IARC's Precautionary Science:

How the WHO Cancer Research Agency Misinforms Regulation and Litigation

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The views and opinions presented in this paper are those of the author, and not necessarily shared by his clients, colleagues, friends, and family, although they should be. I have benefited from many discussions with friends and colleagues, but any errors in this *Monograph* are the sole property of the author. Facts on the other hand are stubborn things, and we are all stuck with them. No artificial intelligence was involved in writing this paper.

FOREWORD

By
Richard A. Williams, Ph.D.[1]

I spent 27 years in the bowels of the Food and Drug Administration working on food safety and nutrition policy as an economist and risk analyst. Following that, I led a group of attorneys and economists at the Mercatus Center studying all federal regulations. One insight I have taken from this career is that despite being stuffed with scientific experts, getting the science right is extremely difficult for agencies. An additional six years as the Board Chair for Center for Truth in Science convinced me it is even harder for courts to get it right. With respect to toxic torts: "Environmental wrongful death litigation is functioning as a de facto public policy instrument—filling regulatory gaps that governments have been too slow or too politically constrained to address."[2] But that works only if they get the science right. Even if they do, justice may be achieved in a lawsuit for individuals who have been harmed, but many who do not have traditional "standing," and who are negatively impacted by the trade-offs inherent the resulting policy, are left unconsidered.

We currently spend somewhere between $2[3] and $3[4] trillion annually on federal regulations, much of that on health and safety risk regulations from FDA, USDA OSHA, EPA, HUD, and DOT. The number of actual rules contained in federal regulations exceeds 1 million[5] and that figure does not include state regulations. One estimate,[6] for example, suggests that the economy is roughly a quarter smaller than it would be without federal regulations. Annual tort costs have been estimated at $443 billion, and they often produce the same type of economic drag as regulations. Worse, when we get the underlying science wrong, we are not just wasting money, we are putting people's lives at risk.

Why? First, both regulations and tort trials produce costs that are borne by consumers, workers, and stockholders in the form of higher prices for consumers, fewer wage increases for workers, and reduced returns for stockholders. It is well known that richer is safer.

Second, reductions in consumer income and lower wages for workers mean that consumers and workers have less money to spend on reducing risks in their own lives—risks that may be considerably larger than those addressed in torts and regulations. Higher prices for healthy foods, gym memberships, safer cars, medical check-ups, and smoke alarms mean that fewer people can afford them. When regulations and lawsuits choke income from consumers, it is usually the least well-off people who see fewer benefits from rules and cannot afford private safety expenditures. As Diana

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[2] Hannah Fischer-Lauder, *From Pollution to Precedent: Corporate Liability in Environmental Wrongful Death*, IMPAKTER, Mar. 16, 2026, https://perma.cc/5RKP-J8Q9.

[3] House Budget Committee, *Competitive Enterprise Institute: Burdensome Federal Regulations Cost Economy $2 Trillion Annually*, Apr. 30, 2025, https://perma.cc/6XQ6-B3ET.

[4] Clyde Wayne Crews Jr., *National Association of Manufacturers Pegs Cost of Regulation at $3.079 Trillion*, FORBES.COM, Dec. 28, 2023, https://perma.cc/7BKL-RJMA.

[5] Patrick McLaughlin, Nita Ghei, & Michael Wilt, *Regulatory Accumulation and its Costs*, Mercatus Policy Brief, May 2016, https://perma.cc/V7GB-SNCY.

[6] *Id.*

Thomas puts it, "By focusing on the mitigation of low-probability risks with higher costs, regulation reflects the preferences of high-income households and effectively redistributes wealth from the poor to the middle class and the rich."[7]

Third, when firms must comply with thousands of regulations and adverse tort decisions, particularly those addressing phantom risks—compliance costs leave fewer resources for firms making investments in safer products and safer workplaces.

When products or practices are restricted by regulation or tort liability, even greater dangers may arise if they are replaced by more harmful substitutes. If, for example, a pesticide such as glyphosate is banned, the chemical that takes its place may carry even higher risk. Some risk/risk issues may be subtle. Playground equipment like swings and teeter totters may be dangerous, but keeping kids away from playgrounds may lead to activities like video games in place of exercise. The safer choice is not always a straightforward thing. As Aaron Wildavsky said, "safety must be discovered, not merely chosen."[8]

These unintended consequences—crowding out more efficient risk spending and creating risk/risk trade-offs—are compounded when we wrongly overestimate target risks in regulatory and legal decisions. How does this happen? One way we get science wrong is by stopping short of getting the evidence needed to make a reasoned judgement about causality. That brings us to the first problem with the International Agency for Research on Cancer (IARC), the subject of this Washington Legal Foundation *Monograph*. By their own admission, IARC determines that a chemical or practice is *linked* to cancer, not that it *causes* cancer.

That link is called a "hazard analysis" and it is only the first step in determining whether something poses an actual risk. "Hazard" is an unfortunate term because it implies that something dangerous has been found. A trained assassin could use a rolled-up magazine or a pencil to kill someone, i.e., hazards, but we don't regulate magazines and pencils on that basis.

The steps in a risk assessment are 1) hazard identification—finding links, i.e., *potential* sources of harm; 2) exposure assessment—evaluating the level or amount of the chemical exposure; 3) dose response assessment—the likelihood of harm based on the amount of exposure (or threshold) and the severity of the health effect; 4) risk characterization—evaluation on whether, for example, the risk is *de minimis* and needs no further consideration; and 5) risk management—assessing options for mitigation of risk.

Using a hazard finding such as IARC's as dispositive evidence of carcinogenicity is like a skilled prosecutor in a grand jury proceeding who can "indict a ham sandwich."[9] The ham sandwich may be guilty, but hazard identification alone is a long way from proving guilt.

But even when exposure information is considered and there is a dose-response relationship, it still doesn't guarantee that there is causation. For example, more old people go to church, and more church goers get cancer so we could say that going to church causes cancer. That is a logical fallacy—*post hoc ergo propter hoc*. In fact, older people get cancer because our evolutionary defenses against carcinogens decline with age.

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[7] Diana Thomas, *Regressive Effects of Regulation*, Mercatus Working Paper, Nov. 27, 2012, https://perma.cc/B8KW-ATUM.

[8] AARON WILDAVSKY, SEARCHING FOR SAFETY, 1988, https://perma.cc/T5PM-LWYY.

[9] Historical Society of the New York Courts, Sol Wachtler, https://perma.cc/HK9H-3YQD.

Causation beyond dose-response is difficult to find as it requires studies like randomized controlled trials, often not possible for human studies (epidemiology) but common with experimental animal studies. Unfortunately, the scientific literature (and regulations and court cases) is full of associations, and it doesn't take a lot of imagination to see why they are not terribly helpful without causation.

Finally, many chemicals or practices are harmful at high doses or exposure levels but have health benefits at low doses. For example, resveratrol in red wine can be toxic at high doses but has anti-aging properties at low doses. High doses of radiation can kill but low doses stimulate resistance to disease. Finally, water is not just beneficial but essential for life at lower doses but drink too much too fast and you will die. Ignoring low-dose or activity benefits (called hormesis) may result in decisions that ban helpful chemicals or activities.

Even if we place IARC hazard findings in their proper role as only the first step in a risk assessment, a further problem remains.

What problem? As Nathan Schachtman explains in detail below, IARC appears to have institutional motivations to classify the chemicals it studies as carcinogens. In this case, the motivation appears to be the same as a similar organization, Collegium Ramazzini and, for that matter, the same as many regulatory agencies. Their shared motivation is to obey a proverb, "better safe than sorry." Europe gave this proverb a fancy name in the 1980s, the "Precautionary Principle," but it still essentially says, if there is doubt, throw it out (find it guilty and eliminate it).

The 1957 movie *12 Angry Men* offers a vivid illustration of motivated reasoning. The final holdout for a guilty verdict—Juror No. 3, played by Lee J. Cobb—ultimately admits under intense scrutiny that he wanted to serve as "judge, jury, and executioner" because the defendant reminded him of his estranged son. Like so many in the science world, he harbored an underlying motivation that pulled him away from an honest search for the truth.

The pattern is reinforced by other incentives. Finding and publicizing a new link gets professors funding, international invitations, and tenure. It also works for the media (if it bleeds it leads), and for regulatory agencies (more funding). For some people, it fits their political philosophy that corporations need to be taken down because they "put profits in front of people."

When truth takes a back seat to other motivations, good science also gets left behind. Evidence of poor practice in synthesizing science includes cherry-picking studies, (including only those that support the predetermined outcome), claiming causation when only associations have been found, overreliance on studies of insufficient duration or sample size, and treating high-dose animal studies as dispositive for human carcinogenicity.

IARC, perhaps because of a deep-seated belief in the precautionary proverb, appears to be guilty of all of these. Many organizations now employ protocols designed to guard against such bias, including pre-registration of research plans with explicit inclusion and exclusion criteria, open and transparent review processes, and requirements for balanced research teams. IARC has not adopted any of them.

Unfortunately, IARC's hazard findings are often the result of motivated reasoning or shortcutting the risk assessment process. Getting science wrong—whether through motivated reasoning, uncritical application of the precautionary principle, or conflating hazard with risk—threatens not only our economic well-being, but our health as well.

IARC's PRECAUTIONARY SCIENCE:
How the WHO Cancer Research Agency Misinforms Regulation and Litigation

INTRODUCTION

The United Nations established the World Health Organization (WHO) in 1948, for the salutary purpose of promoting world health. In 1965, WHO created the International Agency for Research on Cancer (IARC) as an independent organization, with headquarters in Lyon, France. The initial scope of IARC's mission broadly included research on the geographical distribution of cancer, as well as the diagnosis, screening, and prevention of cancer. In 1970, several years into IARC's existence and in the midst of growing debate, rhetoric, and concern over the causes of cancer, IARC created an advisory committee on environmental carcinogenesis, to create a list of carcinogenic chemicals.[1] The committee's goal was to evaluate carcinogenic risk of human cancer from specific chemical exposures, and to prepare monographs that reviewed the evidence for carcinogenicity, with qualitative classifications of the strength of the available evidence, for each chemical. The first IARC working group met in Geneva in December 1971 to consider the carcinogenicity of 19 different chemicals. IARC published volume one in its monograph series, based upon this first meeting, the following year.

The IARC monograph program has continued to the present. The most recent IARC working group meeting took place in November 2025, and will result in volume 140, with evaluations of atrazine, alachlor, and vinclozolin.[2] In the half century or so between volumes one and 140, the IARC monograph and classification program has evolved. The initial focus on chemicals expanded to a broader category of "agents," which includes microbes (e.g., various viruses and bacteria), lifestyle factors (e.g., night shiftwork), and complex exposures (e.g., tobacco smoke, rubber manufacturing, firefighting). The initial goal of identifying risks changed to focus on hazards, which, as we will see, may or may not translate into quantifiable risks.

IARC did not formally adopt hazard classification categories until 1987. Previously, working groups explained their conclusions in narrative descriptions, with qualifications as they saw fit.[3] The evaluative process used by working groups to make their assessments has changed from the first monograph. In 1978, with volume 17, IARC included some introductory comments in the form of a "preamble," which described the working group's approach to its decisions.[4] The *Preamble* was modified on several occasions, with the most recent version published in 2019. The current *Preamble* is a high-level guidance document on IARC's classification scheme, the meaning of the various classes or "groups" of agents, and the requirements for classification into a specific class of carcinogen.

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[1]John Higginson, *The International Agency for Research on Cancer: A Brief Review of Its History, Mission, and Program*, 43 TOX. SCI. 79, 79 (1998); RODOLPHO SARACCI & CHRISTOPHER WILD, THE INTERNATIONAL AGENCY FOR RESEARCHON CANCER: THE FIRST 50 YEARS, 1965 – 2015 (2015).

[2]Russell Cattley, *et al.*, *Carcinogenicity of atrazine, alachlor, and vinclozolin*, 27 LANCET ONCOL. 11 (2026).

[3]RODOLPHO SARACCI & CHRISTOPHER WILD, *supra* note 1, at 145-47.

[4]17 IARC MONOGRAPHON SOME N-NITROSO COMPOUNDS (1978).

In the same half century since IARC began its carcinogenicity assessments, regulatory agencies in the United States have grown dramatically. The concern over environmental carcinogenesis that inspired the creation of IARC, as well as federal regulatory agencies, such as the United States Environmental Protection Agency (EPA), and the Occupational Safety and Health Administration (OSHA), also fueled the genesis and proliferation of environmental activist organizations.

Around the time that IARC embarked on its monograph program, the notion that in matters of safety, we should err on the side of safety, was formally adopted in German law in the 1970s.[5] This notion quickly became the galvanizing principle of the environmentalist movement. In 1992, a United Nations conference on the environment, in Rio de Janeiro, produced a "declaration" of principles, one of which insisted that:

> [w]here there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.[6]

Similar declarations proliferated in Europe, and among environmental activists in the United States. In 1998, a group of activists attended a conference at Wingspread, the headquarters of the Johnson Foundation in Racine, Wisconsin. The group issued a statement, which has come to be known as the Wingspread Statement, and which has both inspired further activism, and misled litigation, regulation, and legislation:

> When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. In this context the proponent of an activity, rather than the public, should bear the burden of proof.[7]

Similar declarations followed, and several versions of this precautionary principle have been incorporated into foreign and domestic regulatory law.

The precautionary principle became a battle cry of the environmentalist movement, but it has also run into withering legal and scholarly criticism.[8] The principle insists that action must be taken in the absence of certainty to prevent harm. Although only a few people seek to perpetuate harm, the precautionary principle's goal of preventing harm is incoherent. If we know that a regulation would prevent harm, then we would know that the prohibited activity actually causes the harm of concern; that is, the prohibited activity is the but-for cause of the harm. Otherwise, the

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[5]GARY E. MARCHANT & KENNETH L. MOSSMAN, ARBITRARY AND CAPRICIOUS: THE PRECAUTIONARY PRINCIPLE IN THE EUROPEAN COURTS 5 (2004).

[6]REPORT OF THE UNITED NATIONS CONFERENCE ON THE ENVIRONMENT & DEVELOPMENT, vol. 1, Rio de Janeiro, 3-14 June 1992 (1993), https://perma.cc/93GT-4W2L.

[7]*Wingspread Conference on the Precautionary Principle* (Jan. 26, 1998), https://perma.cc/R6N7-9PLR (among those in attendance were Carl Cranor and David Ozonoff, both members of the Collegium Ramazzini; their testimonial and litigation adventures are discussed below).

[8]*See, e.g.*, MICHAEL S. GREVE & FRED L. SMITH, eds., ENVIRONMENTAL POLITICS: PUBLIC COSTS, PRIVATE REWARDS (1992); GARY E. MARCHANT & KENNETH L. MOSSMAN, *supra* note 5; CASS R. SUNSTEIN, THE LAWS OF FEAR: BEYOND THE PRECAUTIONARY PRINCIPLE (2005); Frank B. Cross, *Paradoxical Perils of the Precautionary Principle*, 53 WASH. & LEE L. REV. 860 (1996); Søren Holm & John Harris, *Precautionary Principle Stifles Discovery*, 400, 400 NATURE 398 (1999) (urging a precautionary approach to the precautionary principle).

regulatory intervention would be ineffectual. If we had such knowledge of causation, then we would not need the precautionary principle. If we lack knowledge of causation, then we cannot claim that the regulation urged in the name of prevention will prevent the harm at issue. Claiming that we must err on the side of safety implies that the proposed regulation would actually make us safer. Actions taken in ignorance and uncertainty create potential risks of harm from removal of targeted chemicals and the substitution of even less-well-tested alternatives.

There are some common beliefs that underlie the commitment to the precautionary principle. One belief is that real scientific conclusions are simply too difficult to attain, and we must accept hunches and conclusions that are plausible but lacking in sufficient evidence. Precautionists sometimes exaggerate the epistemic standards of science by despairing that we will never have "absolute certainty," and so must accept some ill-defined lesser standard. In practice, the precautionists seek to rely upon scientific evidence that would not meet even a modicum of sufficiency and validity.

Another tenet in the precautionist's belief system is that all or most cancers are caused by man-made chemicals. This tenet is held with an almost religious conviction devoid of support. As a religion, expressions of faith in the precautionary principle are protected by the Constitution, but the Establishment Clause should prevent the conviction from being foisted upon non-believers.

The precautionary principle is rarely invoked with candid acknowledgments of ignorance. By asserting scientific conclusions not backed by sufficient valid evidence, precautionists undermine the scientific enterprise. By misrepresenting preliminary, incomplete, and poorly substantiated claims as supported by science, precautionists abridge and pretermit the important process of further scientific exploration.

The central thesis of this *Monograph* is that IARC has been captured by precautionary principle ideologues. To be sure, at times, IARC officials have denied that their classification of carcinogens was based upon precautionary principles.[9] These denials, however, are not credible, and they are contradicted by other candid admissions.[10] Whether wittingly or not, the precautionary principle has come to control the IARC classification process at almost every turn.

This paper explores in some detail how the IARC classification system in its present form is driven by the precautionary principle. As we will see, in Section I, IARC ranks agents into epistemic categories (1, 2A, 2B, 3), only one of which could arguably be claimed to contain "known" carcinogens. The ranking system distorts the scientific and ordinary meaning of "probably" to mean little more than possibly.

IARC classifies cancer hazards irrespective of whether the agent is creating risks. The distinction supposedly has the precautionary goal of identifying hazards before they create risks. In Section II, this paper explores IARC's distinction between hazard and risk and finds the

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[9]Vincent James Cogliano, *The IARC Monographs: A Resource for Precaution and Prevention*, 64 OCCUP. ENVT'L MED. 572, 572 (2007) ("The IARC evaluations do not build in precaution.").

[10]Kurt Straif, *The Vital Contribution of Independent, Ethically Grounded Research to the Global Health Agenda*, 1 J. MORAL THEOL. 28, 31 (2021) (former head of IARC Monographs Program and its Section of Evidence Synthesis and Classification) ("Consistent with the precautionary principle, sufficient animal cancer evidence can be used to classify an agent as potentially carcinogenic to humans in the absence of adequate human data."). *See also* WHO Working Group, *Evaluation and Use of Epidemiological Evidence for Environmental Health Risk Assessment: WHO Guideline Document*, 10 ENVT'L HEALTH PERSP. 997 (2000) (noting that most of the working group members believed that the precautionary principle should be invoked in fashioning public health regulation in the face of uncertainty).

distinction unhelpful in the real world. IARC insists that its classifications are not precautionary because it is not engaged in risk regulation or risk characterization. The claim, however, is false. Not only does IARC cross its own line between risks and hazards, but by declaring "hazards" in the absence of quantifiable risks and circumstances that give rise to those risks, IARC encourages regulators, scientists, journalists, and the public to treat an IARC hazard as "risky" in all circumstances.

The embedding of the precautionary principle in the IARC classification process goes deeper yet. As we will see in Section III, IARC has propounded elaborate algorithms and checklists that, by design, inflate cancer classifications in several ways. Although the IARC classification process has evolved, it has lagged the standard of care for systematic reviews and for the synthesis of evidence for claimed health effects. In Section IV, this paper discusses IARC's substantial deviations from the standard of scientific care, which push working groups towards finding more carcinogens, with higher qualitative rankings.

In Section V, this paper turns to IARC's adoption of ideological and political definitions of conflicts of interest to exclude industry-funded scientists from the classification process. IARC allows scientists with conflicts, as long as the conflicts do not concern connections with manufacturing industry. IARC working groups allow participation by scientists who are aligned with environmental activist groups and plaintiffs' law firms, notwithstanding those scientists' ideological and positional biases, and their financial alignment with the lawsuit industry that manufactures mass litigation.

The paper identifies the founding and growth of one organization, the Collegium Ramazzini, as having a large influence on IARC's organizational politics and classification process. The Collegium is strongly committed to the precautionary principle, and its American fellows are frequent testifiers for claimants in cancer litigation in the United States. Collegium members have held important leadership positions at IARC, and they have populated many of its working groups.

If IARC were a true regulatory agency, it would be fair to say that it has been captured by Collegium Ramazzini members, who have bent IARC to their precautionary mission. Despite IARC's self-serving claims to transparency and integrity, this paper shows, in Section VI, that IARC has become secretive and driven by ideological concerns.

In Section VII, this paper explores some of the ramifications of, and confusions created by, IARC's carcinogen classifications. The case of glyphosate shows that IARC can readily turn a substance into a carcinogen, despite evidence to the contrary. IARC can also, through its monograph program, turn a substance into a "tortogen," inspiring thousands of litigation filings.

IARC classifications are also consequential in the world of science. As discussed in Section VIII, IARC classifications distort scientific discourse through IARC's ability to induce groupthink and fear of appearing adjacent to manufacturing industry in the form of disagreeing with IARC pronouncements. In the United States, IARC classifications also have the untoward consequence of mindless transformation into risk and warning regulations, notwithstanding IARC's protestations that its classifications do not address risk.

In Section X, this paper concludes with a discussion of the policy implications of IARC's failures. The United States was a key force in creating IARC and supporting its monograph program over the last half century. The felt needs for IARC's program are now largely gone, and can be better managed in the United States by scientific organizations, or perhaps by regulatory agencies with open meetings, public comment and debate, and peer review.

I. SEMANTIC CONFUSIONS IN IARC'S CLASSIFICIATION OF HUMAN CARCINOGENS

One of the projects that IARC has pursued since the early 1970s has been the preparation of monographs that evaluate the carcinogenicity of "agents" to *humans*. The agents are typically specific chemicals, but they can be biological organisms (bacteria or viruses), occupational settings (rubber factory workers, firefighting), or different sorts of radiation (ultra-violet light, radio waves, or ionizing radiation, such as X-rays).

From its beginning, IARC has classified human cancer hazards by the strength of the evidence that an agent causes cancer in humans. IARC's classification is based upon the overall strength of evidence in favor of human carcinogenicity. Its classifications do not tell us anything about potency and whether the agent is a strong or weak carcinogen. In other words, there can be strong evidence that an agent is a weak carcinogen. IARC scientists claim that its hazard evaluations turn on the use of "transparent criteria and descriptive terms."[11]

The four categories of levels of belief in carcinogenicity IARC uses are currently described in a key document called the *Preamble*, which IARC last revised in 2019.[12] The four "buckets"[13] for the IARC hazard classification system are:

> Group 1 – "carcinogenic to humans" (135 agents);
>
> Group 2A – "probably carcinogenic to humans" (95 agents)
>
> Group 2B – "possibly carcinogenic to humans" (323 agents)
>
> Group 3 – "not classifiable as to its carcinogenicity to humans" (500 agents)

Previously, IARC classifications defined a group 4 for agents not likely to cause cancer in humans. After decades of review, IARC had placed only a single agent in Group 4, caprolactam, a chemical used to make a variety of nylon. IARC could not find sufficient evidence for even water, air, basic foods, or vitamins to declare that they do not cause cancer in humans. In 2019, IARC moved caprolactam into group 3 (not classifiable), declared group 4 empty, and abandoned Group 4 as an option for future evaluations.

This recent change and others in IARC's classification scheme reflect the organization's implementation of the precautionary principle. This principle shifts the burden to manufacturers and regulators to show the safety of their products and actions, and welcomes labeling an agent as carcinogenic on an inconclusive evidentiary display.[14] IARC disallows conclusions that anything does not cause cancer, and it now requires everything be considered at least "indeterminate" with respect to its ability to cause cancer.

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[11]Jonathan Samet, *et al.*, "The IARC Monographs: Updated Procedures for Modern and Transparent Evidence Synthesis in Cancer Hazard Identification," 112 J. NAT'L CANCER INSTIT. djz169 (2020).

[12]IARC MONOGRAPHS ON THE IDENTIFICATION OF CARCINOGENIC HAZARDS TO HUMANS – PREAMBLE (2019) [cited herein as *Preamble*], https://perma.cc/LJE4-K7HH.

[13]The tallies are based upon IARC's website. The number of agents in each group is based upon IARC's reviews through June 2025; the numbers changed, and will continue to change, over time. *See IARC Monographs on the Identification of Carcinogenic Hazards to Humans*, https://perma.cc/HT9U-LM8P (last visited Nov. 1, 2025).

[14]*See, e.g.,* Bernard D. Goldstein, *The Precautionary Principle Also Applies to Public Health Actions*, 91 AM. J. PUB. HEALTH 1358 (2001).

The precautionary principle diverges dramatically from the U.S. legal system in which persons claiming to have been harmed must prove, among other things, the harmful nature of the defendants' conduct or products, by a preponderance of evidence. The precautionary principle also diverges from science, where scientific claims are established with ample evidence after severe testing. People can react to threats in many ways, whether real or potential. They may adopt the precautionary approach and choose to err on the side of caution. Or they may adopt the Evel Knievel approach and embrace even known risks. Both approaches are frequently on display in human behavior and in our policy choices; neither is particularly scientific. The phrase "precautionary principle" does not appear in the *Preamble*, but IARC officials have acknowledged the deep influence of the precautionary principle in the classification process.[15]

The Preamble defines some of the words used in IARC's classificatory scheme, in ways that are neither intuitive nor aligned with common usage. Indeed, as we will see, the classifications have frequently confused expert witnesses, courts, and regulators. Understanding IARC's contrived word usage in the context of its commitment to the precautionary principle helps explain, but fails to justify, its idiosyncratic definitions, algorithms, and checklists.

Agents placed in group 1 are, as noted, "carcinogenic to humans." In its *Preamble*, IARC does not refer to group 1 carcinogens as "known human carcinogens," although IARC publications refer to agents in group 1 as "known human carcinogens." Other organizations, such as the American Cancer Society, and regulatory agencies in the United States and elsewhere, identify agents from IARC's group 1 as "known human carcinogens." The implication of calling group 1 agents "known human carcinogens" is to distinguish them from agents in groups 2A, 2B, and 3. A semantic consequence of the distinction is that agents in the last three groups are all "*not known* to cause cancer." IARC may believe that agents in these other groups are potentially carcinogens when more and better evidence becomes available, but it cannot currently justify assertions of human carcinogenicity for group 2A, 2B, or 3 agents.

IARC does not spell out the epistemic implication of its classification system. Its silence seems coy, with an intent to exploit the natural tendency to inflate the importance of group 2A and 2B classifications. As we will see, there are other precautionary principle goals built into the IARC classification system, goals to err on the side of overstating carcinogenicity conclusions in the hopes of preventing human cancer, without sufficient evidence to support those hopes. Of course, overstating conclusions can only prevent cancer if IARC's overstatements are correct, which begs the questions facing IARC and the scientific community.

A. Non-Probable Probabilities

The classification immediately below Group 1 is Group 2A, for agents "probably carcinogenic to humans." Saying something probably causes cancer sounds impressive. No one would expect absolute certainty, so probably causes cancer would be sufficient for government work and for ordinary people to rely upon for their own safety. An IARC evaluation of an agent as belonging in group 2A, or "probably carcinogenic to humans," would appear to satisfy the legal system's requirement that an exposure to the agent of interest *more likely than not* causes the harm in question. Appearances can be deceiving, however, and IARC's use of "probably" is indeed deceptive.

[15]Kurt Straif, *supra* note 10, at 31 ("Consistent with the precautionary principle, sufficient animal cancer evidence can be used to classify an agent as potentially carcinogenic to humans in the absence of adequate human data.").

Buried in its *Preamble*, IARC weakly cautions against the deception by stating that its definition of "probably" is peculiar and out of line with scientific and ordinary usage, and especially with legal usage. The *Preamble* tells that in its idiosyncratic usage:

> The terms *probably carcinogenic* and *possibly carcinogenic* have no quantitative significance and are used as descriptors of different strengths of evidence of carcinogenicity in humans; *probably carcinogenic* signifies a greater strength of evidence than *possibly carcinogenic*.[16]

So probably does not mean probably, but something more like very possible.

IARC's suggestion that its usage of probability has no quantitative meaning is puzzling. In science, mathematics, and in ordinary language, probability is a quantitative concept on a continuous scale. Mathematically, probability is scaled from zero to one, or as a percentage from 0% to 100%. In Bayesian analyses of empirical scientific evidence, 0 and 1 are not available. The exclusion of 0 and 1 has come to be known as "Cromwell's Rule." The extreme absolutes of never and always are prohibited because if either were our starting point, no amount of new evidence could ever change our assessment of the probability under Bayes Rule.[17] In science, even "known" causes can never be absolutely certain, or 100%; they are probable, greater than 50%. Group 1 human carcinogens would thus be more accurately described in scientific parlance as probable human carcinogens. IARC's epistemic inflation is part of its precautionary agenda.

Under IARC's peculiar definition of probably, group 2A classifications are consistent with quantitative probabilities less than 0.5 (or 50%). IARC's inflationary semantic fiat becomes the delight of fear mongers and tort lawyers.[18] A working group could judge the probability of a substance or a process to be carcinogenic to humans to be greater than zero, but no more than say ten percent, and still vote for a 2A classification, in keeping with the IARC *Preamble*. This low probability threshold for a 2A classification converts judgments of "probably carcinogenic" into nothing more than precautionary prescriptions, rendered when the most realistic assessment requires acknowledging ignorance or substantial uncertainty, or embracing lack of causality. There is thus a practical certainty, close to but not quite 100%, that a 2A classification will confuse judges and juries, the media, and the scientific community. Their confusion arguably is stoked by writers such as Carl Cranor, fellow of the Collegium Ramazzini and expert witness for the lawsuit industry, who has written a law review article that specifically propagates the falsehood that IARC's use of "probably carcinogenic" translates into the law's "more likely than not" burden of proof.[19]

The inevitability of confusion caused by IARC's peculiar definition of probably should lead courts to exclude expert witness testimony that an agent causes human cancer because IARC says it "probably" does so. Indeed, the IARC classifications owe so much to semantic legerdemain that

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[16] *Preamble* at 31 (emphasis in original).

[17] DENNIS VICTOR LINDLEY, THE BAYESIAN APPROACH TO STATISTICS 29-32 (1980), https://perma.cc/LLE3-A5HW, last visited Nov. 1, 2025.

[18] *See* David Hackett Fischer, *Fallacies of Semantical Distortion*, chap. 10, in HISTORIANS' FALLACIES: TOWARD A LOGIC OF HISTORICAL THOUGHT (1970) (describing how hyperbole leads authors into semantic distortions such as using "certainly" to mean "probably," "probably" to mean "possibly," and "possibly" to mean "conceivably").

[19] Carl F. Cranor, Milward v. Acuity Specialty Products: *Advances in General Causation Testimony in Toxic Tort Litigation*, 3 WAKE FOREST J. L. & POL'Y 105, 124 (2013).

basic evidence-law principles should exclude courtroom references to the IARC classification.[20] Such documentary evidence and testimony would confuse and mislead the jury and be more prejudicial than probative.

In personal injury litigation against the manufacturer of RoundUp® (glyphosate), Alfred Neugut, an expert witness for claimants with qualifications in epidemiology and oncology, brazenly misrepresented the meaning of an IARC 2A classification. Dr. Neugut opined that an IARC conclusion that an agent is a group 2A probable human carcinogen means "as a practical matter, that the group reached this conclusion with 70-90% certainty." The trial court saw through the ruse and noted that IARC itself states that the term probably has no quantitative significance in its carcinogen classifications, and that IARC's peculiar usage is contrary to ordinary meaning and the legal framing of the burden of proof as "more likely than not."[21]

Notwithstanding its recognition of how misleading IARC's linguistic usage was, the trial court ultimately allowed the plaintiff's counsel and his expert witnesses to refer to IARC's classification of glyphosate as "probably carcinogenic" to address plaintiff's complaint of prejudice from bifurcating the issues at trial. On appeal, the Ninth Circuit affirmed the district court's admission of the IARC group 2A probable classification as not an abuse of discretion.[22] The appellate court acknowledged that Monsanto had challenged the admission of the 2A classification as irrelevant and prejudicial, but the court never mentioned that the classification does not mean what claimants insinuated it to mean.

In a more recent case decided in 2024, a federal trial court excluded an expert witness, Dr. Dipak Panigrahy, who had plagiarized substantially from an IARC monograph on the chemical at issue, and whose report failed to evidence a reliable methodology. Although Dr. Panigraphy copied large segments of an IARC monograph verbatim, when it came to the meaning of "probable human carcinogen," he improvised his own interpretation, and testified that the phrase means "more likely than not, that this chemical will cause cancer in humans."[23] Dr. Panigraphy's errant misinterpretation of the meaning of an IARC group 2A classification is common place among expert witnesses, regulators, and especially journalists.

B. Everything Is Possible

Group 2B classifications reflect an IARC conclusion that an agent is "possibly carcinogenic." In 2023, IARC announced that a working group had concluded aspartame, an artificial sugar substitute, was "possibly carcinogenic."[24] Such an evaluation, however, tells us nothing. If there were no studies at all of an agent, the agent could be said to be possibly carcinogenic. If there were inconsistent studies, even if the better designed studies were exculpatory, scientists might well continue to say that the agent of interest was possibly carcinogenic. The 2B classification does not tell us anything because everything is possible until there is sufficient evidence to inculpate or exculpate it from causing cancer in humans. For IARC, the process of classifying hazards is only one of inculpation; it has discontinued group 4, the only exculpatory hazard classification it previously permitted. Now everything not known to cause cancer in humans is at least possibly

[20]Fed. R. Evid. 403.

[21]*In re Roundup Prod. Liab. Litig.*, 390 F. Supp. 3d 1102, 1108, 1144-45 (N.D. Cal. 2018) (Chhabria, J.).

[22]*Hardeman v. Monsanto Co.*, 997 F.3d 941, 954 (9th Cir. 2021).

[23]*Henderson v. Lockheed Martin Corp.*, 723 F. Supp. 3d 1147, 1151-1152 (M.D. Fla. 2024).

[24]Elio Riboli, *et al.*, *Carcinogenicity of aspartame, methyleugenol, and isoeugenol*, 24 LANCET ONCOL. P848-850 (2023).

carcinogenic to humans. This aspect of IARC's classification is generally unhelpful, but it is not even the most problematic part of the IARC approach.

C. Classification of Agents of Indeterminate Carcinogenicity

Indeterminate carcinogenicity, or group 3, is how IARC classifies agents that cannot be squeezed into groups 1, 2A, or 2B. In one locution, IARC says that indeterminate carcinogens are ones that are not classifiable as to their carcinogenicity. IARC, however, goes ahead and classifies the non-classifiable as group 3. Once again IARC's bias surfaces by classifying agents that are generally regarded as safe and non-carcinogenic as indeterminate, which sounds very much like and is conceptually inseparable from "possibly." Indeed, even when there is strong evidence that an agent (such as saccharin) causes cancer in rats, but not in humans, and the mechanism of carcinogenicity in rats is irrelevant to humans, IARC places the human non-carcinogen in the indeterminate group.

The authors of monographs on agents for which evidence suggests lack of carcinogenicity in humans and in experimental animals are permitted to add "a sentence" to the monograph to describe the agent as having been well studied without evidence of carcinogenicity. The bottom-line classification, however, remains indeterminate, group 3, without qualification. IARC's confusing definitions obscure the reality that group 3 agents are typically ones for which the evidence is insufficient, completely lacking, or even supportive of *lack of* carcinogenicity.

The IARC classifications are based on the level of overall support for calling an agent a carcinogen. A group 1 classification says nothing about the comparative potency of an agent. If an agent were known to cause an excess cancer risk of one excess cancer for every million people exposed, it would be in the same category as another agent that caused cancer in half the people exposed. Potency is typically expressed as a measure of the magnitude of risk from a given exposure. An IARC classification, even a group 1 classification, does not mean that people exposed to the agent will have any risk at all, or that any risk would be substantial.

II. IT'S A HAZARD, NOT A RISK

Risk is a probability that some event will happen, usually an untoward event. As a probability, risk is a quantitative concept, although in many instances we are uncertain about the precise quantity, and we can only estimate. The risk of many cancers is a contentious issue. There appears to be a baseline risk of most types of cancer that is expected because of age, sex, and genetics. In most instances, we are concerned about preventable increased risks of cancer that may result from exposure to an agent, or from lifestyle choices. Labeling an agent or exposure as carcinogenic may suggest that exposure does and will cause cancer in everyone exposed. There are no IARC-classified agents that cause cancer in everyone exposed. When there is a risk for a particular exposure circumstance, the risk is a risk greater than what otherwise would have been experienced by exposed persons had they not been exposed.

When IARC embarked upon its program of evaluating potential human carcinogens in 1971, its resolution was framed in terms of "carcinogenic risks of chemicals to man." Over time, IARC reframed its program to evaluate "hazards" of chemicals and other agents.[25] The *Preamble*, which contains the general principles for IARC's classifications, acknowledges that identifying a cause of human cancer is merely a first step in the process of eliminating preventable cancers. In what could

[25] *Preamble* at 1.

be the most overlooked aspect of IARC's program, the *Preamble* tells us:

> A cancer *hazard* is an agent that is capable of causing cancer, whereas a cancer *risk* is an estimate of the probability that cancer will occur given some level of exposure to a cancer hazard. The *Monographs* assess the strength of evidence that an agent is a cancer hazard. ***The distinction between hazard and risk is fundamental.*** The *Monographs* identify cancer hazards even when risks appear to be low in some exposure scenarios. This is because the exposure may be widespread at low levels, and because exposure levels in many populations are not known or documented.[26]

This attempted explanation fails, in ways explored below, but it does reveal some important aspects of IARC's project. The *Preamble* suggests that IARC focuses on simply labeling an agent as a cancer hazard because there may be widespread low exposures, with attendant low risks. What the *Preamble* fails to note is that for some exposures, especially "low" exposures, the risks may be zero, if there are thresholds of exposure below which an agent does not cause cancer. Similarly, IARC omits any reference to how there may be exposure levels to an agent that carries a risk of benefit. Instead, IARC shrugs and states that it avoids "extrapolating exposure-response relationship beyond the available data," with the knowledge that by identifying a carcinogenic hazard, many people, journalists, legislators, and even regulators, will assume that there is no safe level of exposure to carcinogens.[27] Indeed, by suggesting that the risk may be low, without stating clearly that the risk may be zero or even beneficial, IARC actively fuels non-validated no-threshold assumptions. The IARC classifications' failure to address risk thresholds or potential benefits from exposure is in line with the precautionary philosophy that pervades the *Preamble*.

In IARC parlance, cancer risk requires a finding that an agent is a cancer hazard, that it can cause cancer, but identifying a cancer hazard without more information does not equate to any cancer risk. The assessment and quantification of cancer risk typically requires that we know the route of exposure, the actual conditions under which the agent is used, the level of exposure, peak exposure, and cumulative exposure to humans, the metabolic fate and distribution of the agent in humans, and toxico-kinetics of the agent (rate at which the agent is metabolized or cleared from the human body), the exposure-outcome gradient, and the existence of thresholds of exposure for any risk. The IARC classifications provide little to no information about the sorts of data needed to determine and characterize risk. To the uninformed public and media, hazard sounds a lot like risk, and the two concepts would naturally be confused.

Rather than face the full range of risk scenarios, the *Preamble* asserts that identifying a cancer hazard "should trigger some action to protect public health, either directly as a result of the hazard identification or through the conduct of a risk assessments."[28] This prescriptive language,

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[26] *Preamble* at 2 (bolded emphasis added). The hazard-risk distinction accords with common technical usage. *See, e.g.,* Miquel Porta, *et al.*, eds. A DICTIONARY OF EPIDEMIOLOGY 128 (6th ed. 2014) (defining "hazard" as "[t]he inherent capability of a natural or human-made agent or process to adversely affect human life, health, property, or activity, with the potential to cause a disease, epidemic, accident, or disaster." *Id.* at 250 (defining "risk" as "[t]he probability of an adverse or beneficial event in a defined population over a specified time interval. In epidemiology and in clinical research it is commonly measured through the cumulative incidence and the incidence proportion").

[27] *Preamble* at 2.

[28] *Preamble* at 3. IARC does exhibit some humility in refraining from recommendations of specific regulatory or legislative approaches, but with the knowledge that there will be governmental agencies that possess all the epistemic arrogance needed to convert IARC classifications into precautionary principle inspired regulations.

devoid of scientific meaning, is at the heart of the precautionary mission of IARC's classifications, and reveals its unexamined premise that its classifications alone can and should guide governmental promulgation of preventive measures.

IARC's precautionary mission, and its insistence that identifying "hazards" will advance cancer prevention, unravels under close inspection. Ultraviolet light (wavelength 100-400 nanometers) is an IARC group 1 carcinogen, even though sunlight contains ultraviolet radiation (10-400 nanometers). Most adults understand that too much sunlight can be harmful in the short- and long-term, even though a life without sunlight might not be worth living, and would lead to a deficiency of vitamin D, essential for human health.[29]

Putting aside culture war battles over "toxic masculinity," all adults, male and female, produce testosterone. For many, life without testosterone (an androgenic anabolic steroid) would be diminished in important ways. For IARC, testosterone, whether produced naturally or chemically, is "probably" carcinogenic to humans.

IARC places formaldehyde in group 1, which would make some people fear any exposure as creating a risk of cancer, even though normal healthy human beings produce formaldehyde in the process of producing essential ingredients of life, such as thymidine for DNA, and certain amino acids for protein. Adults produce over an ounce of formaldehyde a day in their bodies. Humans have a normal metabolism of formaldehyde, with their livers metabolizing over a gram an hour, with normal excretion of metabolites. Formaldehyde is present in vegetables, fruits, and meats we ingest. The U.S. Environmental Protection Agency has determined that a lifetime of exposure to formaldehyde in drinking water at the level of 1 milligram per liter is not expected to cause harmful health outcomes.[30] All this is not to contradict that formaldehyde may cause some cancers under *some* exposure circumstances (dose levels and manner and routes of exposure), but conveying information that formaldehyde is a group 1 carcinogen is neither helpful nor informative.

Similarly, IARC classifies ethylene oxide as a group 1 carcinogen, even though this chemical is made and exhaled normally by human beings in their bodies.[31] Encapsulating people or putting warning signs on people's faces will not really help prevent cancer.

IARC is not always consistent in maintaining its own distinction between hazard and risk. Under IARC's approach to the risk-hazard distinction, exposure levels should play no role in the classification process. IARC classifies (ethyl) alcohol beverages in group 1, but to date, it has not proclaimed sacramental wine as a human cancer hazard. The human body makes small amounts of ethyl alcohol,[32] and the agent is present in most fruit and fruit juices. If exposure amount and circumstance are irrelevant to hazard classification, then the IARC group 1 classification applies across the board to humans, fruits, and religious imbibing. The hazard is everywhere, which makes the pronouncement rather useless.

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[29]For an up-to-date score card of IARC's classifications, *see* "List of classifications by cancer sites with sufficient or limited evidence in humans, IARC Monographs Volumes 1–139" (last revised Oct. 24, 2025), https://perma.cc/Z584-TEGD.

[30]Agency for Toxic Substances and Disease Registry, *ToxFAQs™ for Formaldehyde* (May 2015), https://tinyurl.com/y43uc5bd, last visited Nov. 4, 2025.

[31]EPA, *Additional Questions about Ethylene Oxide (EtO)* (Jan. 14, 2025), https://www.epa.gov/hazardous-air-pollutants-ethylene-oxide/additional-questions-about-ethylene-oxide-eto.

[32]Yuri M. Ostrovsky, *Endogenous ethanol--its metabolic, behavioral and biomedical significance*, 3 ALCOHOL 239 (1986).

The agency has pulled its classification punch when not doing so would make its classification efforts look silly. Such an example of hedging occurred when an IARC working group classified crystalline silica as a group 1 human carcinogen. About 12% of the earth's crust is crystalline silica (SiO_2). Silica is the second most common mineral, and it is a constituent mineral of most rocks, and omnipresent in soil. Beach sand in places can be 90% crystalline silica or more. Although crystalline silica is in group 1, the supporting IARC monograph indicates a specific exposure situation for the evaluation by stating that its evaluation pertains only to inhalational "occupational exposures."[33] When IARC evaluated the hazard of coal exposure, it placed coal dust in group 3 (indeterminate), even though coal dust contains crystalline silica.[34] Notwithstanding the IARC silica monograph's qualification about risk not arising in all occupational settings, the group 1 classification has resulted in many bags of play sand carrying cancer warnings. Most environmental and consumer regulatory agencies, however, have not, to date, converted beaches or Mr. Turtle sandboxes into Superfund sites. In 2012, however, IARC embraced the silliness, and in an updated evaluation of crystalline silica, reasserted its group 1 classification, but without any qualification to limit the "hazard" to certain occupational settings.[35]

Another glaring inconsistency in principles and violation of the hazard-risk distinction can be found in the IARC evaluation of coffee. In 2018, IARC classified coffee in group 3 (indeterminate). The problem for IARC's program results when we consider that every drop of coffee contains a chemical known as acrylamide, which IARC classifies as a group 2A "probable human carcinogen." Opportunistic lawyers have attempted to exploit the inconsistency by suing coffee vendors for failing to issue cancer warnings with their coffee. For IARC, not only is the acrylamide component of coffee a "probable" human carcinogen, but so is the water above the temperature of 149°F (65°C).[36] Coffee is brewed at 195°F and often served hot enough to put the hot beverage right back into IARC group 2A. It may take an IARC metaphysician to sort out the acrylamide and the hot water from the coffee.

Another recent striking departure from IARC's claim to evaluate hazards, but not risks, came from its working group that evaluated red meat and processed meat. The group placed processed meat into group 1, and red meat into group 2A. The IARC working group deviated from the *Preamble* by embracing a meta-analysis of observational studies of colorectal cancer, which reported a 17% increased risk of colorectal cancer from daily ingestion of 100 grams of red meat, and 18% increased risk of colorectal cancer from daily ingestion of 50 grams of processed meat. The IARC working group did not simply include the meta-analysis results in the lengthy monograph; it included the risk assessment in its press release and in its short summary of the monograph published in *Lancet Oncology*, to ensure that the media and advocacy groups picked up IARC's conclusions about estimated risks.[37]

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[33]IARC Monograph 68, at 41 (1997) ("For these reasons, the Working Group therefore concluded that overall, the epidemiological findings support increased lung cancer risks from inhaled crystalline silica (quartz and cristobalite) resulting from occupational exposure.").

[34]IARC MONOGRAPH 68, at 337 (1997).

[35]*See* IARC MONOGRAPH VOL. 100C, CARCINOGENIC RISKS TO HUMANS OF ARSENIC, METALS, FIBRES, AND DUSTS (2012).

[36] Dana Loomis, *et al.*, "Carcinogenicity of drinking coffee, mate, and very hot beverages," 17 LANCET ONCOL. 877 (2016).

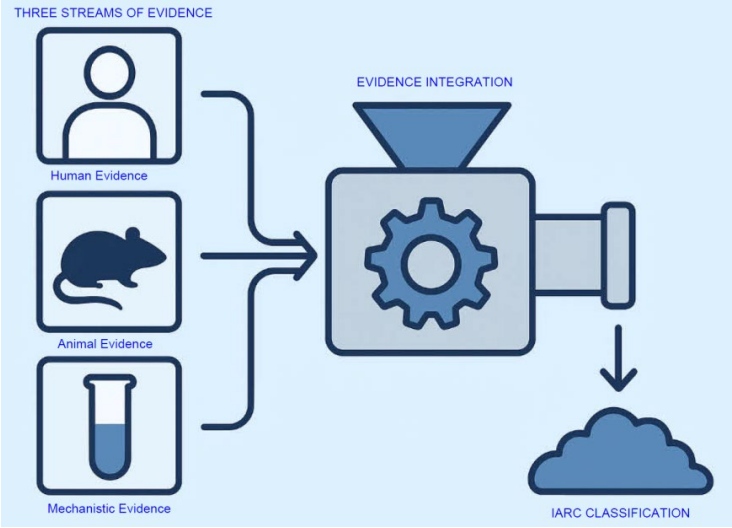
[37] Véronique Bouvard, *et al.*, *Carcinogenicity of consumption of red and processed meat*, 16 LANCET ONCOL. 1599 (2015).

Although IARC's classification of a carcinogen cannot inform us whether specific exposure circumstances increase risks of cancer, the classification can and does result in *tortogenesis*. IARC classifications have been known to spark mass tort claims in the United States, and elsewhere. Careless expert witnesses have been known to misrepresent IARC hazard classifications as risk determinations. As noted above, in litigation against makers of glyphosate, plaintiffs' expert witness Alfred Neugut sought to characterize IARC's classification as more than a hazard determination. Although the trial court lost its way on many important issues, it did recognize and call out Neugut's misrepresentation.[38] Courts must adjudicate, whether without the allegedly tortious exposure to the agent, the claimant would not have developed the complained of harm. Invoking IARC's classification of that agent as a hazard, whether group 1, 2A, 2B, or 3, fails to answer the important litigation questions at issue.

III. THE "IARC'Y" OF EVIDENCE: ALGORITHMS AND CHECKLISTS

A. Classification on Three Streams of Evidence: More Word Games

The *Preamble* describes how working groups should classify agents based upon evidence that is found in three "streams" or types of studies. The streams are distinct kinds of evidence found in studies of cancer in (i) humans, (ii) in experimental animals (typically rodents), and (iii) in mechanistic studies. Human evidence usually comes from the results of observational epidemiologic studies, but, in rare cases, may be found in randomized controlled trials.[39] Animal evidence typically comes from the results of lifetime exposure in laboratory animals. Mechanistic evidence comes from various experiments in cells or tissues outside the bodies of humans or animals.



IARC provides a decision tree of sorts for what kinds of evidence, and what evidential strengths, are needed to place an agent in one of the four classification groups (1, 2A, 2B, or 3). The IARC decision process is represented schematically to the right.

IARC working sub-groups are charged with evaluating and characterizing human (epidemiologic) evidence and animal (toxicologic) evidence in one of four buckets: (a) sufficient, (b) limited, (c) inadequate, or (d) evidence suggesting lack of carcinogenicity. Although those labels are subjective, IARC attempts, without much success, to specify what is required for assigning each adjective label to the human and the animal data.

IARC directs a similar exercise for a subgroup charged with evaluating the mechanistic data as strong, limited, or inadequate. IARC does not explain why mechanistic data has a strong but no sufficient category, and the human and animal data have no strong categories. The *Preamble* then specifies what levels of support from each of the three streams of evidence is required for placing

[38] *In re Roundup Prod. Liab. Litig.*, 390 F. Supp. 3d 1102, 1108, 1144-45 (N.D. Cal. 2018) (Chhabria, J.).

[39] *Preamble* at 16.

an agent in the appropriate classification group.[40]

For example, for an agent to be classified as "carcinogenic to humans (group 1)," without qualification, the working group must find only sufficient human evidence, in which case evidence from animals or mechanistic studies is unnecessary. There is no suggestion that any aspect of a Group I evaluation requires apodictic, conclusive, or even "definitive" evidence, which undermines the interpretation of group 1 as including "known" human carcinogens. A working group can also reach a group 1 classification with even more clearly speculative evidence in the form of insufficient human evidence (limited or even inadequate), if the group finds sufficient animal evidence along with strong mechanistic data.

The concept of sufficient is not only subjective; it is vague. If we think of grading schemes that we experienced in school, we might think of sufficient as adequate, or a grade C, not an A or a B. Perhaps limited is a C-, or a D; and inadequate is an F. Data that are merely sufficient certainly seem different from strong, robust, severely tested, or conclusive data.

Curiously, the "strong" descriptor, which is available for mechanistic data, is not available for human or animal evidence in the *Preamble*. There certainly seems to be an evidential gap between strong and sufficient, but this gap is obscured by the *Preamble*'s decision tree that omits applying the strong qualifier to human or animal evidence.

Strong evidence suggests a level of conclusiveness and robustness that is missing from evidence that is merely sufficient or just over the line for decision. For instance, the human epidemiologic evidence for smoking and lung cancer, or for occupational exposure to crocidolite asbestos and mesothelioma, is much more than merely sufficient for a causal conclusion. For those clearly causal associations with smoking or occupational exposure to crocidolite, there is virtually no room for reasonable resistance to the force of the causal inference. Of course, "strong," in IARC parlance of evidential strength, does not refer to potency, and so the magnitude of the risks created by smoking or crocidolite asbestos is not germane. What makes the epidemiologic evidence strong, and more than merely sufficient, is the consistency, robustness, and certitude of the detected associations. (The very large relative risks are, of course, a factor in ensuring the consistency of the findings in epidemiologic studies for these two known carcinogens, crocidolite and tobacco smoking.) This qualitative difference in strength is obscured by the grading scheme for human evidence, which by omitting strong as a grading choice, smacks of grade inflation by pushing the merely sufficient into the highest-grade category.

Also obscured by the *Preamble*'s grading and verbiage scheme is the unarticulated implication that either limited or inadequate evidence for an evidence stream must mean that the evidence is "insufficient." The limited category turns out to be a euphemism that allows working groups to avoid labelling evidence as insufficient even when labeling an agent as a human carcinogen.

B. Human Evidence

1. *Sufficient Evidence*

IARC attempts to define sufficient in the *Preamble* for each of the human and animal streams of evidence. For epidemiologic studies, "sufficient" evidence is circularly defined as obtaining when "[a] causal association between exposure to the agent and human cancer has been

[40] *Preamble* at 37, Table 4.

established."[41] The *Preamble* provides some specifics, in the context of human evidence, by indicating that sufficient evidence of carcinogenicity requires a positive association between exposure to the agent and cancer "in the body of evidence." Sufficient evidence also requires that the evaluators rule out chance, bias, and confounding "with reasonable confidence."[42]

Even this minimal specificity seems to contain something of a trick in requiring the finding of "a positive association" *in* the body of studies. The phraseology leaves open whether the working group can deem the requirement satisfied by a single study, or a subset that is smaller than the set of all available studies. If the working group has this discretion, then we might well ask what the criteria or guidelines for the exercise of the discretion are. The choice of the preposition "*in*" certainly appears to allow selectivity, discretion, and subjectivity in the choice of which studies count for ascertaining a positive association.

Had the *Preamble* specified that the evaluators must find the positive association *across* the entire body of relevant studies, or consistently *throughout* the entire body of studies, the working group's discretion would have been significantly curtailed, and IARC would be required to pre-specify what criteria could be used to exclude studies from consideration of the entire body of studies. There are no principles or criteria for this selection in the *Preamble*.

The peculiar phrasing in the *Preamble*, devoid of criteria or guidance for which studies count for finding a positive association, means that the working group will have substantial latitude to make *post hoc* judgments in assessing whether there is a positive association. The working group is freed from having to find the association in all the studies, or in a meta-analysis of all the studies (assuming that a meta-analysis can or should be done). The *Preamble* thus opens the door, with a rather conspicuous welcome sign, to "cherry picking," in which a positive association can be embraced from some studies, and the absence of an association in other studies can be ignored, on the road to a forced conclusion of sufficient or limited human evidence.

Further, the definition of sufficient human evidence does not seem to require that the cancer end point be the same in or across studies for a finding of sufficiency. Cancer is not a single disease,[43] and many cohort studies look at risk ratios for dozens or hundreds of different cancers.

This failure to specify a consistent type of cancer in the body of studies to meet the requirement of sufficiency does not appear to be accidental. Even though IARC abandoned a group 4 for agents probably not carcinogenic, the *Preamble* details what is required for a working group to find and report "evidence suggesting lack of carcinogenicity."[44] What is telling is that the specifications for finding evidence of a "lack of carcinogenicity" are longer than the definitions for sufficient, limited, and inadequate evidence combined. Not only is the description of the evidence needed for lack of carcinogenicity more detailed, but it is also more demanding than the evidence required for concluding that the evidence supports carcinogenicity.

For instance, for lack of carcinogenicity, the *Preamble* requires that there must be specificity of the type of cancer by organ or tissue involved. There is no similar requirement of organ and

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[41] *Preamble* at 31.

[42] *Preamble* at 31. Buried elsewhere in the *Preamble* is the qualification that ruling out bias, chance, and confounding includes comprehensively evaluating study quality, an important qualification worthy of including in the definition of sufficient evidence. *Preamble* at 19.

[43] Joel S. Brown, *et al.*, *Updating the Definition of Cancer*, 21 MOL. CANCER RES. 1142 (2023).

[44] *Preamble* at 22, 32.

tissue specificity for finding sufficient evidence in favor of carcinogenicity in human studies. Similarly, the *Preamble* dictates that all studies must be methodologically valid and consistent in failing to show an association (with narrow confidence intervals). A single epidemiologic study that shows a dose-response between agent and a cancer outcome nullifies the body of evidence as showing lack of carcinogenicity. There is no such absolute consistency requirement for dose-response in studies to support a conclusion in favor of carcinogenicity. The *Preamble* limits comment on evidence of lack of carcinogenicity to the precise type of cancer in the study, to the dose and duration of exposure, and observed timing after exposure. In other words, a working group cannot comment about the lack of carcinogenicity without engaging in a fairly detailed risk assessment, whereas for endorsing an agent's carcinogenicity, the group need not provide any similar level of analysis or detail.

IARC obviously imposes a higher standard of evidence for exculpatory conclusions than it does for inculpatory conclusions because the agency prefers inculpatory conclusions. The asymmetry in standards for evidence for and against carcinogenicity reflects an ingrained bias in favor of precaution, and prejudice against exculpatory or even neutral statements. There is no scientific basis proffered for the asymmetrical standards; IARC is advancing *policy* choices, not *scientific* criteria.

Although sufficient epidemiologic evidence of carcinogenicity is the primary decision path to a group 1 classification, IARC altered its *Preamble*, in 2019, to permit group 1 classifications without sufficient epidemiologic evidence, when there are sufficient animal evidence and certain types of strong mechanistic evidence. As the IARC procedures now stand, some group 1 classifications, and ***all*** group 2A, 2B, and 3 classifications are based upon insufficient evidence of carcinogenicity in humans.

2. Limited Evidence

What exactly is limited epidemiologic evidence, aside from being not sufficient? IARC gives an incoherent, unscientific definition that ascribes an inflated weight and understanding to "limited evidence of carcinogenicity," by telling us that

> A causal interpretation of the positive association observed in the body of evidence on exposure to the agent and cancer is credible, *but chance, bias, or confounding could not be ruled out with reasonable confidence*."[45]

This definition is plainly oxymoronic. One the one hand, the evidence must be credible, but the evidence can be invalid due to random or systematic error. But associations for which chance, bias, and confounding remain as potential explanations are not credible causal association in the first place. In other words, for IARC, limited evidence of carcinogenicity requires a credible causal interpretation of a positive association, which no serious scientist would or should accept as a credible interpretation.

In epidemiology, an association that is not the result of chance, bias, or confounding is the starting point for causal analysis. Without credibly excluding sources of systematic and random error, there really is no reason at all to consider a causal interpretation. IARC ignores this obvious point and thus renders its "limited" grade incoherent.

[45] *Preamble* at 31 (emphasis added).

The *Preamble* cites the famous paper by Sir Austin Bradford Hill, who memorialized his after-dinner speech in which he celebrated his success in using observational epidemiology to establish that tobacco smoking causes lung cancer. Sir Austin outlined nine considerations for determining whether an association was a causal association, but only after "[o]ur observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance."[46]

The *Preamble* does incorporate Sir Austin's predicate of excluding systematic and random error into IARC's definitional criteria for sufficient evidence, and in a perversely confused way into its definition of limited evidence (perverse because the association would by definition not be "clear-cut," and the sources of random and systematic error are not ruled out with reasonable confidence). Remarkably, the *Preamble* omits the nine considerations Sir Austin urged for consideration after the predicate is established.[47] To be fair, some of the nine considerations are discussed elsewhere in the *Preamble*, including in the discussion of the other two streams of evidence for non-human (animal and mechanistic) evidence. And the *Preamble* does parse the relevant considerations for reviewing epidemiologic studies for relevancy and quality,[48] but when IARC defines its qualitative categories (sufficient, limited, inadequate, and lack of carcinogenicity), the document fails to explain how the Bradford Hill considerations, which include strength of association, consistency, and dose-response, should be evaluated to support the working group's conclusion.[49]

3. *Inadequate Evidence*

Inadequate human evidence results from the absence of data or from data with "insufficient quality, consistency, or statistical precision." IARC does not say whether insufficient statistical precision is the same thing as failing to rule out chance, or what the criteria are for judging statistical precision. The *Preamble* suggests that the poor quality of available studies, inconsistent results among studies, or inconclusive studies determine an inadequate grade. These suggestions leave inadequate and limited epidemiologic datasets looking very similar. There is no discussion of how the absence of consistency in an inadequate data set relates to the requirement of finding consistency *in* (but not across or throughout) the body of studies that merits a sufficient assessment. Perhaps more problematic, however, the *Preamble* fails to reference any of the existing scales for evaluating the quality of studies; nor does it detail how studies with confounding and bias, which warrant a limited score, differ from studies that are otherwise inconclusive. It would seem inescapable that studies for which bias and confounding could not be ruled out must be inconclusive, and thus the limited score for the stream of human studies would collapse into the inadequate score.

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[46]Austin Bradford Hill, *The Environment and Disease: Association or Causation?* 58 PROC. ROYAL SOC'Y MED. 295, 295 (1965).

[47]Hill's considerations are frequently misrepresented as criteria. The necessary criterion is the perfectly clear-cut association (excluding bias and confounding), beyond the play of chance (and statistically significant), which is the required predicate for studying the considerations. The nine considerations identified by Sir Austin were (1) the strength (size) of the association, (2) the consistency of the association across studies, of varying designs, done by different researchers, (3) the specificity of the outcome for the agent, (4) the temporality of the putative effect occurring after the putative cause, (5) the dose-response relationship, (6) the biological plausibility of the association as shown by mechanism, (7) the coherence between epidemiologic and laboratory findings, (8) the experimental evidence, and (9) the analogies or similarities that may explain the observed association.

[48]*Preamble* at 20-22, § B.2(e).

[49]*Preamble* at 31-32.

IARC implicitly acknowledges that human evidence is the most important type of evidence for classifying human carcinogenicity. On human evidence alone, IARC can put an agent into group 1. The agency thus seems to concede that "[t]he proper study of Mankind is Man."[50] Of course, the human evidence need not be strong for group 1, only sufficient. As for animal evidence, we will see that IARC sometimes conflates humans and rats to advance precautionary principle reasoning.

For none of the other categories (2A, 2B, or 3) does IARC require sufficient human evidence. In other words, if an agent is not in group 1, then the human or epidemiological evidence will be insufficient (limited or inadequate). IARC can place an agent in group 2A (that is, probable human carcinogen, where probable does not mean more likely than not) with only limited (and thus insufficient) human evidence if a working group finds that the animal evidence is sufficient. Group 2B, which in any event is only about possibility, can be reached with insufficient human evidence, depending upon what can be squeezed out of the data on animals and mechanistic studies.

The *Preamble* acknowledges that sufficient animal evidence of carcinogenicity does not mean that there will be human carcinogenicity, but only that human cancer is plausible. Nonetheless, IARC is willing to go much further with sufficient animal evidence in the service of the precautionary principle. If no strong exculpatory mechanistic evidence is available to show that the animal species mechanism does *not* operate in humans, then IARC considers the animal carcinogen to be a "potential carcinogenic hazard to humans." And IARC is willing to spread that plausibility around to any type of human cancer regardless of the type of tumor seen in the animal species.[51]

C. Definitions for Levels of Animal Evidence

For animal toxicological evidence, as for human evidence, IARC creates four levels of supportiveness: sufficient, limited, or inadequate evidence, and evidence suggesting the lack of carcinogenicity. As noted, there is no opportunity in IARC's scheme of things to rate animal evidence of carcinogenicity as strong. Sufficient animal evidence is based upon laboratory animal experiments that find an excess incidence of malignant tumors, or of both malignant and benign tumors. Remarkably, the *Preamble* provides some guidance on the selection of statistical models for evaluating animal studies, but it does not indicate that the disparity in tumors must be statistically significant at any pre-specified level. Because lifetime rodent studies usually look for tumors in dozens of organs, the risk that some results will be driven by random error solely from multiple testing is substantial. The *Preamble* discusses these and other important considerations, but in its section on grading the overall evidence from animal studies, there is no requirement or guidance on how to control statistical error from the multiplicity of testing, or how to approach any number of difficult statistical issues that arise in chronic toxicology experiments.[52]

Similarly, the *Preamble* provides some discussion about when and whether benign tumors will be relevant to a causal judgment for animal studies. The *Preamble* does specify that for the animal evidence to be sufficient, the increased incidence of tumors (malignant and/or benign) must

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[50]Alexander Pope, *An Essay on Man* (1733), in Robin Sowerby, ed., ALEXANDER POPE: SELECTED POETRY AND PROSE at 153 (1988).

[51]*Preamble* at 23.

[52]IARC has published book-length advice on these issues, but there is no requirement that the animal evidence working subgroup follow the IARC textbook or any other guidance on these matters. *See* John J. Gart, Daniel Krewski, Peter N. Lee, Robert A. Tarone & Jurgen Wahrendorf, STATISTICAL METHODS IN CANCER RESEARCH, VOL. III, THE DESIGN AND ANALYSIS OF LONG-TERM ANIMAL EXPERIMENTS (IARC Sci. Publ. No. 79, 1986).

occur in two or more species, or in one species in two or more studies. In describing animal study outcomes and statistical analyses, the *Preamble* notes that observing dose-response relationships increases the strength of causal inference, but in setting out what findings warrant a conclusion of sufficient evidence of animal carcinogenicity, the *Preamble* omits any reference to dose response.[53]

Another indication of IARC's abridging and diluting causal analysis is apparent from the abandonment of any need for consistency of animal studies beyond the minimal requirement of two studies. If there were ten animal studies, and only two studies found tumors in a single species, IARC consistency would be satisfied. Inconsistency between and among animal species does not undermine the sufficiency of the animal evidence. If different strains of rats yield discordant results, some with excess tumors and some without, the evidence can remain sufficient. If studies in rats and mice yield positive and negative results, again the evidence remains sufficient, but humans are left to ask whether they are more like rats or mice.

The asymmetry of IARC's inculpatory and exculpatory standards again surfaces in its criteria for evaluating animal evidence suggesting lack of carcinogenicity. In discussing these criteria, the *Preamble* calls for multiple studies, of both sexes, in two species showing lack of carcinogenicity. There is no one species option as there is for evidence that will support a sufficient finding in favor of carcinogenicity. IARC goes on to indicate that evidence suggesting no carcinogenicity is limited to the species tested, the specific tumor sites reported, and the levels of agent exposure tested. In other words, sufficient evidence of carcinogenicity in animals can help support inferences of human carcinogenicity across the board, but evidence suggesting lack of animal carcinogenicity cannot be extrapolated to humans, or beyond the tumor type or dose reported. Again, this uneven treatment of positive and negative animal evidence suggests prejudice against exculpatory conclusions, and a tweaking of the decision tree so that more and higher human carcinogenicity classifications can be reached.

D. Definitions for Levels of Mechanistic Evidence

The third stream of evidence for classifying agents for human carcinogenicity is characterized as mechanistic evidence. Mechanistic evidence is a large grab bag of studies conducted on human or animal cells or tissue *in vitro* (laboratory glassware), or in living animals and humans (*in vivo*). Mechanistic studies *in vivo* assess outcomes other than cancer or tumors; they assess a variety of outcomes relevant to potential mechanisms. Such studies may assess the metabolic fate and distribution of the agent, or they may identify how the agent interacts with sub-cellular components, DNA, cells, tissues, or whole organisms in a way that is relevant to the induction of cancer, without determining whether the agent actually increased the risk of any cancer in the whole animal or human.

IARC accords some kinds of mechanistic studies great weight in classifying agents. Mechanistic studies in humans, along with sufficient evidence in experimental animals, suffice in the IARC scheme of things to classify an agent as carcinogenic to humans, group 1, even when epidemiologic evidence is non-existent or otherwise plainly insufficient (limited or inadequate).

As noted above, sufficient human epidemiologic evidence is absent for all groups below group 1. The insufficiency of the epidemiologic evidence in groups 2A, 2B, and 3 should inspire serious skepticism about accepting any classification lower than group 1 as a meaningful carcinogen. As also discussed above, even group 1 classifications may result from plainly

[53] *Compare Preamble* at 24 *with id.* at 32-33.

inconclusive sets of data.

The substantial weight IARC accords certain kinds of mechanistic studies can be seen in the four pathways identified in IARC's decision tree for reaching a group 2A classification. Strong mechanistic studies in human cells or tissues, along with sufficient animal studies, can support classifying an agent in group 2A in the complete absence of human epidemiologic evidence (inadequate). Even with limited human and insufficient animal evidence, IARC can place an agent in 2A with strong mechanistic evidence. Indeed, human or animal evidence is not needed at all for a 2A listing if there is strong mechanistic evidence of a certain sort.

IARC diverges from its evidential categories for human and animal evidence in directing the mechanistic working sub-group to evaluate the evidence before it. As noted above, the third stream of evidence, mechanistic evidence, is unlike human or animal evidence in having a different evidential scale, ranging from inadequate, to limited, to strong, while skipping over sufficient. The rationale for having a different scale for mechanistic evidence is not explained.

The qualifiers for evidential strength of mechanistic evidence are given some content in the *Preamble*, but once again the assignment of qualifiers remains highly subjective. Strong mechanistic evidence requires consistent and coherent results in several experiments. The grading advice does not reconcile whether consistent results in several studies in the face of inconsistent results in other studies undermine the assignment of the strong qualifier to the data set. Similarly, the definition of strong mechanistic evidence points to a substantial number of studies in one or more mammalian species, but the definition does not explain how inconsistent studies are to be reconciled or whether inconsistency requires lowering the grade to limited or inadequate.

Mechanistic studies lose their strong rating if they provide only suggestive evidence, in fewer species, and in fewer end points, or if there are unexplained inconsistencies or incoherence in their results. Mechanistic studies become inadequate when there are no studies, or the studies' results are negative (exculpatory), or when the available studies leave unresolved questions about the validity of their design, conduct, or interpretation.

E. Key Characteristics for Assessing Mechanistic Evidence

Another manifestation of IARCs precautionary, non-scientific approach arises in how the *Preamble* approaches the integration of mechanistic evidence into an overall classification of carcinogenicity.

In 2015, an IARC working group developed a relatively novel approach in the form of identifying common mechanisms of carcinogenesis. The working group looked at group 1 agents as of 2012 and characterized ten "key characteristics" (KCs) that they believed were the principal operating mechanisms of cancer causation. The group touted its identification of KCs as a way of organizing and understanding carcinogenesis, but they also advocated using KCs to predict carcinogenesis in the face of insufficient epidemiology and even insufficient animal evidence. The KCs working group was dominated by members of the Collegium Ramazzini[54] and like-minded scientists who seemed frustrated with the inability to identify more carcinogens, more rapidly. Led by Martyn Smith, the group published the results of their approach to KCs in 2016.[55]

[54]The Collegium Ramazzini is discussed in more detail in Section V.D, *infra*.

[55]Martyn T. Smith, Kathryn Z. Guyton, Catherine F. Gibbons, Jason M. Fritz, Christopher J. Portier, Ivan Rusyn, David M. DeMarini, Jane C. Caldwell, Robert J. Kavlock, Paul F. Lambert, Stephen S. Hecht, John R. Bucher, Bernard

Three years later, with the publication of a revised *Preamble* in 2019, the KCs took center stage in the evaluation of mechanistic evidence. The ten KCs embraced by IARC's Preamble were:

- Is electrophilic or can be metabolically activated to an electrophile;
- Is genotoxic;
- Alters DNA repair or causes genomic instability;
- Induces epigenetic alterations;
- Induces oxidative stress;
- Induces chronic inflammation;
- Is immunosuppressive;
- Modulates receptor-mediated effects;
- Causes immortalization; and
- Alters cell proliferation, cell death, or nutrient supply[56]

More important than using these putative mechanisms as a way of thinking about, organizing, or characterizing carcinogenic processes, working groups were given wide latitude to upgrade classifications, from 3 to 2B, from 2B to 2A, and 2A to 1, with judgments of strong mechanistic evidence in the form of KCs. The *Preamble* does not provide much guidance for how finding KCs involved should be integrated into an overall finding, but recent working groups seemed to have made the most of the opportunity of checking off boxes to bolster judgments of the evidential strength in favor of carcinogenicity. The lawsuit industry has also shown an opportunistic zeal to convert the "check the boxes" approach into bolstering or making their litigation claims that some agent causes cancer in humans.[57]

Although it is still early days, the KCs have received a cool reception in courts.[58] Outside the halls of the Collegium Ramazzini, the KCs concept has received an even cooler reception in the scientific community.[59] Some of the many problems with the novel, non-validated KCs

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W. Stewart, Robert A. Baan, Vincent J. Cogliano & Kurt Straif, *Key Characteristics of Carcinogens as a Basis for Organizing Data on Mechanisms of Carcinogenesis*, 124 ENVT'L HEALTH PERSP. 713 (2016) (Of the 16 authors, seven appear to be members of the Collegium Ramazzini; many of the others are scientists who regularly publish with Collegium members.).

[56]*Preamble* at 37 (Table 3. The key characteristics of carcinogens described by Smith *et al.* (2016)).

[57]Heather Pigman & Marchello Gray, *Plaintiffs' Flawed Reliance on the So-Called "10 Key Characteristics" of Cancer*, FOR THE DEFENSE 31 (Apr. 2024).

[58]*Bulone v. Monsanto Co.*, 737 F. Supp. 3d 898, 905 & n.5 (N.D. Calif. 2024) (Chhabria, J.) (observing that identifying key characteristics by plaintiffs' expert witness, Zhang Luoping, Collegium Ramazzini fellow, did not amount to an opinion on causation); *In re Zantac (Ranitidine) Prods. Liab. Litig.*, 644 F. Supp. 3d 1075, 1279 & n.164 (S.D. Fla. 2022).

[59]*See* Julie Goodman & Heather Lynch, *Improving the international agency for research on cancer's consideration of mechanistic evidence*, 319 TOXICOL. & APPL. PHARMACOL. 39 (2017); James S. Bus, *IARC use of oxidative stress as key mode of action characteristic for facilitating cancer classification: Glyphosate case example illustrating a lack of robustness in interpretative implementation*, 86 REG. TOXICOL. & PHARMACOL. 157 (2017); James E Trosko, *Reflections on the use of 10 IARC carcinogenic characteristics for an objective approach to identifying and*

methodology arise from the identification of KCs by looking only at agents IARC deems group 1 carcinogens. The KCs approach was proposed without the use of non-carcinogenic controls when identifying the ten mechanisms.

Since Martyn Smith's 2016 opinion article, independent groups have subjected the KCs hypothesis to empirical testing. The EPA and other health agencies maintain a database of toxicity test results, known as ToxCast, for an initiative known as Tox21, to generate predictive models for screening tests for carcinogenicity.[60] The IARC working group used ToxCast, but only looked at group 1 agents, to abstract their list of ten KCs. When an independent group conducted a more extensive statistical analysis to examine the predictive relationships between KCs and carcinogenicity, but added non-carcinogens to their models, they found that the KCs' ability to predict carcinogenicity was no better than chance in distinguishing carcinogens from non-carcinogens.[61] The KCs' lack of predictive power is expected; they were developed by reviewing Group 1 agents that had already been classified by 2012 without the need for KCs.[62]

Another key problem for KCs is the approach's failure to control for dose, dose duration, and dose timing. At high exposure levels, many chemicals provoke various toxic reactions that are irrelevant to their primary mode of action as potential carcinogens. As cells approach their cytotoxic threshold, they become distressed and start to die. While *in extremis*, cells undergo processes known as cytoxicity bursts, which create false-positive signals suggesting that specific biological pathways are activated, when in reality, the assays are only measuring the chemical chaos of cells approaching death. For some assays, over 80 percent of positive results may be burst phenomena rather than specific chemical-mechanistic target positivity.[63]

With their KCs checklist, Martyn Smith and his Collegium Ramazzini fellows have created a powerful tool for IARC to manipulate classifications in the direction of creating more carcinogens. The utility of invoking KCs in support of labeling agents as carcinogens can be seen in the recent working group on two polyfluoroalkyl substances (PFAS). PFAS is a broad class of chemicals that are not fungible and which would be expected to have disparate toxicological effects. IARC has treated only two PFAS substances to date.

IARC evaluated one such chemical, perfluorooctanoic acid (PFOA) in 2014, in monograph volume 110, but chose to revisit PFOA in 2023. In 2014, a working group classified PFOA as possibly carcinogenic, group 2B. Under the 2019 *Preamble* with its emphasis on KCs, without any significant new, stronger human evidence, the second IARC working group moved PFOA up two categories to group 1, in its monograph, which was published in 2024. The second working group declared the animal evidence sufficient, and it found strong mechanistic evidence that PFOA shows

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organizing results from certain mechanistic studies, 1 TOXICOL. RES. & APPL. 1 (2017); Carr J. Smith, *et al.*, *Categorizing the characteristics of human carcinogens: a need for specificity*, 95 ARCH. TOXICOL. 2883 (2021); Richard A. Becker, Michael Dourson, Chijioke Onyema & Jessica Ryman, *Beyond key characteristics of carcinogens: an archetypal MOA-based evidence system for hypothesis testing to advance carcinogen risk assessment*, 1 J. TOX. & REG. POL'Y 1 (2025).

[60]US EPA, *Toxicity forecasting: advancing the next generation of chemical Evaluation* (Last updated on Sept. 17, 2025), https://perma.cc/Z5P5-XC74.

[61]Richard A. Becker, *et al.*, *How well can carcinogenicity be predicted by high throughput "characteristics of carcinogens mechanistic data?* 90 REG. TOXICOL. & PHARMACOL. 185 (2017).

[62]Julie E. Goodman, Heather N. Lynch & Lorenz R. Rhomberg, *Letter to the editor re: Guyton et al. (2018), 'Application of the key characteristics of carcinogens in cancer hazard identification'*, 39 CARCINOGENESIS 1089, 1089 (2018).

[63]*Id.*

KCs of carcinogens. The human evidence was insufficient (limited) for two organ sites, testis and kidneys (renal cell carcinoma), and inadequate otherwise. The two sites for which limited evidence was available were not related to the organ sites claimed positive for rodents.[64]

The grade inflationary implications of invoking KCs were also apparent from the 2023 working group's treatment of another PFAS, perfluorooctanesulfonic acid (PFOS). The working group classified PFOS as "possibly carcinogenic to humans," 2B, on the basis of "strong mechanistic evidence" in the form of KCs of immunosuppression and induction of genetic alterations. The working group acknowledged that the human evidence was inadequate, and the animal evidence was insufficient (limited). In other words, the classification of PFOS was based primarily upon only KCs.

These recent classifications of PFOA and PFOS illustrate the ability of the KCs tool to overstate carcinogenicity evidence and manipulate IARC classifications. These exaggerated findings are the predictable result of using a novel, non-validated methodology, and they have been criticized in the scientific literature.[65]

IV. IARC MONOGRAPHS DEVIATE FROM STANDARDS OF CARE FOR SYSTEMATIC REVIEWS

A. The Development of Standards for Systematic Review

When IARC embarked on its monograph program in 1971, the field of evidence integration and synthesis was in its infancy. In the early 1970s, causal claims about human carcinogens typically showed up in narrative reviews that identified studies selectively, typically those that favored the author's preferred result. One or two contrary studies were identified, criticized, and dismissed as unworthy of any serious consideration. Review articles and textbook chapters could easily be found for any outcome, taking opposite positions, and rarely citing the same evidentiary display. At the time, writing reviews was not considered by many as a serious scientific endeavor. The glamour and glory of science was in conducting and publishing new research. Fifty years ago, writing up the findings of other scientists' research was often seen as a parasitic enterprise, not worthy of first-rate scientists. IARC had a void to fill.

In the 1980s and 1990s, the scientific community dramatically changed its views about the value of writing reviews. Scientists came to see that the adversarial cherry picking of data sets, interpreted through post hoc, subjective criteria, led directly to biased, subjective, and inaccurate claims to knowledge.[66]

By the end of the 1990s, the scientific community had embraced the notion that reviews should comprehensively assess the extant evidence and systematically assess what was worthy of reliance and synthesis into a judgment of causality through pre-specified interpretative principles. In 1992, a group of scientists established the Cochrane Collaboration, named after the Scottish

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[64]Shelia Zahm, *et al.*, *Carcinogenicity of perfluorooctanoic acid and perfluorooctanesulfonic acid*, 25 LANCET ONCOL. 16 (2024).

[65]*See* Nicholas L. Drury, Robyn L. Prueitt & Barbara D. Beck, Understanding IARC's PFOA and PFOS carcinogenicity assessments, 154 REG. TOXICOL. & PHARMACOL. 105726 (2024); Yumiao Sun, *et al.*, *Rethinking the Carcinogenic Classification of Perfluorooctanoic Acid (PFOA)*, 59 ENVT'L SCI. & TECH. 11433 (2025).

[66]Cynthia D. Mulrow, *et al.*, *Systematic Reviews: Critical Links in the Great Chain of Evidence*, 126 ANN. INTERN. MED. 389 (1997).

clinical epidemiologist Archibald Cochrane. The Collaboration's goal was to collect and evaluate the available randomized controlled trials for various clinical interventions, and to provide comprehensive, systematic reviews and clinical guidance. The notion quickly spread to encompass observational studies on all health effects research. By the end of the century, two textbooks had been published on systematic reviews, by respected authors.[67]

Over the last 30 years, the systematic review process evolved through discussion and debate, and the publication of several guidance documents on the conduct and reporting of systematic review. In 2009, some leading methodologists published *Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)*, which was updated in 2021.[68] Although the PRISMA guidance ostensibly addresses reporting, if authors have not done something that should be reported, their failure to do it and report about it can be identified as a significant omission from their publication.

A protocol document, with pre-specified inclusion and exclusion criteria for studies to be evaluated, with a careful statement of how risk of bias in studies will be considered, became an important feature of the evolving systematic review. PRISMA called for authors to identify their protocol, and to make it available to the scientific community and the public, in advance of performing and publishing their reviews. The requirement of pre-registration can damp down data dredging in observational studies and experiments, to identify post hoc and tendentious evaluations, and to help readers see when authors have reverse engineered systematic reviews by declaring their criteria for inclusion and exclusion after reading candidate studies and their conclusions.[69]

In the evolutionary development of systematic reviews, a protocol that specified the precise clinical question to be addressed, with well-defined inclusion and exclusion criteria for studies to be considered, became an important feature. In 2011, the Centre for Reviews and Dissemination, at the University of York in England, developed an internet database archive, the International Prospective Register of Systematic Reviews (PROSPERO), for prospectively registering systematic reviews and their protocols. In addition to reducing duplication of systematic reviews, PROSPERO aimed to increase transparency, validity, and integrity of the systematic reviews by making topic-specific protocols available to the scientific community and the public in advance of the authors' undertaking and publishing their reviews.[70]

The other feature that came to be seen as key to careful, unbiased, transparent systematic reviews was the pre-specification for how authors would evaluate studies and their risk of bias. Although assessing risk of study bias cannot be reduced to checklists, various guidance documents bring some rigor to the assessments by reducing inter-subjective variations in disagreements and

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[67]Iain Chambers & Douglas G. Altman, eds., SYSTEMATIC REVIEWS (1995); Cynthia Mulrow & Deborah Cook, eds., SYSTEMATIC REVIEWS: SYNTHESIS OF BEST EVIDENCE FOR HEALTH CARE DECISIONS (1998).

[68]Alessandro Liberati, *et al.*, *The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration*, 6 PLoS MED. e1000100 (2009); Matthew J. Page, *et al.*, *The PRISMA 2020 statement: an updated guideline for reporting systematic reviews*, 10 SYSTEMATIC REV. 89 (2021).

[69] Lisa Bero, *Evaluating Systematic Reviews and Meta-Analyses*, 14 J. L. & POL'Y 569, 576 (2006).

[70]Alison Booth, *et al.*, The *nuts and bolts of PROSPERO: an international prospective register of systematic reviews*, 1 SYSTEMATIC REV. 1 (2012); Alison Booth, *et al.*, *An international registry of systematic review protocols*, 377 LANCET 108 (2011); Matthew J. Page, *et al.*, *Registration of systematic reviews in PROSPERO: 30,000 records and counting*, 7 SYSTEMATIC REV. 32 (2018).

post hoc judgments. Compliance with these structured assessments of study bias, such as the *Risk of Bias in Non-randomised Studies – of Interventions (ROBINS-I)*,[71] or the *Grading of Recommendations, Assessment, Development and Evaluation (GRADE)*,[72] or the Newcastle-Ottawa Scale,[73] have become standard steps in conducting truly systematic reviews.

The sophistication of systematic reviews has grown over time with the enormous growth in the number of such reviews published. Although the systematic review has historical antecedents before 1980, such reviews were quite unusual until the 1980s. According to one historical account, there were 3,000 systematic reviews published between 1980 and 2000, with over 10,000 published each year for the last decade or so.[74]

This explosive growth has led some curmudgeons to deprecate the "mass production of redundant, misleading, and conflicted systematic reviews."[75] The growth, however, at least has had the benefit of fueling refinements to, and standards for, the evaluation of systematic reviews, and to studies that have explored the determinants of reliable reviews.[76] Along with the proliferation of guidance documents, the slim volumes of the 1990s on systematic review methodology have grown into substantial tomes.[77]

B. IARC's Claim to Produce Systematic Reviews

Over the last half century, IARC reviews have also evolved. The most recent *Preamble* maintains that

> [t]he principles of systematic review are applied to the identification, screening, synthesis, and evaluation of the evidence related to cancer in humans, cancer in experimental animals, and mechanistic evidence.[78]

IARC's monographs have some features of systematic reviews, but important features are missing. As the PRISMA and AMSTAR papers show, not all systematic reviews are equal and worthy of trust.

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[71]Jonathan A.C. Sterne, *et al.*, *ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions*, 355 BRIT. MED. J. i4919 (2016).

[72]Gordon H. Guyatt, *et al.*, *GRADE: an emerging consensus on rating quality of evidence and strength of recommendations*, 336 BRIT. MED. J. 924 (2008).

[73]Claudio Luchini, *et al.*, *Assessing the quality of studies in meta-analyses: Advantages and limitations of the Newcastle Ottawa Scale*, 5 WORLD J. META-ANALYSIS 80 (2017).

[74]Mike Clarke & Iain Chalmers, *Reflections on the history of systematic reviews*, 23 BRIT. MED. J. EVID. BASED MED. 121, 121 (2018).

[75]John P. Ioannidis, *The mass production of redundant, misleading, and conflicted systematic reviews and meta-analyses*, 94 MILBANK Q. 485 (2016).

[76]The AMSTAR "A MeaSurement Tool to Assess systematic Reviews" tool assesses methodological quality of systematic reviews. *See* Beverley J. Shea, *et al.*, *Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews*, 7 BMC MED. RES. & METHODOL. 10 (2007); Beverley J. Shea, *et al.*, *AMSTAR 2: a critical appraisal tool for systematic reviews that include randomised or non-randomised studies of healthcare interventions, or both*, 358 BRIT. MED. J. j4008 (2017).

[77]*See, e.g.*, MATTHIAS EGGER, *et al.*, eds. SYSTEMATIC REVIEWSIN HEALTH RESEARCH: META-ANALYSIS IN CONTEXT (3rd ed. 2022) (over 500 pages); Julian P.T. Higgins, *et al.*, COCHRANE HANDBOOK FOR SYSTEMATIC REVIEWS OF INTERVENTIONS (2nd ed. 2019) (over 600 pages).

[78]*Preamble* at 9.

1. *It Is a Matter of Protocol*

A written and registered protocol, publicly available before the identification and collection of relevant studies, is missing from IARC's working procedures. The *Preamble* may function as a high-level flow chart for a working group, but it is devoid of specific information on any given agent that the working group is classifying. The study of any specific agent raises myriad methodological issues, potential study biases, and evidence integration challenges that are unique to the agent and not addressed in the general prescriptions of the *Preamble*. Taking IARC's *Preamble* in its most favorable light, we must acknowledge that it has left many directives to assess study quality to vague, subjective generalizations. IARC's substitution of its *Preamble* for agent-specific protocols, a one-size-fits-all approach, fails to do justice to the complexity of the working group's project on any specific agent, with the result that there is undue opportunity for post hoc assessments of studies and their integration into a classification.

The absence of a proper protocol seriously detracts from the transparency and rigor of IARC's monographs and the credibility of its classifications. In analyses of systematic reviews conducted with and without a prior published protocol, those reviews that were preceded with proper protocols generally were methodologically more rigorous.[79]

2. *The Only Studies that Count*

The *Preamble* indicates that working groups consider, evaluate, and integrate the results from published studies relevant to the three streams of evidence. With respect to unpublished studies, working groups are given discretion to consider the data. This discretion can be biased and outcome-determinative. High quality studies may go unpublished for a variety of reasons. Studies may go unpublished because they are so-called "null" studies that fail to find an adverse outcome and are thus deemed less interesting and "newsworthy" by journal editors. Dissertations may go unpublished completely, or after the graduate students earn their degrees, they may publish selectively, leaving null results out. Even when scientists publish parts of their dissertations in peer-reviewed journals, their full dissertations will provide much more important information and data than can be captured in the six or seven pages permitted by journal editors. Many companies and regulatory bodies conduct high-quality toxicological and mechanistic research, which goes unpublished for proprietary or other reasons. When such studies are before working groups, there appears to be no strong reason to allow the groups discretion over whether to consider them. IARC should, in the interest of comprehensiveness and transparency, shift the burden to the working group to show why, for methodological or qualitative reasons, unpublished research before the working group was not considered.[80]

3. *Cut on the Bias*

Much of the mischief to be avoided by systematic reviews lies in the subjective evaluation of studies for quality and for risk of bias. The *Preamble* calls for working groups to evaluate these aspects of study design, conduct, and reporting, but it omits any structured approach such as ROBIN-I or GRADE. The omission allows undue latitude to study inclusions and exclusions made

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[79] *See* Julia M. L. Menon, *et al., The methodological rigour of systematic reviews in environmental health*, 52 CRIT. REV. TOXICOL. 167 (2022); Sofia Sideri, *et al.*, *Registration in the international prospective register of systematic reviews (PROSPERO) of systematic review protocols was associated with increased review quality*, 100 J. CLIN. EPIDEM. 103 (2018).

[80] Julie E. Goodman, *et al.*, *Recommendations for further revisions to improve the International Agency for Research on Cancer (IARC) Monograph program*, 113 REG. TOXICOL. & PHARMACOL. 104639, at 2 (2020).

to reach a desired conclusion.

IARC is aware of the seriousness of the omitted guidance on evaluating studies for bias. In 2023, IARC leaders met to discuss the omission of standards for evaluating study bias, without conceding that anything had been lacking in the previous 50 years of classification activities.[81] In 2024, IARC published a book-length treatment of bias assessment of epidemiologic studies, but it still does not require working group members to read and apply any of its recommendations, or any other guidelines, in the current *Preamble*.[82]

Like its descriptions for evaluating epidemiologic and toxicological studies, the *Preamble* suggests that working groups may have occasion to study quantitative reviews, such as meta-analyses or pooled studies that attempt to provide summary estimates of association from a group of studies. The pitfalls and potential invalidities of such quantitative reviews are many and have also been the subject of guidance documents and text-length treatments, but the *Preamble* is silent on the many methodological problems that working groups should identify and avoid.[83]

4. Peerless Review

IARC working groups obviously can debate their evaluations among themselves, but their monographs receive no external peer review. The working group, like any collaborative group, is vulnerable to all the vagaries of committee work, including "groupthink" and capture by special interest groups such as the Collegium Ramazzini, which is discussed below. In the controversial 2015 working group's assessment of glyphosate, (discussed in Section IV) editing after the meeting led to mysterious changes no one can or will explain. IARC is hard pressed to argue that its monographs and classifications could not benefit from outside reviewers, especially considering the Agency's idiosyncratic formula for integrating three diverse and often incommensurate streams of evidence and its asymmetrical, biased evaluation of conflicts of interest.

V. THE HAZARDS OF IARC'S GATEKEEPING CONFLICTS OF INTEREST

A. Broadcasting Conflicts of Interest — EMF

In 2011, an IARC working group of 31 scientists met to evaluate whether electromagnetic frequency radio waves (EMF) cause cancer to humans. Anders Ahlbom of the Swedish Karolinska Institute was slated to participate in this working group, but shortly before the group met, IARC disinvited him because of an apparent conflict of interest. The basis for Ahlbom's disqualification was his part-time involvement in his brother's consulting firm that helped telecommunication clients on energy and environmental issues. It is not clear that his consulting issues even involved the supposed carcinogenicity of EMF.[84]

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[81]Mary K. Schubauer-Berigan, *et al.*, *IARC-NCI workshop on an epidemiological toolkit to assess biases in human cancer studies for hazard identification: beyond the algorithm*, 80 OCCUP. & ENVT'L MED. 119 (2023).

[82]Amy Berrington de González, David B. Richardson & Mary K. Schubauer-Berigan, eds., STATISTICAL METHODS IN CANCER RESEARCH, VOL. V: BIAS ASSESSMENT IN CASE-CONTROL AND COHORT STUDIES FOR HAZARD IDENTIFICATION (IARC Sci. Publ. No. 171 2024).

[83]*See, e.g.*, MATTHIAS EGGER, *supra* note 77; Donna F. Stroup, *et al.*, *Meta-analysis of Observational Studies in Epidemiology: A Proposal for Reporting*, 283 J. AM. MED. ASS'N 2008 (2000).

[84] James C. Lin, *The Unusual Story of the IARC Working Group on Radio-Frequency Electromagnetic Fields and Mobile Phones*, THE RADIO SCI. BULL. 54 (Sept. 2011).

To show that it can strain at gnats while swallowing camels, IARC, in assembling the EMF working group, did not challenge the interests of Lennart Hardell. An outspoken advocate for the harmfulness of EMF from mobile phones, Hardell is a Swedish scientist who, at the time of the IARC EMF working group, had been involved in EMF cancer litigation for over a decade as an expert witness for plaintiffs. Despite these conflicts, IARC permitted Hardell to serve on the EMF working group, and even to serve as a subgroup leader. One of Hardell's accomplishments was his testimonial adventures in federal court, in Maryland, as an expert witness for an EMF brain cancer claimant. After a comprehensive hearing, the court excluded Hardell's proposed expert witness testimony, and his exclusion was affirmed on appeal.[85] It is difficult to imagine a more serious conflict of interest than the one possessed by Hardell, who clearly had staked advocacy positions on EMF, and who was well motivated to seek vindication for his exclusion of his testimony, by having IARC recognize EMF as a carcinogen.

B. Weeding Out Conflicts — Glyphosate

The IARC working group on glyphosate met from March 3 to 10, 2015. The journal *Lancet Oncology* announced the group's 2A classification, on March 20, 2015, with its online release of an article.[86] Christopher Portier was invited to participate in the working group, not as a member, but as an "invited specialist," which was peculiar because by his own testimony, Portier had not previously looked at the scientific evidence for or against the carcinogenicity of glyphosate.[87]

Portier's participation in the IARC glyphosate working group looks even more peculiar given other facts about his various activities. In 2014, he somehow managed to push his way onto the IARC Advisory Committee and obtain a leadership role, with the result that glyphosate would be evaluated the following year by an IARC working group. Portier is by training a statistician; he is neither an epidemiologist nor a toxicologist.

For about two months before the 2015 working group meeting, Portier had been consulting with a law firm, Lundy, Lundy, Soileau & South. Portier insists, however, that his consultations with the Lundy firm before the March 2015 working group meeting concerned another agent reviewed by IARC, not glyphosate. Within two weeks of IARC's announcement of glyphosate's 2A classification, Portier signed a lucrative agreement with two law firms, Lundy and Weitz & Luxenberg. Soon after, the law firms were advertising for clients to sue glyphosate manufacturers.

When asked in an examination before trial about his lucrative consulting for the two plaintiffs' law firms, Portier became defensive and evasive about his relationship with the lawsuit industry and claimed his discussions were confidential. When pressed for when he first discussed a glyphosate consulting arrangement with the Lundy lawyers, Portier's said he couldn't recall.[88] The inquiry was certainly a relevant and fair one. The facts indicated that two plaintiffs' law firm selected him as an expert witness for glyphosate claimants within days of his return from the IARC

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[85]*Newman v. Motorola, Inc.*, 218 F. Supp. 2d 769 (D. Md. 2002), *aff'd*, 78 F. Appx 292 (4th Cir. 2003) (per curiam).

[86]Kathryn Guyton, *et al.*, "Carcinogenicity of tetrachlorvinphos, parathion, malathion, diazinon, and glyphosate," 16 LANCET ONCOL. 490 (2015) (published online on Mar. 20, 2015, according to the journal's website, https://tinyurl.com/5ttksw93.

[87]Deposition of Christopher Portier at 40, in *In re Roundup Prods. Liab. Litig.*, MDL No. 2741, case no. 16-md-02741-VC (Sept. 5, 2017) [cited as Portier Dep.]

[88]Portier Dep. at 77.

meeting. Law firms do not make such retention decisions at the drop of the hat.

While Portier was chairing the 2014 advisory group that selected glyphosate for evaluation, and then serving as an invited specialist for the 2015 working group, he was employed by the Environmental Defense Fund (EDF), an extreme environmentalist group of the sort that Sir Richard Peto criticized back in 1980 for exaggerating hazards and ignoring benefits, with biased judgments and quasi-religious certainty. Since its inception in 1967, EDF has campaigned against the use of pesticides. For his participation in the 2014 advisory group and the 2015 working group, Portier was required to file a disclosure statement of his interests that might be real or apparent conflicts with disinterested scientific analysis. Portier did not disclose his work for EDF because that work involved air pollution, climate change, and hydraulic fracking, and the work was not in his opinion a conflict of interest.[89]

IARC boasts that it selects working group scientists for expertise and the absence of conflicts, real or apparent.[90] The Portier affair reveals the hypocrisy in that boast. Up until his dubious participation in the 2015 working group, Portier claimed he was *tabula rasa* on glyphosate, but his anti-industry and environmentalist zealotry were well-established priors. Within weeks of the meeting, Portier was a retained expert witness for plaintiffs, and he was globe-trotting on a lobbying and speaking campaign to seek to ban the herbicide. Portier's litigation consulting on glyphosate evolved quickly into a testimonial role, with his appearances in several of the early glyphosate civil trials.

Over a year after the glyphosate working group meeting, *Reuters* published an investigative report that detailed significant alterations between the draft and the final monograph for glyphosate.[91] The preparation of monographs is normally a closed, non-transparent process, but litigation in the United States resulted in the release of drafts of the animal toxicology section. Journalists at *Reuters* obtained the drafts and published a careful comparison, which detailed deletions, additions, and re-analyses made in the final draft. Although only the animal toxicology section was available for review, the IARC working group's finding of "sufficient" animal evidence was crucial to its 2A classification for glyphosate.

Reuters reported ten substantial alterations made in the animal evidence section of the draft monograph. In every case, an interpretation of a study as null (showing no effect) was replaced with an interpretation that the study was either neutral or showed a relevant effect. Although changes in draft reports are expected as the result of deliberations and discussions, the changes that *Reuters* found were suspicious for their timing, substance, and bias towards finding the animal evidence to be sufficient. When the chairman of the IARC glyphosate toxicology sub-group, Charles Jameson, testified in litigation as an expert witness for plaintiffs, he was unaware of who altered his drafts, or when or why the changes were made. Christopher Portier admitted in sworn testimony that the animal toxicology subgroup evaluated the toxicology evidence as "limited evidence of animal carcinogenicity" (insufficient) during the subgroup's working sessions. Portier could not explain when or how the subgroup and the entire working group flipped and came to regard the animal evidence as sufficient, which was necessary for IARC to classify glyphosate in group 2A.[92]

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[89]Portier Dep. at 39.

[90]*Preamble* at 4.

[91]Kate Kelland, *In glyphosate review, WHO cancer agency edited out 'non-carcinogenic' findings*, REUTERS (Oct. 19, 2017), https://tinyurl.com/sfezzctw.

[92]Portier Dep. at 50-57.

In a demonstration of its own non-transparency, IARC responded to the *Reuters* article with self-serving denials, claims of confidentiality, and calls to the working group members "not to feel pressured to discuss their deliberations" outside the confines of IARC.[93] In response to requests for emails and documents to glyphosate working group members under freedom of information laws, IARC instructed the scientists not to disclose documents, and claimed that IARC was the "sole owner of such materials."[94]

When the House of Representatives held hearings in 2018 on glyphosate,[95] it requested that Christopher Wild, then Director of IARC appear to testify. Not only did Wild refuse to appear, but he also submitted a truculent letter that blamed the agro-chemical industry for having the temerity to launch an "unprecedented, coordinated" campaign to undermine IARC.[96] Wild insinuated that the unflattering journalistic coverage of the glyphosate monograph came from media associated with the agro-chemical industry. *Reuters*' reporter Kate Kelland's coverage of IARC's glyphosate shenanigans had won journalism awards from both the American Association for the Advancement of Science, and the Foreign Press Association.[97]

In his statement to Congress, Wild accused "industry" (the manufacturing type not the lawsuit variety) of having misrepresented IARC's work, but he failed to engage with detailed criticisms of data dredging, statistical errors, biased analyses, and omitted and cherry-picked evidence.[98] Wild inaccurately asserted that most of the questioned edits of the glyphosate monograph concerned one review article. In the face of the flood of criticisms, Wild implausibly claimed that IARC monographs are "transparent and open to scrutiny." On the sensitive issue of Christopher Portier, IARC's Director claimed ignorance on whether Portier was under a contractual relationship with a law firm when the glyphosate working group met in March 2015.

IARC's and Wild's performance in response to Congressional oversight was the very antithesis of transparency. Wild never furnished the information requested by the House Committee on Science, Space and Technology. In both the glyphosate hearing and generally, IARC refuses to share drafts or minutes of discussions, eschews public comment, and vilifies any negative comment as industry's efforts to manufacture doubt.

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[93]IARC, *IARC rejects false claims in* Reuters *article* (Oct. 24, 2017), https://perma.cc/QS4S-3UUU; IARC, Monograph on Glyphosate - Other Related Information (Sept. 10, 2017), https://perma.cc/2WRB-UJTK.

[94]Kate Kelland, *WHO cancer agency asked experts to withhold weedkiller documents*, REUTERS (Oct. 25, 2016), https://tinyurl.com/byvskshu.

[95]In Defense of Scientific Integrity: Examining the IARC Monograph Programme and Glyphosate Review, Hearing before the Committee on Science, Space, and Technology of House of Representatives, 115th Congress, 2nd Session, Serial No. 115-46 (Feb. 6, 2018).

[96]Christopher Wild, Director, *IARC response to criticisms of the Monographs and the glyphosate evaluation* (Jan. 2018), https://perma.cc/SB55-YWCU.

[97]Reuters, *Reuters wins journalism awards from Foreign Press Association and American Association for the Advancement of Science*, REUTERS (Nov. 22, 2017), https://tinyurl.com/39cbfnkb.

[98]*See, e.g.*, Robert E. Tarone, *Conflicts of interest, bias, and the IARC Monographs Program*, 98 REG. TOXICOL. & PHARMACOL. A1-A4 (2018); Geoffrey Kabat, *Who's Afraid of Roundup?* 64 ISSUES SCI. & TECH. 64 (Fall 2019); Kenny Crump, *et al.*, *Accounting for Multiple Comparisons in Statistical Analysis of the Extensive Bioassay Data on Glyphosate*, 175 TOXICOL. SCI. 156 (2020).

C. *Milward* and CERT

Martyn Smith is a toxicologist at the University of California, and a fellow in the Collegium Ramazzini. His paper on the "key characteristics" of carcinogens influenced IARC's modification of its *Preamble* to permit mechanistic evidence to support group 1 classifications for agents with insufficient human epidemiology, and group 2A classifications with insufficient epidemiologic and toxicological evidence. Smith has served as the United States representative on IARC's Scientific Council (2010 to 2014), as well as on several IARC working groups, including one that met in 2018 to address benzene.

Smith has also testified as a paid expert witness for claimants in chemical-exposure litigation. While sitting on IARC's Scientific Council, Smith testified as an expert witness in a notorious benzene exposure case, *Milward*.[99] Carl Cranor, a philosophy professor and member of the Collegium Ramazzini, testified in turn, as a retained expert witness, in support of Smith's idiosyncratic methodology. The *Milward* case has since become legendary in the American jurisprudence of medico-legal evidence for its misdirection of legal and scientific principles.[100]

After an extensive evidentiary hearing, the *Milward* trial court found numerous deviations from scientific and statistical standards of care and excluded Smith's proffered testimony. Milward appealed, as was his right to do, but on appeal, the Center for Research and Education on Toxics (CERT) filed an amicus brief in support of Milward, and in support of the excluded testimony given by Smith and Cranor.

Readers of the CERT amicus brief would not know much about the organization other than its self-aggrandizing claims to promote education and research on toxins. The brief had over 25 signatories, many of whom are frequent testifiers for the lawsuit industry in litigation of cancer claims, and members of the Collegium Ramazzini. Nevertheless, CERT's brief made the following questionable disclaimer:

> None of the amici has any financial or other similar interest in the outcome of this lawsuit. Amici appear on their own behalf to inform this Court of the substantial medical knowledge and understanding of leukemia arising from exposure to benzene.[101]

The claim that none of the amici had a financial interest in the outcome was not entirely accurate, given that the trial court's searching scrutiny and exclusion of Smith's testimony, if applied in other courts, would curtail the lawsuit industry's entrepreneurial endeavors and the work of the signatories as expert witnesses. There was also the matter of CERT's business model

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[99] *Milward v. Acuity Specialty Products Group, Inc.*, 664 F. Supp. 2d 137 (D. Mass. 2009), *rev'd*, 639 F.3d 11 (1st Cir. 2011), *cert. denied sub nom.*, *U.S. Steel Corp. v. Milward*, 565 U.S. 1111 (2012). On remand from the First Circuit, Mr. Milward's case was dismissed after his expert witnesses failed to show specific causation with sufficient reliable evidence. *Milward v. Acuity Specialty Products Group, Inc.*, 969 F. Supp. 2d 101 (2013). Given that the general causation opinion is no longer necessary to the final judgment in the case, its continuing validity as precedent is doubtful.

[100] Note, *Federal Rule of Evidence 702 - Judicial Conference Amends Rule 702*, 138 HARV. L. REV. 899, 903 (2025); Thomas D. Schroeder, *Toward a More Apparent Approach to Considering the Admission of Expert Testimony*, 95 NOTRE DAME L. REV. 2039, 2044 (2020); David E. Bernstein & Eric G. Lasker, *Defending* Daubert*: It's Time to Amend Federal Rule of Evidence* 702, 57 WM. & MARY L. REV. 1, 23, 42 (2015) ("*Milward* was incorrectly decided and should be overruled."); Nathan A. Schachtman, *Desultory Thoughts on* Milward v. Acuity Specialty Products, in DAUBERT PRACTICE 2013 (Practicing L. Instit. 2013), DOI: 10.13140/RG.2.1.5011.5285, https://tinyurl.com/azpwv6s9.

[101] Nathan Schachtman, *The Council for Education and Research on Toxics*, TORTINI (July 9, 2013), https://perma.cc/6J68-CHMK. A copy of the CERT amicus brief is on file and available from the author.

as bounty hunter for damages under California's Prop 65 law for failing to label carcinogens, as in its infamous litigation crusade against Starbucks for selling coffee without a cancer warning label.[102] CERT's financial interest was clear. The lower the epistemic standards for reaching conclusions of carcinogenicity, the more targets become available for CERT's extortionate lawsuits.

The most remarkable conflict of interest not disclosed in CERT's appellate brief was one that the Court of Appeals certainly had a right to know. CERT was urging the admissibility of testimony by Smith and Cranor, without whose testimony Milward would have had no case at all. The 2001 California records of CERT's establishment identify a lawyer, Raphael Metzger, as the contact person for the organization. Metzger has been the lawyer for CERT's Prop 65 enforcement actions, bounty hunting expeditions against manufacturing and marketing industries. The California incorporation records for CERT identify four co-founders, two of whom were the plaintiff's paid expert witnesses in *Milward*: Carl Cranor and Martyn Smith.

By the time Smith and Cranor testified in *Milward*, they were no longer listed as directors or officers, but CERT had been funding the research of Smith and his students at the University of California, through directed donations to the school.[103] In one meta-analysis funded by CERT,[104] the corresponding author was an official in the California Office of Environmental Health Hazard Assessment, an office that participates in the Prop 65 process that enables CERT's bounty-hunting litigation goals. You could not ask for a better example of regulatory capture, and deep, undisclosed conflicts of interest.

Other than funding Martyn Smith, and playing gotcha with corporations under Prop 65, there is scant evidence of CERT's "educational" activities. Besides the amicus brief filed in *Milward*, CERT has filed other amicus briefs, always in support of the lawsuit industry's objectives. On one occasion in 2008, a not-for-profit organization, the Green Science Policy Institute, acknowledged CERT's generous support. In one bankruptcy case, CERT intervened with a claim for $9 billion as reimbursement for its "educational" efforts. Bankruptcy Judge Robert Gerber was not impressed with CERT's efforts and dismissed its claim.[105]

IARC has never seemed troubled by Smith's founding of and participation in CERT, his receiving support from the lawsuit industry in the form of expert witness fees, or the funding that flows to him and his laboratory from CERT's Prop 65 litigation efforts.

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[102] Michael Waters, *The secretive non-profit gaming California's health laws*, THE OUTLINE (June 18, 2018), https://perma.cc/6F2E-CRRA; Beth Mole, *The secretive nonprofit that made millions suing companies over cancer warnings: Meet the obscure group behind the fight over cancer warnings on coffee*, ARS TECHNICA (June 6, 2019), https://tinyurl.com/4ynbtkx9. *See* Alexander Nazaryan, *Will coffee in California come with a cancer warning?* L. A. TIMES (Feb. 18, 2018), https://perma.cc/Y2MU-XSDP.

[103] *See, e.g.*, Jimmy Phuong, *et al.*, *Predicted Toxicity of the Biofuel Candidate 2,5-Dimethylfuran in Envt'l & Biological Systems*, 53 ENVT'L & MOL. MUTAGENESIS 478 (2012); Michele Fromowitz, Joe Shuga, Antonio Wlassowsky, Zhiying Ji, Matthew North, Chris Vulpe, Martyn T. Smith, and Luoping Zhang, *Bone Marrow Genotoxicity of 2,5-Dimethylfuran, a Green Biofuel Candidate*, 53 ENVT'L & MOL. MUTAGENESIS 488 (2012); Reuben Thomas, *et al.*, *Using Bioinformatic Approaches to Identify Pathways Targeted by Human Leukemogens*, 9 INT'L J. ENVT'L RES. & PUB. HEALTH 2479 (2012).

[104] Frolayne Carlos-Wallace, Luoping Zhang, Martyn T. Smith, *et al.*, *Parental, In Utero, and Early-Life Exposure to Benzene and the Risk of Childhood Leukemia: A Meta-Analysis*, 183 AM. J. EPIDEM. 1 (2016).

[105] Caroline Humer, *Judge rules against big Chemtura bankruptcy claim*, REUTERS (April 8, 2010), https://tinyurl.com/3khas8vh; John Parry, *Chemtura hits back at $9 billion claim over toxins*, REUTERS (Mar. 24, 2010), https://tinyurl.com/2e7kyfh9.

D. The Dodgy Influence of Collegium Ramazzini

Bernardino Ramazzini, a 17th century Italian physician, is sometimes called the father of occupational medicine.[106] His children have been an unruly lot.

The Collegium Ramazzini was founded by an American physician, Dr. Irving Selikoff, in 1982. At the time, Selikoff was well known for his ability to influence legislation and regulations for occupational health, largely as a result of advocating for the 1970 law that established OSHA, and for stricter asbestos workplace exposure limits. Selikoff was masterful at marketing and proselytizing. According to a news report in 1983, Selikoff represented that his Collegium would not lobby or seek to initiate legislation; rather it would interpret scientific findings in accessible language, show the policy implications of these findings, and make recommendations. Selikoff announced that the Collegium:

> will advise on the adequacy of a standard, but will not lobby to have a standard set. Our function is not to condemn, but rather to be a conscience among scientists in occupational and environmental health.[107]

Selikoff's early pronouncements suggested that union officials *and* business leaders would be welcome within the Collegium,[108] but in fact business leaders and industry scientists were excluded from the inception. Only later would Selikoff's *bona fides* come under scrutiny for misrepresenting his medical training,[109] misrepresenting his extensive role in testifying for asbestos claimants,[110] and for falsely downplaying the use of crocidolite asbestos in the United States, among other things.[111] In 1990, Selikoff, along with asbestos plaintiffs' lawyer Ron Motley and the Collegium Ramazzini, paid for judges to attend a one-sided *ex parte* meeting with plaintiffs' expert witnesses.[112] The result was the disqualification of a federal judge who presided over an asbestos property damage class action.

Selikoff's promises about refraining from lobbying ultimately proved to be disingenuous. In 1997, Douglas Liddell, a scientist who had published extensively on asbestos hazards, spoke up

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[106] Giuliano Franco & Francesca Franco, *Bernardino Ramazzini: The Father of Occupational Medicine*, 91 AM. J. PUB. HEALTH 1382 (2001).

[107] Drew Von Bergen, *A group of international scientists, backed by two senators*, UNITED PRESS INT'L ARCH. (May 10, 1983), https://perma.cc/ME6J-VABB.

[108] Paula Butturini, *New medical group seeks to prevent work-related diseases*, UNITED PRESS INT'L ARCH. (Oct. 19, 1983), https://perma.cc/BLF5-AB2J.

[109] Peter W. J. Bartrip, *Irving John Selikoff and the Strange Case of the Missing Medical Degrees*, 3 J. HIST. MED. 58 (2003); *see also* Nathan A. Schachtman, *Selikoff Timeline & Asbestos Litigation History (Revised)* TORTINI (Feb. 26, 2023), https://perma.cc/DNZ6-7EUF.

[110] *See* Nathan A. Schachtman, *Selikoff and the Mystery of the Disappearing Testimony*, TORTINI (Dec. 3, 2010), https://perma.cc/9RY6-TXK7.

[111] Nathan A. Schachtman, *Selikoff and the Mystery of the Disappearing Amphiboles*, TORTINI (Dec. 10, 2010), https://perma.cc/W4C9-K79G.

[112] *In re School Asbestos Litigation*, 977 F.2d 764 (3d Cir. 1992). *See* Cathleen M. Devlin, *Disqualification of Federal Judges – Third Circuit Orders District Judge James McGirr Kelly to Disqualify Himself So as to Preserve 'The Appearance of Justice' Under 28 U.S.C. § 455* – In re School Asbestos Litigation (1992), 38 VILL. L. REV. 1219 (1993); Bruce A. Green, *May Judges Attend Privately Funded Educational Programs? Should Judicial Education Be Privatized?: Questions of Judicial Ethics and Policy*, 29 FORDHAM URB. L. J. 941, 996-98 (2002).

about the insinuations and bad science from Selikoff and his minions at Mount Sinai School of Medicine. Breaking with the genteel norms of science, Liddell called out his detractors:

> [A]n anti-asbestos lobby, based in the Mount Sinai School of Medicine of the City University of New York, promoted the fiction that asbestos was an all-pervading menace, and trumped up a number of asbestos myths for widespread dissemination, through media eager for bad news.[113]

By the time that Liddell called out Selikoff, "the Lobby" had become institutionalized informally as a world-wide network of physicians and scientists, and formally as the Collegium Ramazzini. Selikoff's promises of neutrality and consensus in his Collegium soon dissolved.

Selikoff's professed neutrality was belied by Collegium membership and fellowship rules that disqualify "[p]ersons who have any type of links which may compromise the authenticity of their commitment to the mission of the Collegium Ramazzini."[114] In other words, persons who are too close to manufacturing industry, and not sufficiently friendly to the lawsuit industry and environmentalist advocacy groups, need not apply.

In science, as in the law, the burden of showing that an agent is carcinogenic resides with those claiming causation. The precautionary principle, sometimes subtly, more often overtly, shifts the burden to those claiming non-carcinogenicity. The Collegium and its members are committed to the precautionary principle,[115] which poses a major ideological conflict of interest when its members publish, testify, or participate in the IARC monograph process. The list of the Collegium's American fellows reads like a *Who's Who* of expert witnesses for the lawsuit industry. An examination of the signatories to the CERT amicus brief in the *Milward* appeal reveals that 15 of the 27 were Collegium fellows. Other signatories were adjacent to the Collegium in their publication and testimonial activities.

Many of the Collegium fellows have had important roles in the IARC program.[116] Kurt Straif, a Collegium fellow, was head of the IARC monograph program (2011 - 2018) and later head of IARCs Section of Evidence Synthesis and Classification (until 2018). Another Collegium fellow, Vincent Cogliano, has also served as head of the IARC monograph program (2003 - 2010). Collegium fellow Harry Vaino headed the monograph program (1983 - 1994). James Huff, Collegium fellow, served at IARC as a senior scientist on chemical carcinogenesis (1980 – 2013). Huff later became an associate director of the National Institute of Environmental Health Sciences, in the United States. Collegium fellows, such as Bernard Goldstein, Leslie Stayner, Christopher Portier, Martyn Smith, Peter Infante, David Hoel, Zhang Luoping, and Ronald Melnick, have appeared on several IARC working groups, as well as in multiple American tort cases as expert witnesses. Some Collegium fellows who have played an important role in specific IARC working groups—without disqualification—include Lennart Hardell, Christopher Portier, and Martyn Smith.

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[113]Francis Douglas Kelly Liddell, *Magic, Menace, Myth and Malice*, 41 ANN. OCCUP. HYG. 3, 3 (1997).

[114]"Collegium Ramazzini Membership," https://perma.cc/9B25-WXB9.

[115]Council of Fellows, *Seventh Collegium Ramazzini Statement - The Precautionary Principle: Implications for Research and Policy Making*, (Oct. 25, 2003), 45 AM. J. INDUS. MED. 380 (2004); Collegium Ramazzini, *The Precautionary Principle: Implications for Research and Policy Making*, 11 HUM. & ECOL. RISK ASSESS. 3 (2005).

[116]*See Photo gallery: 50 years of the IARC Monographs*, https://perma.cc/S7X4-EATK.

In addition to the undue influence the Collegium exercises by its members' participation in IARC Working groups, the group also floods the zone of cancer publications through journals controlled or dominated by its members. Ruth Etzel and Philippe Grandjean, the editors in chief of the journal *Environmental Health*, for instance, are both Collegium fellows, as was the journal's founding editor in chief, David Ozonoff. Many other associate editors and members of the editorial board for *Environmental Health* come from the ranks of the Collegium. Other journals, such as the *American Journal of Industrial Medicine*, are less monolithically aligned with the Collegium, but still show sizable presence of its fellows and members amongst their editorial boards.

Perhaps scientists who are ideologically committed to the precautionary principle can put aside their strongly held views when they travel to Lyon, France, to participate in IARC working groups. What they cannot do is avoid the vagueness, ambiguity, loopholes, and infelicitous definitions that tilt in favor of reaching conclusions that favor precaution over established fact.

E. Positional Conflicts Created by Past Publications

Scientists are invested in the results of their own research and have been known to engage in motivated reasoning to disregard contrary results or criticisms of their own work. Scientists who have published about a specific agent may be the most obviously expert in the relevant studies, but they may also be especially keen to see their studies validated and endorsed in providing "sufficient" evidence of carcinogenicity. In the face of criticism over their studies, some authors become zealous in seeking the vindication that IARC can deliver through its classification process. The professional success of authors may turn or fall upon whether their studies are evaluated favorably by an IARC working group.

IARC openly accepts study authors as working group members for classifying the agents that were the focus of authors' work. The problem created is twofold. First, the presence of scientists whose work will be under evaluation creates a palpable conflict of interest, even if those authors recuse themselves from directly weighing in on their own studies. Others in the room know that they are evaluating the potentially contested work in front of the work's authors. Second, by accepting study authors, whose views are declared in print, IARC can cull the herd in advance to ensure a working group that will deliver a desired verdict.

As with the EMF working group, IARC openly embraced the participation of Lennart Hardell, although he had been an outspoken advocate, an expert witness for the lawsuit industry, and an author of an epidemiologic study that was part of the controversial claim that EMF causes cancer. Similarly, IARC winked at the positional biases and expert witness work of Martyn Smith by allowing his participation in a benzene working group. In 1996, IARC convened a working group to consider crystalline silica, and made epidemiologist Harvey Checkoway the chair of the group, notwithstanding that Checkoway was the author of a key paper under working group scrutiny. Not surprisingly, Checkoway's paper received substantial weight in the working group's evaluation,[117] while other important studies were ignored.[118]

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[117]Harvey Checkoway, *et al.*, *Mortality among workers in the diatomaceous earth industry*, 50 BRIT. J. INDUS. MED. 586 (1993).

[118]*See* Patrick A. Hessel, *et al.*, *Silica, Silicosis, and Lung Cancer: A Response to a Recent Working Group Report*, 42 J. OCCUP. & ENVT'L MED. 704, 717-18 (2000) ("The data demonstrate a lack of association between lung cancer and exposure to crystalline silica in human studies. Furthermore, silica is not directly genotoxic and has been shown to be a pulmonary carcinogen in only one animal species, the rat, which seems to be an inappropriate model for assessing particulate carcinogenesis in humans.").

When confronted with criticisms about the selection of working group members, IARC has responded with defensive rationalizations. In the aftermath of the glyphosate debacle, Kurt Straif responded hyperbolically and without evidence that IARC's working groups are made up of "the world's best experts," who presumably have super-human powers of not being influenced or swayed by their own work in the field, and are immune to bias created by reputation or career advancement.[119]

F. Teams of Rivals — IARC Needs to Reform its Handling of Conflicts

IARC has badly mismanaged the process of policing conflicts of interest. IARC's failure stems largely from the Agency's single-minded obsession with the interests of manufacturing industry, while burying its head in the sand when it comes to the conflicts of those who are aligned with the lawsuit industry or environmental activism. Of course, any process for reviewing conflicts will fail if candidates make their own determination of what is relevant for others to know about their potential biases and prejudices. As shown by the case studies above, IARC's process for reviewing potential conflicts of interests could be vastly improved by considering non-financial conflicts that arise from ideological and political commitments, expert witness engagements, institutional and personal affiliations, and others.[120]

One strand that runs through all the cases discussed in this section is the tight connection between IARC's leadership, some influential working group participants, and the Collegium Ramazzini. The Collegium is committed to the precautionary principle, and many of its American fellows and members are active participants in American tort cases on behalf of the lawsuit industry. The infiltration of IARC by Collegium members and environmentalists committed to the precautionary principle goes a long way toward explaining why IARC classifications may disagree with other scientific groups on several agents such as glyphosate[121] and the artificial sweetener aspartame.[122]

As in politics and culture, there are echo chambers in the world of science. IARC believes that its process works to forge consensus classifications. Its consensus appears to be achieved, however, by excluding dissenting points of view and excusing conflicts of interest of those advocating compensation schemes or environmentalist goals. IARC's procedures thus lend themselves to faux consensus, and overstatements of hazards, which is in line with the precautionary principle commitments of its leadership and many working group participants.

IARC's exclusionary approach yields not only faux consensus, but also methodologically bad science. In 1960, the biophysicist and Nobel laureate Georg von Békésy described how having

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[119]Kate Kelland, How the World Health Organization's cancer agency confuses consumers, REUTERS (Apr. 18, 2016) (quoting Straif), https://tinyurl.com/5akkd7tr.

[120]*See, e.g.*, Saroj Niraula & Ian F. Tannock, *Non-financial conflicts of interest*, 22 NATURE REV. CLIN. ONCOL. 895 (2025); David Resnik, *Disclosing and managing non-financial conflicts of interest in scientific publications*," 19 RES. ETHICS 121 (2023); David Resnik, *Conflicts of Interest in Scientific Research Related to Regulation or Litigation*, 7 J. PHIL., SCI. & LAW 1 (2007); The PLoS Medicine Editors, *Making Sense of Non-Financial Competing Interests*, 5 PLoS MED. e199 (2008).

[121]*See, e.g.*, EPA, *Glyphosate*, (May 9, 2025) ("glyphosate is unlikely to be a human carcinogen"), https://perma.cc/5W95-5GL5; Jose V. Tarazona, *et al.*, *Glyphosate toxicity and carcinogenicity: a review of the scientific basis of the European Union assessment and its differences with IARC*, 91 ARCH. TOXICOL. 2723 (2017). Virtually every other national agency has concurred with EPA's exculpatory verdict on glyphosate.

[122]*See, e.g.*, *FDA Response to External Safety Reviews of Aspartame* (Feb. 27, 2025), https://www.fda.gov/food/food-additives-petitions/aspartame-and-other-sweeteners-food.

an "enemy" was more valuable for correcting errors than a multitude of friends because enemies are "willing to devote a vast amount of time and brain power to ferreting out errors both large and small, and this without any compensation."[123] The great scholar and scientist of cognitive bias, Daniel Kahneman, elaborated on von Békésy's idea, with the suggestion that adversaries actually collaborate on the same team to devise more stringent tests that might resolve disagreements and advance knowledge.[124] Several scientists concerned about poor reproducibility of scientific results have recently urged Kahneman's proposal of adversarial collaboration to promote more severely tested and robust research.[125] If IARC were to adopt this approach, and engage with adversarial points of view, it might have fewer consensus monographs, but more credible conclusions in the end. The presence of opposing points of view would serve as a therapeutic for close-mindedness and insularity all around.[126]

VI. THE OPACITY OF IARC'S TRANSPARENCY

IARC asserts that its monograph program is open and transparent. The words "transparency" and "transparent" each occurs five times in the *Preamble*. Despite these aspirational claims, there is much that is opaque in IARC's operations. Both scientists and lawyers have criticized IARC for its lack of transparency.[127]

IARC classification meetings are cloaked in secrecy. Members of IARC Advisory or working groups promise in writing to keep the proceedings of the group confidential.[128] In particular, members agree "not to communicate the deliberations and decisions of the Advisory Process to third parties except as agreed by IARC/WHO." When working group candidates receive their forms for disclosing interests, they also receive IARC's Code of Conduct, which requires that they "respect the confidential nature of committee or meeting deliberations or of the advisory function assigned by IARC/WHO and not make any public statements regarding the work of the committee or meeting or regarding the expert's advice without prior consent from IARC/WHO."[129]

Monograph number 112, which includes the IARC glyphosate classification, illustrates some of the problems that arise from the secrecy oath. As noted above, the subgroup for animal toxicology rated the available studies as insufficient ("limited") to support animal carcinogenicity in interim meetings, but the final evaluation flipped to "sufficient," a conclusion that was necessary to push glyphosate into group 2A. No one at IARC can or will come forward to explain the change. Instead of transparency, IARC has chosen a strategy of obfuscation and *ad hominem* attacks on its

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[123]GEORG von BÉKÉSY, EXPERIMENTS IN HEARING at 8 (E.G. Wever, ed. 1960).

[124]Daniel Kahneman, *Experiences of collaborative research*, 58 AM. PSYCH. 723 (2003). *See also* MICHAEL WHITE, RIVALS: CONFLICT AS THE FUEL OF SCIENCE (2001).

[125]Stephen J. Ceci, *et al.*, *Adversarial Collaboration: An Undervalued Approach in Behavioral Science*, AM. PSYCH.; in press (Aug. 15, 2024), https://dx.doi.org/10.1037/amp0001391; Stephen J. Ceci, *et al.*, *Teams of Rivals* 113 AM. SCIENTIST 336 (2025).

[126]Muqtafi Akhmad, *et al.*, *Closed-mindedness and insulation in groupthink: their effects and the devil's advocacy as a preventive measure*, 4 J. COMPUT. SOC. SCI. 455 (2021).

[127]*See, e.g.*, Julie Goodman & Heather Lynch, *Improving the International Agency for Research on Cancer's consideration of mechanistic evidence*, 319 TOXICOL. & APPL. PHARMACOL. 39 (2017); David B. Fischer, *The IARC Monographs Program – sowing public confusion, controversy, and criticism: a commentary*, 1 J. TOX. & REG. POL'Y 1 (2025).

[128]IARC, *Confidentiality Undertaking* (2022), https://perma.cc/4WT9-AS2E.

[129]*IARC Code of Conduct for IARC/WHO Experts* (2022), https://perma.cc/2XXM-YF4C.

critics. In October 2017, in response to revelations and criticisms about irregularities in the glyphosate working group, IARC issued a statement that the deliberations leading to a monograph are confidential and not for public view.[130]

IARC claims that monographs represent consensus conclusions among the working group members, but this claim is fiction. Consensus may be little more than a bare numerical majority. Whether a consensus or a bare majority, the votes might be the result of confusion, mistake, or error introduced into the discussions and debate by one or more working group members.

Scientists should, of course, be free to discuss and deliberate, and even to change their interpretations in response to the debate. The opacity of IARC's procedure prevents external assessment of the reasons for members' change of mind. Instead of owning its lack of transparency, IARC blames industry by claiming that its secretiveness is required to protect working group members from undue external influence or pressure. In a vitriolic turn, IARC points to the glyphosate case as one that justifies its secrecy by alleging stakeholders' efforts to undermine the agency's credibility with an "unprecedented number of orchestrated actions."[131] The agency complained about media inquiries and subpoenas to glyphosate working group members, but it ignored that IARC participants were responsible for creating the need for the inquiries and document requests by taking advocacy roles in the lawsuit industry. In any event, IARC seems to have been able to undermine its credibility without any help or external influence. Reacting poorly to criticism is yet another aspect of IARC's lack of transparency.

IARC's response to the glyphosate controversy reveals a great deal about its institutional mindset. While complaining about "vested interests," IARC's leadership and many of its working group members have special interests because of their membership in the Collegium Ramazzini. Many of the Collegium's membership work in service of the American lawsuit industry as consultants or testifying expert witnesses. Given the ideological and positional conflicts of interest rife throughout IARC, it should be opening its deliberative process to inspection, not trying to shame stakeholders into silence. IARC's failure to manage its leaders' and its working groups' conflicts of interests testify to the non-transparency of the agency's processes.

A. Moral Panics over Industry Influence

More panics over the possibility of undue commercial influence over the Agency and its working groups have become commonplace at IARC.[132] In 2003, the editors of *The Lancet* campaigned to limit what they claimed was undue influence from commercial interests. The IARC Director pushed back against suggestions that non-industry third parties (environmental advocacy groups) attend working groups but acquiesced in the call for procedures that allow undisclosed interests to be identified in advance of meetings.[133]

One of the interlocutors in this 2003 debate, Dr. Cornelia Baines, offered insightful points to which the IARC Director never responded. Baines noted that some of the most intense conflicts

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[130]IARC – *Statement on IARC Monographs deliberations* (Oct. 2017), https://perma.cc/R9PM-CGPX.

[131]Christopher Wild, *IARC response to criticisms of the Monographs and the glyphosate evaluation* (Jan. 2018), reproduced in *In Defense of Scientific Integrity: Examining the IARC Monograph Programme and Glyphosate Review*: Hearing Before the House Comm. on Science, Space & Tech., 115 Cong. Serial No. 115–46, at 128 (Feb. 6, 2018).

[132]*Editorial: Transparency at IARC*, 361 LANCET 189 (2003).

[133]Paul Kleihues, *Transparency at the International Agency for Research on Cancer (IARC)*, 361 LANCET 781 (2003).

of interest will be found not in ties with commercial influencers, but in those working group members who "come armed with their zealously-promoted and ferociously-defended versions of the 'truth', making predictable what they will recommend at the end of the review process."[134] Baines described IARC's disclosure process as a naive procedural "minuet." Presciently for 2003, Baines recommended that working group members should have the chance to see and comment upon the editing that takes place *after* the group meeting ends and before publication. These calls for transparency went unheeded and are unheeded still.

B. Lack of Peer Review

After IARC working group meetings, the groups' classifications are announced in short articles, published in *Lancet Oncology*. The groups' monographs are eventually published as volumes, usually within a few months of the meetings, available on the IARC website. The monographs are not peer-reviewed publications.

Nemo iudex in causa sua (no one should be a judge in his own case) is a maxim in law and morality, and it would be a good rule for IARC as well. IARC monographs result from a deliberative process that is closed and secretive, and the monographs lack many of the indicia of high-quality systematic reviews. Once a group has committed to a conclusion, its members have an interest in defending it against criticism. The natural tendency of groups to engage in "groupthink,"[135] to deflect or refuse criticism, combined with the deficiencies in the IARC process, make the absence of external peer review problematic.

The absence of peer review that hurts the transparency of the IARC monograph process is uncommon in the production of true systematic reviews. The Cochrane Collaboration, for example, requires that all Cochrane Reviews, and their protocols, be subjected to external peer review by outside experts, independent of the authors. Cochrane anticipates that the external reviews will require revisions to attain desired quality and rigor in the systematic review.[136] The myriad systematic reviews published each year in higher quality journals undergo peer review of varying quality. IARC, however, remains steadfast in rejecting external review of its monographs before publication.

C. Secret Elections

In 2008, when IARC held an election for a new director, the agency officially refused to release the names of candidates. IARC rebuffed calls for transparency by claiming that it feared that "special interests" might attempt to influence the outcome.[137] The agency was clearly unconcerned about the special interests represented by the Collegium Ramazzini, whose members pervade IARC's leadership and who would be both aware of the candidates and in positions to influence the selection process.

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[134]Cornelia J. Baines, *Transparency at the International Agency for Research on Cancer (IARC)*, 361 LANCET 781 (2003).

[135]*See generally* DAVID M. ALLEN& JAMES W. HOWELL, eds., GROUPTHINK IN SCIENCE: GREED, PATHOLOGICAL ALTRUISM, IDEOLOGY, COMPETITION, AND CULTURE (2020); Gregory Moorhead, *et al.*, *The Tendency toward Defective Decision Making within Self-Managing Teams: The Relevance of Groupthink for the 21st Century*, 73 ORGANIZATIONAL BEHAV. & HUMAN DECISION PROCESSES 327 (1998).

[136]*See Cochrane Database of Systematic Reviews: editorial policies*, https://perma.cc/X76C-NS58.

[137]Adrian Burton, *IARC election: candidates' names kept secret...again*, 9 LANCET ONCOL. 517 (2008).

Transparency is a widely agreed-upon value, but too often transparency is in the eye of the beholder. Cloudy, biased vision may blur the distinction between the transparent and the opaque. The call for transparency must give way to specific, operational criteria for conditions that foster transparency.[138]

The lack of transparency afflicts not only IARC procedures; IARC's methodology is plagued by non-transparency as well. IARC has criteria for evaluating and labeling carcinogenicity, but as discussed in sections I, II, and III, its definitions and criteria are directionally biased in favor of more and higher classifications of carcinogenicity. The porosity of IARC's methodology permits ideological and precautionary biases to infiltrate the classification process.

D. False Consensus

Working groups vote in the open, but IARC maintains confidentiality as to who voted in what way. Although IARC claims transparent processes, its open (to those persons in the secret meeting) but unrecorded voting creates subtle but real pressures to agree with the crowd. The failure to record votes undermines transparency and accountability.

IARC claims that its goal for working groups is consensus,[139] but decisions are made based on a bare numerical majority and sometimes reflect deep divides among the voters. In the 1996 working group meeting on crystalline silica, one member (writing before IARC imposed stricter secrecy oaths) reported "a seemingly interminable debate," which ended "in a narrow vote, reflecting the majority view of the experts present at that particular time."[140] This working group member's account suggests that the working group was "required to reach a conclusion, and *plumped* for Group 1."[141] IARC's claim that it disallows advocacy at working group meetings is false and unsustainable.

E. IARC's Opaque Methodology

As discussed in section III, above, the *Preamble* fails to specify how working groups will evaluate the risk of bias in human and animal studies. After elevating the role of mechanistic studies in its new 2019 *Preamble*, IARC fails to describe how this stream of evidence should be integrated with animal and human evidence. Despite its 40-page *Preamble*, IARC approaches its review of each specific agent's carcinogenicity without a preregistered agent-specific protocol. IARC has failed to conduct its evaluations and classification in accord with evolving standards of systematic review of causal studies. These and other indicia of non-transparency include problems that are discussed above, in Section IV, on IARC's deviations from systematic review methodology.

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[138]Sander Greenland, *Transparency and disclosure, neutrality and balance: shared values or just shared words*, 66 J. EPIDEM. COMM. HEALTH 967, 967 (2012).

[139]*Preamble* at 7.

[140]Corbett McDonald, *Silica and Lung Cancer: Hazard or Risk*, 44 ANN. OCCUP. HYG. 1, 1 (2000) (emphasis added).

[141]*Id.* at 2.

VII. IARC GOES TO COURT

A. Reference Manual on Scientific Evidence

The Federal Judicial Center and the National Academies of Science, Engineering and Medicine publish the *Reference Manual on Scientific Evidence*, a primer for judges in need of remedial education in statistics and science. The third edition of the *Reference Manual on Scientific Evidence* was published in 2011; a fourth edition was released in the last days of 2025. Because the *Reference Manual* is published by the research and education arm of the federal judiciary, it can be influential in court cases involving scientific evidence.

Without any support or analysis, the third edition of the *Reference Manual* accords IARC undeserved accolades. Various chapters of the text refer to IARC as "well-respected and prestigious," "well regarded," and "reputable."[142] An introductory chapter by the late Professor Margaret Berger, misleadingly characterizes IARC's review processes as "not review[ing] each scientific study individually for whether it reliably supports the causal claim being advocated or opposed."[143] Berger offered this tendentious, incorrect interpretation of IARC's reviews to bolster her own personal belief that courts should not look at the validity of individual studies. A single human epidemiologic study rarely is offered as the basis for a causal claim, and a single animal study could only be offered to show causality in a specific species and strain of laboratory animal. According to the *Preamble*, however, IARC's working groups are very much supposed to review individual epidemiologic studies for whether they reliably report an association, or animal studies for whether they support a causal interpretation (for the animals in the study). In her field of evidence law, Professor Berger was known for having advanced several antic proposals, including abandoning both general causation in tort law and gatekeeping of expert witness testimony in evidence law.[144]

The new, fourth edition of the *Reference Manual*[145] continues the tradition of giving IARC a higher regard than the evidence would warrant. The new edition's chapter on toxicology carries forward the characterization of IARC as "respected."[146] Perhaps even more uncritical and unscholarly, the new *Reference Manual*'s chapter on epidemiology describes IARC monographs as "well regarded," and that courts "generally recognize" IARC monographs as "authoritative."[147] The *Manual*'s claim that courts recognize IARC monographs as authoritative is completely

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[142]National Academies of Science, Engineering & Medicine and Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE at 20, 564 n.46, 646 (3d ed. 2011).

[143]Margaret A. Berger, *The Admissibility of Expert Testimony*, in REFERENCE MANUAL, *id.*). The chapter was published after Professor Berger died, and it cites cases that were decided after her death. The error noted above cannot be fully attributed to her; it may have been introduced gratuitously by an overzealous editor. The gist of the mistaken representation of IARC's process, made without any reference to the Preamble, is, however, consistent with Professor Berger's publications in her lifetime.

[144] Margaret A. Berger, *Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117 (1997).

[145]National Academies of Sciences, Engineering, and Medicine & Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (4th ed. 2025).

[146]David L. Eaton, Bernard D. Goldstein, & Mary Sue Henifin, *Reference Guide on Toxicology*, at 1027, 1045, in *supra*, note 139. Bernard Goldstein is a fellow of the Collegium Ramazzini.

[147]Steve C. Gold, Michael D. Green, Jonathan Chevrier, & Brenda Eskenazi, *Reference Guide on Epidemiology*, at 897, 919-20 & n.68, in note 139, *supra*. Professor Eskenazi is a fellow of the Collegium Ramazzini.

unsupported by the one case cited for the proposition, which never addressed the supposed authority of the glyphosate or any other IARC monograph.[148]

B. Judicial Opinions

IARC classifications surface in most litigations of cancer causation claims, even when they are not dispositive. In a 40-year-old federal court case involving a claim that tobacco snuff caused plaintiff's oral cancer, the plaintiff sought to have the relevant IARC monograph and classification admitted directly into evidence as a "governmental report" or otherwise hearsay for which there is sufficient guarantee of trustworthiness. The court rejected this attempt to place IARC's work product directly into evidence. The Tenth Circuit affirmed.[149] IARC reviews become a part of court proceedings, as do other studies and reviews, not by being directly admissible, but by having been considered and relied upon by the parties' expert witnesses in presenting opinion testimony for or against the cause of a claimant's particular cancer.

When IARC working groups evaluate the potential for an agent to be carcinogenic to humans, they do not lump all cancers together and look for an increase in cancer incidence or mortality. Working groups focus on specific cancer types by organ or tissue type and evaluate the evidence with respect to one or more *target* organs.[150] Unfortunately, and misleadingly, when IARC communicates its classifications, it often omits information about the target organ. Such communications mislead the public, and even many scientists, into believing that an IARC carcinogen causes every imaginable sort of cancer. Although IARC does not explicitly endorse such fallacious inferences, it fails to block them with clear communication of its conclusions.

In 1996, the Fifth Circuit expounded upon the fallacious extrapolation from an IARC classification with respect to a target organ to another type of cancer that had afflicted the claimant. In *Allen v. Pennsylvania Engineering Corp.*, the court affirmed the trial court's dismissal of a plaintiffs' claim that exposure to a sterilizing agent, ethylene oxide, had caused Walter Allen's brain cancer. The trial court excluded plaintiffs' expert witnesses because their proffered testimonies were unreliable and thus inadmissible under Federal Rule of Evidence 702 and then granted summary judgment to the defendant. The plaintiffs' expert witnesses relied heavily upon IARC's classification of ethylene oxide as a group 1 agent. The trial and appellate courts, however, were not impressed with the reliance upon IARC's classification.[151]

First, the court excluded Allen's expert witnesses for having advanced what it characterized as an amorphous, subjective "weight of the evidence" methodology in support of their opinions. The Court of Appeals explained that it was not persuaded by this methodology used by IARC as well as regulatory agencies. The court insightfully observed that IARC and other groups evaluate evidence from a "preventive perspective" to reduce exposure or promulgate prophylactic rules.[152] Second, the court saw through the disconnect between IARC's evaluation of ethylene oxide as a

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[148] *See id.*, citing *Hardeman v. Monsanto Co.*, 997 F.3d 941, 967 (9th Cir. 2021) (affirming lower court's admission of IARC categorization of glyphosate as "a probable carcinogen"), *cert. denied*, 142 S. Ct. 2834 (2022). The *Hardeman* case is discussed in more detail, *infra* at 45-48).

[149] *Marsee v. United States Tobacco Co.*, 639 F. Supp. 466, 470 (W.D. Okla. 1986), *aff'd*, 866 F.2d 319, 325 (10th 1989).

[150] *Preamble* at 21, 24, 27, 29.

[151] *Allen v. Pennsylvania Engineering Corp.*, 102 F.3d 194 (5th Cir. 1996).

[152] *Id.* at 198. It might have been more accurate for the court to have characterized IARC's perspective as precautionary.

cause of lymphopoietic cancer and the plaintiffs' expert witnesses' reliance upon the classification to support a claim that ethylene oxide causes brain cancer.

Not all courts have written with the clarity and insight of the court in *Allen*. The West Virginia Supreme Court demonstrated the consequence of IARC's poor communications, with a shift from one target organ to another organ, as if the evidence for cancer claimed was of the same quality and quantity as that for the target organ described by IARC. This misuse of IARC classifications, rejected by *Allen*, is common in legal arenas. In the West Virginia case, Ronald Harris claimed that he developed multiple myeloma from his workplace exposure to diesel exhaust fumes. IARC classified diesel fumes in group 1 based on the evidence of a causal association with lung cancer, not multiple myeloma. Undeterred, Harris enlisted the help of two expert witnesses, both fellows of the Collegium Ramazzini, Bernard Goldstein and Peter Infante, who extrapolated from one type of cancer to another. IARC has never considered the epidemiology of diesel fume exposure to be sufficient to show an association with multiple myeloma. The trial court saw through the ruse and excluded plaintiffs' expert witnesses. The West Virginia Supreme Court, however, reversed (3-2), and remanded to the trial court for trial.[153]

The appellate majority opinion, despite much hand wringing, noted the IARC group classification for diesel fumes, but it never came to grips with the insufficiency of the relevant epidemiologic evidence for diesel fumes and the cancer at issue, multiple myeloma, which IARC had never endorsed as sufficient, either on epidemiology or toxicology.

Courts seem prone to misunderstand the modal qualifiers "probable" and "possible," as used in IARC classifications. In one case, plaintiffs claimed they developed various cancers from exposure to hydrazines released from defendant's plant. The district court noted that IARC had classified hydrazines in group 2B, and then proceeded to give a wildly inaccurate interpretation that 2B "means that it has determined that there is sufficient evidence to conclude that hydrazines are carcinogenic in animals and that it is probable that hydrazines are carcinogenic in humans."[154] The opinion does not reveal who was the source of the misinformation. The court never reached the medical causation questions; it dismissed the cases on grounds that the plaintiffs had failed to show exposure to hydrazines.

Another case that illustrates the use and abuse of IARC classifications arose from a claim that exposure to perchloroethylene (PCE) causes acute myelomonocytic leukemia (AMML), a subtype of acute myelogenous leukemia (AML). Kathy Magistrini worked for a dry cleaner for about two years, and then, over a year later, she developed AMML. With the support of expert witness David Ozonoff (a Collegium Ramazzini fellow), Magistrini sued her former employer's suppliers of dry-cleaning fluid PCE. For his opinion that PCE caused plaintiff's AMML, Ozonoff relied upon an IARC monograph and classification of PCE as a "probable" carcinogen, which classification had been based upon the finding of leukemia in rats exposed to PCE, and limited (insufficient) epidemiology in the form of a few studies that reported elevated rates of esophageal, cervical cancer, and non-Hodgkin's lymphoma, in PCE-exposed persons.[155]

Ozonoff claimed he used a weight-of-the evidence review, like IARCs approach in its 1995 monograph. He explicitly relied upon the IARC 2A classification that suggested PCE was a

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[153] *Harris v. CSX Transportation, Inc.*, 232 W.Va. 617, 753 S.E.2d 275 (2013).

[154] *Renaud v. Martin Marietta Corp.*, 749 F. Supp. 1545, 1547 (D. Colo. 1990), *aff'd*, 972 F.2d 304 (10 Cir. 1992).

[155] *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 589, 600 (D.N.J. 2002), *aff'd*, 68 Fed. Appx. 356 (3d Cir. 2003).

probable carcinogen, but he ignored the target organ information, and the idiosyncratic definition of "probable" in IARC parlance. Citing to a defense expert toxicology witness, the trial court's opinion incorrectly described the meanings of the IARC classifications.[156] The court never acknowledged that, for IARC, "probable" has no quantitative meaning and is merely somewhat more possible than merely possible. From the published opinion, it is impossible to tell whether the failure to acknowledge IARC's contrived meaning of "probable" was a failure of advocacy or of judging.

After discovery and briefing, the trial judge held pre-trial hearings, with the assistance of a disinterested technical advisor, to determine the admissibility of Ozonoff's opinions. Magistrini attempted to show the reliability of Ozonoff's methodology with additional witnesses, including testimony from Philip Landrigan, also a Collegium fellow, and Sander Greenland, a statistician who often testified for plaintiffs.

Ozonoff gave his own study the greatest weight, although plaintiff's statistical expert witness, Greenland, testified that the Ozonoff study's paucity of cases and huge confidence interval made the results unstable and statistically imprecise. Ozonoff also failed to explain his giving sleight weight to other studies that found either no association or an inverse one, with more precise, narrower confidence intervals than his own study. Greenland, an accomplished statistician, testified that he could not identify what, if any, methodology was used by Dr. Ozonoff in weighing the evidence.[157] On this record, the trial court found that Ozonoff's opinion was inadmissible, and the Third Circuit affirmed.

A long-standing inconsistency in IARC's hazard-risk distinction was its disparate classification of benzene (group 1) and gasoline (group 2B), even though gasoline contains 1 to 2 percent benzene. In 2025, IARC removed the inconsistency by re-classifying gasoline in group 1, in what will likely be yet another controversial classification.[158] The lower classification of gasoline before 2025 resulted in adverse rulings on plaintiffs' claims that gasoline exposure had caused them to develop cancer.

In a 2015 federal court case, a plaintiff's claim that gasoline caused her husband to develop and die of AML foundered on the insufficiency of the epidemiologic evidence for gasoline exposure and AML. The trial court upheld the defendant's challenge to the admissibility of plaintiff's expert witness testimony, largely because IARC had classified gasoline differently from benzene.[159] The witness the court excluded, Robert Harrison, is a fellow of the Collegium Ramazzini.

C. IARC Monographs as Evidence of State-of-the-Art for Knowledge of Hazard

IARC classifications have occasionally been used defensively. Christopher Lightfoot claimed he developed nasal cancer from working as a boy in his father's workshop, from 1981 to 1992. He sued the lumber companies, which raised a state-of-the-art defense, in which the defendants asserted that until IARC classified wood dust in group 1, in 1995, they did not have a duty to warn about a cancer risk. The trial court granted summary judgment based on this defense,

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[156] *Id.* at 600 & n.18.

[157] *Id.* at 606-08.

[158] Michelle Turner, *et al.*, *Carcinogenicity of automotive gasoline and some oxygenated gasoline additives*, 26 LANCET ONCOL. 549 (2025).

[159] *Burst v. Shell Oil Co.*, 120 F. Supp. 3d 547, 554 (E.D. La. 2015).

which the Fourth Circuit upheld.[160]

D. IARC Turns Glyphosate into a Tortogen

The glyphosate litigation probably represents the most extensive consideration given to an IARC classification in the adjudication of personal injury cancer claims. Through concert of action between the lawsuit industry and a key participant in IARC's working group, litigation claims were filed shortly after IARC announced its 2015 classification of glyphosate as a 2A "probable" carcinogen. The federal courts consolidated cases in a multi-district litigation (MDL) before District Judge Vince Chhabria, in San Francisco, for pre-trial procedures.

Judge Chhabria quickly realized that the IARC 2A classification was the centerpiece of the plaintiffs' cases.[161] Following modern MDL practice, Judge Chhabria heard pre-trial Rule 702 motions from both sides, to exclude the other sides' expert witnesses' opinions as inadmissible for not being based on sufficient facts and data, and for not being based upon reliable (valid) methods, reliably applied to the facts of the case. Despite the plaintiffs' rhetoric, Judge Chhabria recognized that, notwithstanding the IARC classification, the plaintiffs' expert witnesses would have to do their own analysis and synthesis of the evidence; the opinions of IARC (or for that matter the opinions of regulatory agencies) would be of secondary importance. His decision is perhaps one of the clearest expositions of why plaintiffs cannot satisfy the legal requirements simply by showing up with an IARC classification.[162]

Plaintiffs' counsel and their expert witnesses relied heavily on the IARC glyphosate monograph and classification in supporting the admissibility of their witnesses' opinions.[163] Judge Chhabria held hearings and heard from the challenged expert witnesses, after which hearings, he ruled that the plaintiffs' case, through at least some of their designated expert witnesses, could proceed.[164] The decision to green-light the plaintiffs' expert witnesses was surprising and hard to reconcile with the court's comments that the plaintiffs' witnesses' testimony was "shaky," and that the admissibility *vel non* of the challenged expert witnesses was a "very close question."[165] Judge Chhabria characterized the plaintiffs' case on general causation, even when viewed "in its totality," as "too equivocal to support any firm conclusion that glyphosate causes Non-Hodgkin's Lymphoma (NHL). This calls into question the credibility of some of the plaintiffs' experts, who have confidently identified a causal link."[166] Claiming to follow decisions of the Ninth Circuit, which were dubious at the time and since repudiated by a 2023 revision to Rule 702, Judge Chhabria

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[160]*Lightfoot v. Georgia-Pacific Wood Prods., LLC*, 441 F. Supp. 3d 159, 171 (E.D.N.C. 2020), *aff'd*, 5 F.4th 484 (2021).

[161]*In re Roundup MDL Prods. Liab. Litig.*, MDL No. 2741, Pretrial Order No. 15, Third-Party Discovery and Pending Motions to Seal (Mar. 13, 2017), https://perma.cc/WN5W-2X55.

[162]*Id.* at 1113 (citing the hazard-risk distinction).

[163]*In re Roundup MDL Prods. Liab. Litig.*, MDL No. 2741, Plaintiffs' Response in Opposition to Monsanto Company's *Daubert* and Summary Judgment Motion Based on Failure of General Causation Proof and *Daubert* motion to Strike Certain Opinions of Monsanto Company's Expert Witnesses (Oct. 27, 2017), https://perma.cc/77RX-SZPY.

[164]*In re Roundup Prods. Liab. Litig.*, MDL 2741, 390 F. Supp. 3d 1102 (N.D. Cal. 2018). The six expert witnesses challenged by the defense were Beate Ritz, Christopher Portier, Alfred Neugut, Charles Jameson, Dennis Weisenburger, and Chadhi Nabhan. *Id.* at 1111.

[165]*Id.* at 1108, 1136, 1151-52.

[166]*Id.* at 1109.

concluded that the plaintiffs' expert witnesses' opinion, "while shaky," were admissible.[167]

Aside from applying an incorrect standard favoring admissibility, Judge Chhabria's opinion is one of the few reported judicial decisions to come to terms, at least partially, with the nature of IARC classifications. Judge Chhabria acknowledged that the plaintiffs and their hired expert witnesses relied heavily on the IARC classification of glyphosate as a group 2A agent, "probably carcinogenic to humans." As a "hazard" determination, however, IARC's classification did not and could not answer the relevant legal question about risk; namely, whether the herbicide glyphosate as actually used causes NHL at real-world exposures experienced by the plaintiffs. In addition, Judge Chhabria made the uncommon acknowledgement that when IARC classifies an agent in group 2A, it has no intention of giving "probable" a quantitative meaning. The classification thus has nothing to do with science, where probability is most definitely a quantitative concept, or with law, where courts conceptualize probably as more likely than not, a quantitative probability greater than 0.5 (50%).[168]

By engaging with the meaning of an IARC 2A classification, Judge Chhabria's gatekeeping steered clear of some basic errors, only to fall prey to others. The court failed to engage with the full range of mistakes put forward by plaintiffs' expert witnesses. After calling out some of the plaintiffs' expert witnesses for misrepresenting the meaning of an IARC "hazard," in group 2A, or simply parroting IARC's analysis and conclusions,[169] the MDL court got lost in the weeds.

The court's discussion of basic statistical concepts, such as the confidence interval, reflected a basic misunderstanding of random error. The court's decision repeated the common mistake that "a 95% confidence interval, the standard interval, is a range that would capture the actual odds ratio 95% of the time if the study were conducted repeatedly."[170] Judge Chhabria confessed from the bench that he did not understand statistical inference.

Judge Chhabria evaluated the plaintiffs' expert witnesses who claimed to have used something like Sir Austin Bradford Hill's approach for assessing causality in associations. Unfortunately, Judge Chhabria acquiesced in serious misrepresentations about Sir Austin's approach, which requires in the first instance that chance, bias, and confounding be ruled out before going on to ask whether the association is rendered more plausible from animal or mechanistic evidence.[171] Judge Chhabria allowed plaintiffs' expert witnesses to brush off the need to eliminate chance, bias, and confounding, and then misrepresent weak associations as moderate

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[167] *Id.* at 1151-52. For instance, Judge Chhabria cited to language in Ninth Circuit decisions that a district court must make its analysis of admissibility in view of the "liberal thrust favoring admission," even knowing that such favoritism could determine the decision. *Id.* at 1112. Presumptions and favors violate the statutory mandate that the burden of showing admissibility is on the proponent of an expert witness's proffered testimony.

[168] *Id.* at 1108-09.

[169] *Id.* at 1115.

[170] *Id.* at 1117. Sadly, Judge Chhabria correctly cited the *Reference Manual on Scientific Evidence* for this erroneous definition. REFERENCE MANUAL, *supra* note 139, at 580-81. No one confidence interval captures the true population estimate 95% of the time; rather it is a long run of resampling for sample estimates with all their confidence intervals, for which 95% of all those confidence intervals will cover the true estimate.

[171] Austin Bradford Hill, *The Environment and Disease: Association or Causation?* 58 *PROC. ROYAL SOC'Y MED.* 295, 295 (1965) ("Disregarding then any such problem in semantics we have this situation. Our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?").

ones,[172] ignore the need for consistency, and urge animal evidence that was unrelated to non-solid cancers of white blood cells. Judge Chhabria acknowledged that one of plaintiffs' witnesses, epidemiologist Beate Ritz, studiously ignored confounding issues in her review of the epidemiologic studies, which in the court's view called her "objectivity and credibility into question."[173] It is difficult to imagine a more serious deviation from the standard of care for epidemiologists, or something that would be more disqualifying under Rule 702; yet the court denied the motion to exclude Ritz's testimony.[174]

After the glyphosate working group decamped from Lyon, France, in 2015, the evidentiary display of epidemiology, which was insufficient for IARC, became even more insufficient (and more inconsistent) with the publication of the Agricultural Health Study, in 2018.[175] Plaintiffs' expert witness Christopher Portier agreed with the IARC evaluation of the epidemiology as insufficient ("limited") because chance, bias, and confounding had not been ruled out, but then for purposes of reaching an admissible opinion in litigation, Portier insisted that these three varieties of error had been ruled out.[176] The court described Portier's characterization of the association between glyphosate and NHL as strong, as a "stretch," with fuzzy data and virtually all the study risk ratios were below 2.0 (and some below 1.0), many not statistically significant.[177]

Further failures of gatekeeping occurred in evaluating how plaintiffs' expert witnesses relied upon animal studies. The MDL court did not consider the jugglery at the IARC working group that led the initial insufficient ("limited") grade for animal evidence to be recast inexplicably as sufficient. The court acknowledged that the "sufficient" evaluation for animal evidence was what allowed the IARC group to reach a 2A classification, but the court failed to appreciate the implications of IARC's algorithmic process. Such upgrading was inconsistent with Bradford Hill's approach, which requires the association to be "perfectly clear-cut and beyond what we would care to attribute to the play of chance," before moving on to his nine considerations, one of which was animal experimental evidence.

Additionally, the upselling of "probable" conclusions follows from IARC's commitment to precautionary principle policy; it does not result from scientific principles. The IARC *Preamble* candidly acknowledges that the utility of "*sufficient evidence of carcinogenicity* in experimental animals" lies largely in its ability to make human carcinogenicity more plausible, not more

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[172]"Weak associations showing odds ratios of less than (say) three to one are often due to chance, bias, or confounding." Richard Doll, *Weak Associations in Epidemiology: Importance, Detection, and Interpretation*, 6. J. EPIDEM. S11, S11 (1991). Labeling an association as weak, moderate, or strong is imprecise and subjective, but the range of magnitudes for relative risks for established causal associations provide some perspective and guidance. The causal association between maternal use of thalidomide and phocomelia among children is measured in risk ratios over 1,000, as is the causal association between occupational exposure to crocidolite asbestos and mesothelioma. The causal association between long-term cigarette smoking and lung cancer is two orders of magnitude lower, with risk ratios around 30 to 40. The risk ratios in the epidemiology of glyphosate and NHL range from below 1.0, to close to1.0, to some, inconsistently just above 1.0. IARC itself judged the 2015 epidemiology as insufficient, and it is hard to see how any honest expert witness could say anything other than that the epidemiologic evidence was weak and inconsistent.

[173]*In re Roundup Prods. Liab. Litig.*, *supra* note 161, at 1140.

[174]*See* Mark A. Behrens & Andrew J. Task, *The Rule of Science and the Rule of Law*, 29 SW. L. REV. 436, 445 - 48 (2021) (criticizing judicial abrogation of statutory requirement for gatekeeping methodological dubious expert witness opinion testimony).

[175] Gabriella Andreotti, *et al.*, *Glyphosate Use and Cancer Incidence in the Agricultural Health Study*, 110 J. NAT'L CANCER INSTIT. djx233 (2018).

[176]*In re Roundup Prods. Liab. Litig.*, *supra* note 161, at 1131.

[177]*Id.* at 1133.

factual.[178] Kurt Straif, who headed up the IARC monograph program for many years, has candidly acknowledged "[c]onsistent with the precautionary principle, sufficient animal cancer evidence can be used to classify an agent as potentially carcinogenic to humans in the absence of adequate human data." The scientific patina with which IARC cloaked its precautionary conclusion misled Judge Chhabria and subverted the factual adjudication of expert witness admissibility in the glyphosate litigation.[179]

Furthermore, plaintiffs' expert witnesses failed to explain the significance of solid non-malignant and malignant tumors in rodents to the judgment that glyphosate causes a cancer of white blood cells in humans. Instead, the court accepted the glib generalization that solid tumors in rodents would somehow increase the biological plausibility that glyphosate caused a blood cancer in humans.

The court recognized that the "evidence of a causal link between glyphosate exposure and NHL in the human population seems rather weak,"[180] but it failed to follow through with the appropriate exclusionary ruling. The MDL district court's decision was not reviewed on appeal until the first federal case was tried, lost by Monsanto, and appealed. The Court of Appeals perfunctorily reviewed and affirmed Judge Chhabria's superficial evaluation of plaintiffs' expert witnesses' opinions as not an abuse of discretion.[181] Neither Judge Chhabria nor the Ninth Circuit ever held that the IARC classification was authoritative.

Notwithstanding that punitive damages must be based upon an intentional or reckless disregard of a *known* risk, the trial court allowed a punitive damage award to stand (although reducing the amount as excessive). How an evidentiary display of causation that is weak, inconsistent, shaky, and a "close call," can support imposing punishment for ignoring a *known* risk is one of the great mysteries of 21st century American tort law.

VIII. IARC'S INFLUENCE ON SCIENCE AND REGULATION

When the IARC monograph program started in 1971, there was no comparable source for comprehensive reviews on the carcinogenicity of chemicals and other agents. With the establishment of EPA and OSHA in the early 1970s, there was an immediate need for comprehensive reviews and syntheses of scientific evidence on various chemicals of concern.

Although IARC professes abstinence from recommending regulations, legislation, or policy decisions, the agency understands its classifications are grist for the regulatory mill. Indeed, the Agency seems well satisfied with its ability to influence regulations internationally.[182] Although the IARC *Preamble* eschews regulatory risk assessments, IARC once again reveals its precautionary bias when it declares that its classifications, without more, may be the basis for regulatory action.

When the European Food Safety Authority (EFSA) rejected IARC's 2015 classification of glyphosate,[183] IARC Director Christopher Wild wrote an imperious letter to Dr. Bernhard Url, the

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[178] *Preamble* at 23.

[179] Kurt Straif, *supra* note 10, at 31.

[180] *In re Roundup Prods. Liab. Litig.*, *supra* note 161, at 1108.

[181] *Hardeman v. Monsanto Co.*, 997 F.3d 941 (9th Cir. 2021).

[182] *Preamble* at 3.

[183] European Food Safety Authority, *Conclusion on the peer review of the pesticide risk assessment of the active*

EFSA Executive, to demand corrections.[184] When the EFSA refused the Wild demands, Christopher Portier and over 80 other "authors," including Portier's brother, mobbed the EFSA in a journal commentary.[185] Most of the authors had no reasonable pretense to expertise about the controversy they were attempting to create. Many of the authors were current or former IARC officials, as well as members of the Collegium Ramazzini. Only three authors provided a disclosure of conflicts of interest (Christopher Portier, Martyn Smith, Dennis Weisenburger), by acknowledging that they had engaged in "providing advice to a US law firm involved in glyphosate litigation." Given the absence of information about the name of the law firm, the financial arrangements for providing the "advice," the clients of the firm, and the benefit to the firm and its clients monetarily if the authors' position prevailed, some observers might well find their disclosure anemic. As for the numerosity of the authors, Dr. Url and the EFSA may have found support and comfort in the wisdom of Albert Einstein, who reportedly responded to criticisms of his work in physics, published in a pamphlet with 100 authors,[186] with the quip, that "[i]f I were wrong, then one would have been enough."[187]

Portier and his colleagues at the Collegium Ramazzini and IARC may have been surprised by the willingness of regulators at EFSA and other agencies around the world to resist IARC pronouncements and bullying on glyphosate. Historically, IARC classifications result in regulatory agencies, especially in the United States, to walk in lock step with IARC's views on carcinogen classification. Even without a letter and editorial writing campaign, IARC classifications pressure regulators to label substances as carcinogens, regardless of how equivocal the evidence. And the lawsuit industry stands ever ready to jump on the opportunity to cash in on a new classification.

A. Bending Science to Fit IARC Classifications

IARC classifications also pressure scientists to interpret their data to line up with IARC's classifications. The pressure can be subtle but insidious. The environmental and occupational medicine communities are riveted by fear that they may be seen as too supportive or adjacent to

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substance glyphosate, 13 EFSA J. 4302 (2015).

[184]Wild letter (Feb. 5, 2016), https://perma.cc/9PCP-MM82.

[185]Christopher J. Portier, Bruce K. Armstrong, Bruce C. Baguley, Xaver Baur, Igor Belyaev, Robert Bellé, Fiorella Belpoggi, Annibale Biggeri, Maarten C Bosland, Paolo Bruzzi, Lygia Therese Budnik, Merete D Bugge, Kathleen Burns, Gloria M. Calaf, David O. Carpenter, Hillary M. Carpenter, Lizbeth López-Carrillo, Richard Clapp, Pierluigi Cocco, Dario Consonni, Pietro Comba, Elena Craft, Mohamed Aqiel Dalvie, Devra Davis, Paul A. Demers, Anneclaire J. De Roos, Jamie DeWitt, Francesco Forastiere, Jonathan H. Freedman, Lin Fritschi, Caroline Gaus, Julia M Gohlke, Marcel Goldberg, Eberhard Greiser, Johnni Hansen, Lennart Hardell, Michael Hauptmann, Wei Huang, James Huff, Margaret O. James, C. W. Jameson, Andreas Kortenkamp, Annette Kopp-Schneider, Hans Kromhout, Marcelo L. Larramendy, Philip J. Landrigan, Lawrence H. Lash, Dariusz Leszczynski, Charles F. Lynch, Corrado Magnani, Daniele Mandrioli, Francis L. Martin, Enzo Merler, Paola Michelozzi, Lucia Miligi, Anthony B. Miller, Dario Mirabelli, Franklin E. Mirer, Saloshni Naidoo, Melissa J. Perry, Maria Grazia Petronio, Roberta Pirastu, Ralph J. Portier, Kenneth S. Ramos, Larry W. Robertson, Theresa Rodriguez, Martin Röösli, Matt K Ross, Deodutta Roy, Ivan Rusyn, Paulo Saldiva, Jennifer Sass, Kai Savolainen, Paul T. J. Scheepers, Consolato Sergi, Ellen K. Silbergeld, Martyn T. Smith, Bernard W. Stewart, Patrice Sutton, Fabio Tateo, Benedetto Terracini, Heinz W. Thielmann, David B. Thomas, Harri Vainio, John E. Vena, Paolo Vineis, Elisabete Weiderpass, Dennis D. Weisenburger, Tracey J Woodruff, Takashi Yorifuji, Il Je Yu, Paola Zambon, Hajo Zeeb & Shu-Feng Zhou, *Differences in the carcinogenic evaluation of glyphosate between the International Agency for Research on Cancer (IARC) and the European Food Safety Authority (EFSA)*, 70 J. EPIDEM. CMTY. HEALTH741 (2016).

[186]HUNDERT AUTOREN GEGEN EINSTEIN (1931).

[187]STEPHEN HAWKING, A BRIEF HISTORY OF TIME 193 (10th Anniv. Ed. 1996) (quoting Einstein, without source).

positions that industry might favor. Even without accusing those who disagree as having ethical conflicts and using unscientific procedures, IARC, through its classifications, can change the scientific community's opinion on an agent, virtually overnight. The unsafe influence is especially apparent for occupational and environmental exposures, for which many members of the public health community are driven by white-hat bias to avoid appearing too supportive of any industry or industry position.

Crystalline silica presents a disturbing case study. In 1996, an IARC working group classified crystalline silica as a human carcinogen, group 1. Despite IARC's claims that its classifications represent scientific consensus, the vote on silica was a simple majority, reached with difficulty.[188] Historically, because crystalline silica was known to cause a pneumoconiosis silicosis, researchers asked whether silica or silicosis might cause lung cancer by analogy to asbestos minerals that cause a pneumoconiosis, asbestosis, and lung cancer. After World War II, as public health shifted its focus from infectious to chronic disease, epidemiology became the dominant scientific tool to study probabilistic risk factors.[189] From 1945 through the early 1990s, the public and occupational health professional communities looked closely at whether silica caused lung cancer. They found the evidence unconvincing, and even exculpatory.[190] Up until the IARC working group meeting in 1996, most epidemiologists and public health specialists did not regard crystalline silica, a ubiquitous mineral that makes up a large percentage of the earth's surface, as a cancer hazard.

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[188]Corbett McDonald, *Silica and Lung Cancer: Hazard or Risk*, 44 ANN. OCCUP. HYG. 1,2 (2000) (Prof. McDonald was a member of the 1996 silica working group; the IARC code of secrecy has tightened in the years since 1996).

[189]*See* Colin Talley, *et al.*, *Lung Cancer, Chronic Disease Epidemiology, and Medicine, 1948 – 1964*, 59 J. HIST. MED. & ALLIED SCI. 329 (2004).

[190]*See* Cuyler Hammond & W. Machle, *Environmental and Occupational Factors in the Development of Lung Cancer*, Ch. 3, in E. Mayer & H. Maier, *Pulmonary Carcinoma: Pathogenesis, Diagnosis, and Treatment* at 41-61 (1956); Gerret Schepers, "Occupational Chest Diseases," Chap. 33, at 455, in A. FLEMING, *et al.*, eds., MODERN OCCUPATIONAL MEDICINE (2d ed. 1960) ("Lung cancer, of course, occurs in silicotics and is on the increase. Thus far, however, statistical studies have failed to reveal a relatively enhanced incidence of pulmonary neoplasia in silicotic subjects."); Joseph Wagoner, *et al.*, *Unusual Cancer Mortality among a Group of Underground Metal Miners*, 269 NEW ENGL. J. MED. 284, 287 (1963) ("Since epidemiologic studies have demonstrated that silicosis and occupational exposure to free silica dust *do not predispose to the development of cancer* of the respiratory tract, attention must be paid to specific components of the ore that have been suspected of carcinogenic activity in man.") (emphasis added; internal citations omitted); Richard Doll, *The Statistical Approach to Industrial Lung Cancer*, in Douglas Teare & Joan Fenning, eds., SOME ASPECTS OF CARCINOMA OF THE BRONCHUS AND OTHER MALIGNANT DISEASES OF THE LUNG 5, 14 (1966) ("I should like to draw your attention to the absence of a risk, although one has been looked for very carefully, among men with silicosis."); WILHELM HEUPER, OCCUPATIONAL AND ENVIRONMENTAL CANCERS OF THE RESPIRATORY SYSTEM at 2-6 (1966) (Hueper was chief of the NCI) ("The bulk of the available epidemiologic evidence on the association of silicosis and lung cancer supports the view of a mere coincidental role of silicosis in this combination.");Harriet Hardy, *Current Concepts of Occupational Lung Disease of Interest to the Radiologist*, 2 SEM. ROENTGENOL. 225, 231-32 (1967) ("cancer of the lung is not a risk for the silicotic. It is a serious risk following asbestos exposure ... ."); Kaye Kilburn, Ruth Lilis & Edwin Holstein, *Silicosis*, in MAXCY-ROSENAU, ed., PUBLIC HEALTH AND PREVENTIVE MEDICINE at 606 (11th ed. 1980) ("Lung cancer is apparently not a complication of silicosis."); W. Raymond Parkes, *Diseases Due To Free Silica*, Chap. 7, OCCUPATIONAL LUNG DISORDERS 157 (2d ed. 1982) ("Bronchial carcinoma occasionally occurs in silicotic lungs but there is no evidence of a causal relationship between it and silicosis or siliceous dusts... ."); U.S. Surgeon General, THE HEALTH CONSEQUENCES OF SMOKING: CANCER & CHRONIC LUNG DISEASE IN THE WORK PLACE, Chap. 8, at 348 (1985) ("the evidence does not currently establish whether silica exposure increases the risk of developing lung cancer in men."); NIOSH Silicosis and Silicate Disease Committee, *Diseases Associated With Exposure to Silica and Non-fibrous Silicate Minerals*, 112 ARCH. PATH. & LAB. MED. 673, 711 (1988) ("The epidemiological evidence at present is insufficient to permit conclusions regarding the role of silica in the pathogenesis of bronchogenic carcinoma.").

For its 1996 working group on silica, IARC appointed an epidemiologist, Harvey Checkoway, as its chair. Checkoway had just published a study that found a small but statistically significantly increased risk of lung cancer among men who mined and processed diatomaceous earth, which involves exposure to crystalline and amorphous silica, as well as to asbestos in processing.

Based upon the 1996 working group, IARC published a monograph on crystalline silica in 1997. Although IARC classifications typically do not qualify a cancer hazard by exposure circumstances, the silica monograph identified carcinogenicity in some, not all, occupational exposures.[191] The IARC classification, however, was an unqualified group 1. The consequence of this classification was a sandstorm of fear mongering about every possible exposure, from environmental exposure at beaches, to exposure to sand spread on icy roads, to neighborhood exposures from sand quarries, and beyond. Predictably, California's regime of warnings required that bags of play sand for sandboxes carry a cancer warning.

Scientific opinion on the carcinogenicity of silica changed virtually overnight. Leading textbooks, which had questioned the carcinogenicity of silica before 1997, suddenly endorsed the IARC conclusion after 1997.[192] Only a few intrepid souls declined to acquiesce in the new dogma.[193]

Remarkably, scientists started to skew their interpretations of data in incomprehensible ways to make the data and analyses fit the new paradigm. One study published in the aftermath of the IARC 1996 silica classification is instructive for the power of IARC groupthink. The authors studied mortality from various diseases, including lung cancer, using death certificates of certain occupations in the United States, never the best way to document exposure. When the authors analyzed whether there was any risk among the workers who had *both* silicosis (which guaranteed that there had been credible exposure of sufficient dose and duration to cause the non-malignant pneumoconiosis) and lung cancer compared with a rate of lung cancer among non-silicotic exposed workers, they found that the mortality odds ratio was 0.70, a 30% *reduction* in lung cancer risk that was highly statistically significant.[194]

The authors also conducted many other comparisons (without adjusting statistically for multiple analyses). Unlike the silicosis analysis, where substantial occupational exposure was demonstrated by the presence of pneumoconiosis, and which showed a 30% reduction in lung cancer risk, the authors' other comparisons were based on estimated silica exposure implied from the trades or occupations listed on death certificate. Only when the authors compared lung cancer mortality between workers ever exposed to silica (with crudely estimated medium, high, and super-high categories) did the authors find a 13% increased risk of lung cancer mortality in some, but not

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[191]IARC MONOGRAPH no. 68, at 210-11 (1997).

[192]*Compare* Hans Weill, Robert Jones & W. Raymond Parkes, *Silicosis and related diseases*, chap. 12, at 316, in W. RAYMOND PARKES, OCCUPATIONAL LUNG DISORDERS (3d ed. 1995) ("It may be reasonably concluded that the evidence to date that occupational exposure to silica results in excess lung cancer risk is not yet persuasive.") with David Rees & Jill Murray, *Silica*, chap. 18, at 193, in ANTHONY TAYLOR, *et al.*, eds., PARKES' OCCUPATIONAL LUNG DISEASE (4th ed. 2017) ("Lung cancer is convincingly linked to quartz and cristobalite exposure.").

[193]*See, e.g.*, Bryan Corrin, PATHOLOGY OF THE LUNGS at 289 (1st ed. 2000); Patrick A. Hessel, *supra* note 115 at 717-18 ("The data demonstrate a lack of association between lung cancer and exposure to crystalline silica in human studies. Furthermore, silica is not directly genotoxic and has been to be a pulmonary carcinogen in only one animal species, the rat, which seems to be an inappropriate model for assessing particulate carcinogenesis in humans.").

[194]Geoffrey M. Calvert, *et al.*, *Occupational silica exposure and risk of various diseases: an analysis using death certificates from 27 states of the United States*, 60 OCCUP. & ENVT'L MED. 122, 125 (2003).

all subgroups. The authors had no smoking histories for their study subjects, and thus they had no way to evaluate this small increased risk of lung cancer (13%) given the potential confounding effect of smoking, which can increase risk 2,000 to 3,000%. On this tortured evidentiary basis, the authors, from the National Institute of Occupational Safety & Health, claimed that their study corroborated the association between crystalline silica exposure and lung cancer.

Silicosis litigation has a long history in the United States. IARC's 1996 re-classification of crystalline silica sparked a recrudescence of litigation with both silicosis and lung cancer claims, thus illustrating the tortogenic potency of IARC classifications. In the case of crystalline silica, however, most of the IARC-inspired tort cases were dismissed by a federal judge who observed that the plaintiffs' counsel had fraudulently mass produced the lawsuits by refilling claims previously filed as asbestos claims.[195]

B. Regulatory Implications

IARC classifications are also consequential outside courtrooms. The media give IARC classifications a great deal of attention, often uncritical, and without regard to IARC's technical, idiosyncratic methods and definitions. In the private sector, the American Cancer Society (ACS) publishes lists of "known and probable" carcinogens, largely taken from IARC, but without the contextual and qualifying information given in the *Preamble*.[196] Group 1 carcinogens, which IARC takes to be established by "sufficient" evidence, receive an epistemic upgrade to *known* carcinogens, in ACS parlance. And the ACS lists IARC 2A carcinogens as probable carcinogens without the qualification of IARC's unscientific definition of probable.

IARC has deep ties to United States regulatory agencies. For many years, the National Cancer Institute (NCI) has been the major funder of the IARC monograph program.[197] The NCI participates in the IARC process and frequently relies on IARC classifications.[198] Most environmental and occupational exposure regulatory rule-makings reference and are heavily influenced by IARC classifications. In theory, the National Toxicology Program (NTP) of the National Institute of Environmental Health Sciences evaluates carcinogenicity independently of IARC, but IARC classifications are extremely influential, and the NTP frequently cites IARC determinations.

OSHA requires manufacturers and sellers to provide so-called material safety and data sheets for products and raw materials. The sheets must include NTP and IARC carcinogen classifications.[199] Beyond OSHA, IARC's work has wide implications for state and federal regulatory agencies. In large part, these governmental agencies are engaged in risk assessment and

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[195]*In re Silica Prods. Liab. Litig.*, 398 F. Supp. 2d 563 (S.D. Tex. 2005); *see* Nathan A. Schachtman, *Silica Litigation: Screening, Scheming & Suing*, WASH. LEG. FOUND. WORKING PAPER (Dec. 2, 2005), https://perma.cc/GE4J-PXUZ.

[196]ACS, *Known and Probable Human Carcinogens* (Aug. 1, 2024), https://perma.cc/US42-VQFY.

[197]Neil Pearce, *et al.*, IARC Monographs: 40 Years of Evaluating Carcinogenic Hazards to Humans, 123 ENVT'L HEALTH PERSP. 507, 509 (2015) (noting that "[t]he IARC Monograph Programme is mainly funded by the U.S. National Cancer Institute... .").

[198]Vincent James Cogliano, *et al., The Science and Practice of Carcinogen Identification and Evaluation*, 112 ENVT'L HEALTH PERSP. 1269 (2004).

[199]OSHA, *Hazard Communication Standard*, 29 C.F.R. Part 1910, 89 *Fed. Reg.* 44144 (May 20, 2024); OSHA, Inspection Procedures for the Hazard Communication Standard; OSHA Instruction, Directive No. CPL 02-02-079 (effective July 9, 2015), https://perma.cc/BW33-LQ88.

risk regulation. Exposure to an IARC classified carcinogen does not mean that everyone exposed will develop cancer. Close to 40% of the American population will develop cancer in their lifetimes, and the rate would be much higher if cancers routinely found at autopsy (thyroid, prostate, etc.) were included.[200] IARC's hazard classification system does not answer the important question whether people in the population actually have a greater risk of any specific cancer than they would have had absent the exposure to the agent; nor does it quantify the increased risk, which is virtually always a function of the exposure amount and duration.

The process of risk assessment begins with a hazard classification, such as provided by an IARC group 1 classification. For IARC classifications below group 1, for exposures to agents in groups 2A, 2B, and 3, risk assessments should not get off the ground because agents in group 2A provide no quantitative probability that they even cause cancer in humans, and groups 2A and 3 are cloaked in possibilities and indeterminacies. Even group 1 classifications are reached, not with scientific inference, but with precautionary principle zeal, and disregard for thresholds of exposure below which no cancer would be expected. Regulators often, however, allow risk assessors to play various "what ifs"—if humans were rats, and if rat carcinogens were human carcinogens, and if high-dose rat carcinogens caused cancer in humans at low doses, if there were no thresholds or the absence of any no observable effect levels, and so forth. Risk assessment is a risky business with many sources of error that can and do arise when extrapolations are made between species or exposure levels, and when contrary to fact assumptions are made, such as there are no bodily defenses to the development of cancer from external causes.

C. Prop 65

Probably the most dramatic and direct consequence of an IARC classification occurs in California, because of a law popularly known as Prop 65. California voters approved Proposition 65 in 1986. The referendum led to California's adoption of the Safe Drinking Water and Toxic Enforcement Act of 1986, and the creation of a regulatory bureaucracy under California's Office of Environmental Health Hazard Assessment (OEHHA). The OEHHA has created an elaborate regulatory scheme that imposes "right to know" warning requirements upon what it takes to be cancer or reproductive hazards. The statute and related regulations, known simply as Prop 65, mandate that

> No person in the course of doing business shall knowingly and intentionally expose any individual to a chemical known to the state to cause cancer or reproductive toxicity without first giving clear and reasonable warning to such individual... .[201]

Some readers might discern an anti-business animus in the statutory targeting of "doing business," with a warning requirement that would appear not to apply to a municipality's or the state's activities.

In a bold exhibition of epistemic arrogance, California claims to know that any substance listed as a human or animal carcinogen by IARC does cause cancer and is subject to Prop 65. The OEHHA specifies that, at a minimum, businesses must warn about all group 1 agents, all group 2A agents based upon "sufficient" animal evidence, and group 2B agents but only if based upon

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[200]Nat'l Cancer Institute, Surveillance, Epidemiology, and End Results Program, Lifetime Risk of Developing Cancer, https://perma.cc/T8X9-REFL.

[201]Cal. Health & Safety Code § 25249.5 (2024).

sufficient animal evidence. So California knows that "probable" carcinogens, when probable does not really mean probable, and even some possible carcinogens, will cause cancer.[202] By agency fiat, "[t]he absence of a carcinogenic threshold dose shall be assumed and no-threshold models shall be utilized."[203]

Prop 65 takes IARCs classifications further than any rational inference by assuming that hazard equals risk. Consequently, enforcement actions under Prop 65 need allege only that the product at issue has some of the listed chemical in it, regardless of the amount and whether the chemical poses a demonstrable harm to any human being. The California regulatory regime manages to engage simultaneously in both fearmongering and in saturating the public's sensitivity to warnings by requiring warnings everywhere. Labeling an agent as carcinogenic to humans without contextual details of whether anticipated or experienced exposures cause cancer predictably misleads people, with conflation of warnings about truly dangerous chemicals and exposures as innocuous as sand in a child's sandbox.

To make a bad regulatory environment worse, the regulations encourage the growth of a predatory lawsuit industry. Prop 65 regulations have created a racket by allowing private parties to commence enforcement actions "in the public interest." These actions can be extremely lucrative; private parties may retain 25 percent of all penalties recovered, which may include up to $2,500 for each violation, for each day, as well as attorneys' fees.[204] These "bounty hunter" provisions could be the reason Collegium Ramazzini members Martyn Smith and Carl Cranor created CERT, which has been at the heart of many such lawsuits. Despite revelations of abuses and diversion of recoveries to pay the legal fees of the bounty hunters,[205] the Prop 65 regime, tied to IARC classifications, has been in place for 40 years. The most serious harm inflicted by the California regulations is the deliberate misrepresentation of what can reasonably known about the causes of cancer.

IX. POLICY QUESTIONS AND CONCLUDING THOUGHTS

The IARC monograph process is broken. What IARC advertises as scientific assessments of carcinogenicity are often policy-laden pronouncements reached by non-scientific precautionary principle reasoning. The precautionary approach manifests in the semantic distortions of IARC's classifications, in the tortured distinction between hazard and risk, and in the substantive distortions created by IARC's contrived, unscientific algorithms and checklists for the purpose of declaring more carcinogens. The precautionists have their thumbs on the scales of science, and they have usurped policy decisions from the democratic process.

Governmental regulators, with appropriate statutory authorization, may choose to undertake precautionary measures. There is nothing inherently wrong about regulating in the face of uncertainty, but it is policy, not science. Regulators and scientists should be honest about when they are driving faster than their headlights illuminate, and they should acknowledge the costs imposed by their policy choices rather than claiming support in "science," and demonizing

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[202]27 Cal. Code Regs. § 25904 (Chemical Listings by Reference to California Labor Code § 6382(b)(1) (West 2024).

[203]27 Cal. Code Regs. § 25703(5).

[204]CAL. HEALTH & SAFETY CODE § 25249.12(d); CAL. CODE CIVIL PROC. § 1021.5.

[205]*See* Anthony T. Caso, *Bounty Hunters and the Public Interest - A Study of California Proposition 65*, 13 J. FEDERALIST SOC'Y PRAC. GROUPS 68 (2012); Carole Levine, *Where Are the Millions This Nonprofit Made from Cancer Warnings?* NON-PROFIT QUART. (June 11, 2019), https://perma.cc/PAG2-UAVU.

dissidents as science deniers.

IARC, in propagating a contrived system of carcinogen classifications, has not been candid about its methods and procedures. The semantic and conceptual confusions of the IARC classification scheme create public misunderstanding, feed media fear mongering, and fuel a hyper-vigilant lawsuit industry.

The IARC monograph and classification project has become controversial in the scientific community. The glyphosate working group's classification has forced scientists and policy experts to look more closely at IARC's methodology and its inner working. The resulting views have not been flattering.[206] The glyphosate and other IARC controversies have thus had the salutary effect of encouraging scientists to question the underlying premises of IARC classifications, such as conflating hazard and risk, ignoring potency, and failing to define real-world exposure circumstances in which humans, not rats, have actual risks of specific cancers.[207]

By the end of the 20th century, the need for the IARC classifications had waned, as the methods of systematic review and meta-analyses had evolved significantly, and had become more widely standardized and practiced. The informational ecosystem has changed dramatically since IARC's founding. The studies considered in systematic reviews are now widely accessible through on-line publishing. IARC classifications, reached after several days of meetings, cannot compete with the quality of state-of-the-art systematic reviews that are prepared with months and years of deliberation and care.

Scientists aligned with IARC and its precautionary approach have trumpeted the importance of IARC to the American regulatory regime.[208] While they are correct that IARC monographs receive close attention, and often slavish acceptance in federal and state regulatory agencies, they are wrong that IARC is necessary or helpful to the American regulatory process.

The reviews undertaken by IARC working groups are redundant of, and often inferior to, the systematic reviews now published in the open scientific literature. The IARC process is also redundant of efforts already made by federal agencies. In the United States, the National Toxicology Program is an interagency group, supported by the National Institute of Environmental Health Sciences, the National Institute for Occupational Safety and Health of the Centers for Disease Control and Prevention, and the National Center for Toxicological Research of the Food and Drug Administration. Congress requires the NTP to prepare a science-based public health document, "The Report on Carcinogens,"[209] which includes classifications of carcinogens. The EPA conducts yet another, different carcinogen classification system.[210] While there is much that could be improved in the domestic classification systems, they have the advantage of being produced by regulators subject to open meeting, public comment, and freedom of information requirements. IARC classifications, on the other hand, are created in a secretive process, hidden behind closed

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[206]Kai Kupferschmidt, *High-Profile Cancer Reviews Trigger Controversy*, 352 SCI. 1504, 1504 (2016).

[207]Alan R. Boobis, *et al.*, *Classification schemes for carcinogenicity based on hazard identification have become outmoded and serve neither science nor society*, 82 REG. TOXICOL. PHARMACOL. 158 (2016); John E. Doe, *et al.*, *A new approach to the classification of carcinogenicity*, 96 ARCH. TOX. 2419 (2022).

[208]Neil Pearce, *et al.*, *IARC monographs: 40 years of evaluating carcinogenic hazards to humans*, 123 ENVT'L HEALTH PERSP. 507, 508 (2015).

[209]*See* 15th Report on Carcinogens, NAT'L TOXICOLOGY PROGRAM, U.S. DEP'T HEALTH & HUM. SERVS., (last updated Dec. 21, 2021), https://perma.cc/CA3B-6KEL.

[210]*See Risk Assessment for Carcinogenic Effects* (Oct. 9, 2025), https://perma.cc/H5W3-YKKY.

doors, protected from public inspection and comment, and issued without outside scientific peer review. Along with the published systematic reviews in the scientific journals, the multiple federal classification systems render IARC's process redundant and otiose. Perhaps most important, the efforts of federal agencies are subject to legislative oversight and financial control.

Delegation of regulatory and legislative fact-finding to secretive IARC working groups prevents public comment and undermines democratic control over carcinogen policy. Over 50 years ago, Congress passed legislation designed to curb the secrecy of non-governmental committees advising federal agencies.[211] The Federal Advisory Committee Act (FACA) imposes significant disclosure and transparency requirements on advisory committees either "established or utilized by" federal agencies.[212] One legal observer has argued that FACA's remedial goals should apply to IARC working groups to render their work open and transparent, given how close the National Cancer Institute and other agencies work with IARC, and how interwoven IARC determinations are interwoven with federal regulations.[213] Congress designed the FACA statute "to cure specific ills, above all the wasteful expenditure of public funds for worthless committee meetings and biased proposals... ."[214] Indeed, one of the principal goals of FACA was "to enhance the public accountability of advisory committees," an area in which IARC is clearly deficient.[215] Although judicial precedents are unclear in supporting the application of FACA to IARC's working group meetings, the remedial spirit of the legislation cries out for some legislative or executive curb on delegations to the special interests at work in IARC, and their undue influence over federal decision makers.

IARC rationalizes its secrecy oaths and closed-door deliberations by claiming fear of "outside" influences. Reading between the lines, we can see that what IARC fears most is the influence of manufacturing concerns and their ability to put forward persuasive data and scientific inferences. By closing the doors to the IARC process, the agency has ensured that other groups, such as the Collegium Ramazzini and its lawsuit industry allies will dominate the secret deliberations on carcinogen classifications. While IARC officials complain about criticism from manufacturing industry stakeholders, they openly embrace scientists who serve as litigation expert witnesses and consultants for the lawsuit industry. Tellingly, IARC does not regard as dangerous special interests such as the vast influence of the Collegium Ramazzini, and its fellows' ideological commitment to the precautionary principle.

Whatever else the IARC monograph and classification regime tries and fails to accomplish, it succeeds at having several untoward consequences. The IARC classification process clearly is a known litogen and tortogen, with a clear ability to create personal injury and environmental litigation. The lawsuit industry, made up of lawyers who pursue such litigation, along with their litigation lenders and funders, and aligned with environmental advocacy groups constitute a loud and persistent, rent-seeking, support group for the IARC classification process, with its precautionary principle leanings.

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[211]Federal Advisory Committee Act, Pub. L. No. 92–463, 86 Stat. 770 (1972), 5 U.S.C. § 1001 *et seq.*

[212]5 U.S. Code § 1001(2)(a).

[213]David B. Fischer, *The IARC Monographs Program and the Federal Advisory Committee Act - Never the Twain Shall Meet?* 44 WM. & MARY ENVT'L L. & POL'Y REV. 531, 535 (2020).

[214]*Public Citizen v. U.S. Dep't of Justice*, 491 U.S. 440, 441 (1989) (interpreting FACA).

[215]*Id.* at 459.

In February 2018, the House Congressional Committee on Science, Space, & Technology conducted oversight hearings on IARC, with special reference to its controversial glyphosate classification.[216] The United States funds IARC mostly through the National Institutes of Health. Scientists appeared and testified about IARC's methodology generally and its glyphosate classification specifically. Several of these scientists raised serious questions about the lack of scientific integrity and rigor in IARC procedures and classifications. A staff scientist from the environmental advocacy group, Natural Resources Defense Council, which seeks to regulate by litigation, appeared to decry industry attacks on "independent scientists," and to praise IARC. Although IARC has accepted federal funding since its founding, agency officials refused to participate meaningfully in the hearing.

Instead of attending and testifying under oath, IARC Director Christopher Wild sent a letter in which he falsely asserted that IARC working groups "make use of the latest scientific data and methodologies" and are "transparent and open to scrutiny."[217] Wild gave a self-serving, biased assessment of IARC procedures as transparent and free from conflicts of interests. Under the circumstances of IARC's pattern of behavior, Wild's decision not to testify in person and to submit to examination was sadly all too understandable.

After the hearing, the Competitive Enterprise Institute issued lengthy statements in support of ending financial support for IARC.[218] Others have argued that the United States should remain involved in the funding and staffing of IARC and try to reform the agency from within.[219] Given IARC's denial of its conflicts and non-transparent procedures, and its willingness to attack the messengers who document its failings, the prospect for reform seems dismal.

Defunding or withdrawing from IARC is not about saving money; it is about making a statement that IARC is unnecessary and counter-productive to both the scientific and regulatory enterprises. Some have argued that defunding IARC would inhibit our ability to work within that agency to reform it. Several considerations make reform unlikely if not impossible. First, our laws accord IARC and its carcinogen classifications undue prestige and influence. Nothing short of repudiating IARC's classification program in its current incarnation will undo the ill effects of empowering IARC. Second, IARC is a secretive international organization with financial support from many nations. Although scientists from the United States have had leadership roles in IARC, the government of the United States has a minority stake in the funding of the agency. There are simply too many independent governments who have acquiesced in the IARC mission as it has come to be. Third, and most important, IARC has been captured by other special interests, represented by groups such as the Collegium Ramazzini. When we add to these considerations, the redundancy, inaccuracy, and unscientific nature of IARC classifications, the simplest solution is to abandon the entire enterprise of IARC hazard classifications.

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[216] *In Defense of Scientific Integrity: Examining the IARC Monograph Programme and Glyphosate Review*: Hearing Before the House Comm. on Science, Space & Tech., 115 Cong. Serial No. 115–46 (Feb. 6, 2018).

[217] Christopher Wild, *supra* note 131.

[218] *See* Angela Logomasini, *U.S. Should Stop Funding the International Agency for Research on Cancer: Misleading Classifications Promote Counterproductive Bans and Adverse Market Impacts*, CEI (Sept. 18, 2018), https://perma.cc/B9G6-3JYL; Angela Logomasini, *End All National Institutes of Health Grants to the World Health Organization's International Agency for Research on Cancer*, CEI (June 17, 2020), https://perma.cc/VRW3-L8T3.

[219] *See, e.g.*, Julie E. Goodman, *et al.*, *Recommendations for further revisions to improve the International Agency for Research on Cancer (IARC) Monograph program*, 113 REG. TOXICOL. & PHARMACOL. 104639 (2020).

In March 2026, the U.S. National Institutes of Health announced that it was cutting ties with IARC.[220] Assessing this announcement is complicated by its lack of specificity. In addition to the monograph classification program, IARC engages in other activities, such as standardizing diagnostic criteria for different cancers, and measuring epidemiologic cancer trends around the world. The problems that have transformed the monograph working groups are not necessarily shared by the IARC's other activities. Based upon the limited information available on this announcement, cutting all ties to IARC seems like an overly broad response to the problems created by the monograph program and its carcinogenicity classifications.

[220] Jocelyn Kaiser, *United States cuts ties with WHO's cancer research arm*, 391 SCIENCE 1197 (Mar. 19, 2026).

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