' experts rely on. So Plaintiffs strategically chose not to have Mr. Petty calculate any Plaintiff's dose. Instead, he conducted unrecorded interviews of Plaintiffs, created summaries that contradict Plaintiffs' sworn testimony, and then destroyed his contemporaneous notes. Mr. Petty also offers opinions based on numerous unsupported analytical leaps, cherry-picked data, and a for-litigation methodology that contradicts scientific consensus. In any event, there is no evidence that paraquat causes PD even at Mr. Petty's (and Dr. Fitsanakis's) massively inflated theorized exposures.

Ms. Cynthia Rando presents testimony on the supposed insufficiency of paraquat's warnings, opining that no warning could ever be sufficient if a product carries a risk of PD, as she assumes (based on instructions from Plaintiffs' counsel) that paraquat does. She therefore concludes that paraquat should not have been sold (with any label) unless the product itself was somehow redesigned. In other words, Ms. Rando purports to opine that paraquat is defectively designed—yet does so with no expertise whatsoever in pesticides, and by applying only her own idiosyncratic standard rather than applicable law. Moreover, Ms. Rando's opinions are based on unsupported assumptions that not even Plaintiffs' other experts sponsor, and an unreliable methodology that does little more than depict in visual form those baseless assumptions.

Plaintiffs offer two experts, ***Dr. Peter Spencer*** and ***Dr. Michael Siegel***, who opine on what they believe Defendants should have known or done differently over time: Dr. Spencer focuses on the period prior to 1986, while Dr. Siegel's opinions reach to the present. Neither expert followed any reliable methodology. Dr. Spencer admitted he viewed his role as looking for ***any*** evidence that paraquat could have neurological effects—regardless of what the overall scientific literature

showed. After selecting this biased sample, Dr. Spencer declined to apply any objective standard, asking himself only "what Peter Spencer would have done prior to 1980." And while Dr. Siegel claims he applied a weight-of-the-evidence approach, all the weighing took place only in his mind, and his conclusions are connected to the evidence based only on his *ipse dixit*. These unreliable, nonreplicable approaches cannot pass muster under Rule 702 or *Daubert*.

Finally, Plaintiffs rely on ***Dr. David Mortensen***, an agronomist, to opine that Plaintiffs had viable alternatives to paraquat. But those Plaintiff-specific opinions were provided *after* Plaintiffs' expert report deadline, yet *before* Defendants served their own reports. Because Dr. Mortensen's Plaintiff-specific opinions were not offered in either his principal report nor in a true rebuttal report, those opinions are improper and should be stricken. Regardless, the opinions are also unreliable because Dr. Mortensen did not follow any of his usual methods in reaching them. He merely reviewed a one-page bullet-point summary of facts prepared by Plaintiffs' counsel.

III. THE COURT SHOULD GRANT SUMMARY JUDGMENT TO DEFENDANTS.

Defendants have also filed three summary judgment motions, which should be granted even if Plaintiffs' expert testimony is admitted—and *a fortiori* if it is excluded.

As the ***Joint Motion*** explains, Plaintiffs' design-defect claims fail as a matter of law because paraquat is the characteristic ingredient of paraquat products—not a design choice. The proper theory in this situation is failure-to-warn, but that theory also fails here: For one, Plaintiffs failed to heed the already-extensive label warnings on paraquat, making it impossible to conclude that a PD warning would have avoided their injuries. For another, failure-to-warn claims are preempted by federal law given the EPA's position that there is no established causal link between paraquat and PD. The counts under the Illinois Consumer Fraud Act fail for the same and additional reasons, including that they are barred by a statutory safe harbor and that Plaintiffs lack statutory standing. And the implied warranty claims are coextensive with all the others; under Florida law,

moreover, implied warranty plaintiffs must be in privity with the defendant, which Mr. Coward admittedly was not. Finally, Plaintiffs fail to establish causation as to all claims because their experts do not provide either a threshold dose at which paraquat is capable of causing PD, or calculate specific doses that the individual Plaintiffs absorbed.

Chevron's Motion explains that it is independently entitled to summary judgment because its predecessor left the paraquat market over 35 years ago, in 1986. Combined with Plaintiffs' complete failure to establish even one of the three elements of fraudulent concealment tolling, that means the Illinois strict liability and warranty claims are barred by the applicable statutes of repose and limitations. And because even Plaintiffs' own experts admit there was no scientific consensus in 1986 that paraquat could cause PD, all of the remaining claims are barred by the state-of-the-art doctrine. Sellers are not expected to know what science does not. At minimum, those facts mean there is no basis for any punitive damages claims against Chevron.

Finally, ***Syngenta's Motion*** explains that Syngenta is independently entitled to summary judgment on certain claims because they are time-barred. In particular, testimony by Plaintiffs Burgener, Richter, and Coward conclusively establishes that many of their claims accrued outside the applicable statutes of limitation and repose. Those three Plaintiffs also cannot rely on the doctrine of fraudulent concealment to toll the limitations period because Syngenta acknowledged the paraquat-PD hypothesis many years ago, and Plaintiffs' ordinary diligence into the nature of their injuries would have revealed the existence of their asserted claims—implausible as they are.

* * *

In sum, Plaintiffs lack the necessary support in fact, law, and expert opinion to support their various claims at trial. The Court should thus grant summary judgment to Defendants.

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CERTIFICATE OF SERVICE

The undersigned hereby certifies that on June 7, 2023, the foregoing was electronically filed using the CM/ECF system, which will send notification of such filing to all counsel of record.

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