CGHA

When Evidence Becomes Authority

IARC hazard classifications academic influence and litigation

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Abstract

IARC hazard classifications can influence research priorities, public communication, regulation and litigation far beyond the formal question they answer. This paper examines that transmission through five illustrative comparisons and selected policy cases. Differences between institutions can arise from evidence dates, agent definitions, study selection, analytical methods or mandates. They cannot be measured reliably by counting unlike labels. The institutional hypothesis is that an authoritative classification can reduce the cost of building a campaign or common litigation theory. Establishing improper influence requires separate evidence of conduct, funding and causation; a classification or disagreement alone is insufficient.

Main conclusion

IARC identifies carcinogenic hazards. It does not decide what risk a person faces at a specified exposure, which intervention is proportionate, or whether a product caused an individual claimant’s disease. Its classifications can nevertheless influence all three judgments. A widely recognized category can become a premise in research, public communication, regulatory action and litigation. The accountability question is whether each later use preserves the limits of the original assessment.

That function is especially valuable to litigators. Mass litigation requires a scientific proposition capable of being reused across thousands of claims. An IARC category can help provide that common platform even though it says nothing by itself about a claimant's dose, latency, disease subtype or alternative causes. A classification need not win a case to create leverage. If it helps claims survive long enough to attract finance, generate discovery costs and expose a company to a small number of exceptional verdicts, it can alter the settlement value of the whole inventory.

This paper develops an institutional hypothesis, rather than a finding of coordinated misconduct. Scientific assessments, research funding, advocacy and litigation may reinforce one another, but the existence, direction and consequences of those relationships require documentary evidence. Critical commentary can identify leads; it cannot substitute for grant records, study methods, disclosures and court decisions. The climate comparison extends the question to research whose anticipated legal use may influence demand for particular models or estimates.

This is not an argument that every IARC classification is wrong, that every claimant is undeserving, or that corporate evidence should be trusted without scrutiny. Corporate ghostwriting, suppression and selective publication require the same examination. The argument is that no source of influence should receive an ethical exemption. When hazard is translated into ordinary-use risk and then individual causation, every inferential step should be visible and proved.

The institutional switch

IARC is the World Health Organization’s specialized cancer agency. Its Monographs Programme identifies carcinogenic hazards, while exposure-specific risk assessments ask about the likelihood and magnitude of

harm under stated conditions. Courts ask further questions under the applicable law, including duty, admissible evidence, exposure and causation. The required proof varies by jurisdiction and claim. None of these institutions can simply borrow another's answer without identifying the proposition it actually supports (IARC, Preamble, 2019).

Decision maker	Question	What the answer does not establish
IARC	Can the agent cause cancer under some circumstances?	Risk at ordinary exposure, a legal duty to warn, or claimant specific causation
Regulator	Does expected exposure create an unreasonable population risk?	Whether one claimant's disease was caused by the product
Civil court	Did this exposure cause this injury under the governing legal standard?	A general scientific consensus applicable beyond the record before the court

The categories are easy to state and routinely blurred in practice. 'Probably carcinogenic' sounds to a lay reader like a prediction about danger in ordinary use. In IARC's system it is a classification of the strength and pattern of evidence. The gap matters because civil litigation is not financed, recruited or tried in the language of technical institutional distinctions. A globally recognized cancer label is short, memorable and authoritative. Dose reconstruction, latency, confounding and alternative causes are slower and harder to explain.

How IARC produces a classification

IARC's influence begins before a Working Group meets. The Monographs Programme does not examine a representative sample of all products and exposures. It selects agents suspected of causing cancer for which human exposure occurs and enough evidence exists to conduct an evaluation. The public, scientists, health agencies and other organizations may nominate agents. An international Advisory Group recommends priorities, after which IARC chooses the programme of meetings. Selection therefore signals that an agent is already considered scientifically and publicly consequential, even though nomination is not evidence of carcinogenicity.

For each Monograph, IARC convenes international experts and assigns them to review evidence on exposure, cancer in humans, cancer in experimental animals and mechanisms. The modern Preamble emphasizes systematic evidence identification, evaluation of study quality, integration of three evidence streams and consensus classification. Members with disqualifying conflicts are not meant to serve as Working Group members; invited specialists and observers may participate under more limited roles. Draft chapters are prepared before the meeting and finalized through deliberation. The Working Group then assigns one of four classifications ranging from Group 1, carcinogenic to humans, to Group 3, not classifiable (IARC, Preamble, 2019).

Stage	Formal process	Source of authority	Accountability question
Priority setting	Public nominations and expert advisory priorities	Agenda is presented as expert selected	Who nominates agents and which questions never reach review?
Expert selection	Working Group members specialists and observers with declared interests	International expertise and formal independence	Are intellectual institutional and future litigation interests captured?

Stage	Formal process	Source of authority	Accountability question
Evidence review	Published and publicly available human animal and mechanistic evidence	Systematic synthesis under a stable Preamble	Does public availability privilege the academic literature over confidential regulatory studies?
Consensus	In person deliberation and categorical judgment	A single collective conclusion replaces dispersed uncertainty	Are disagreements ranges and sensitivity to assumptions sufficiently visible?
Communication	Lancet Oncology summary press release full Monograph and classification list	WHO affiliation and concise global label	Does the headline preserve the hazard risk distinction?

These procedures are more disciplined than ordinary advocacy reports, and their strengths explain IARC's standing. The concern is not that expert judgment can be eliminated. It is that several consequential judgments are compressed into a categorical label: what to review, which studies are usable, how study limitations are weighted, how mechanistic evidence is integrated, and whether remaining uncertainty fits Group 2A, Group 2B or Group 3. Once the label is issued, downstream users rarely reproduce those judgments. They cite the category.

The academic authority loop

An authoritative synthesis can influence subsequent research by identifying questions, mechanisms and gaps. Later authors may cite the classification to explain why an agent warrants study, and funders may use it to prioritize work. This creates a possible feedback loop: later summaries can appear to provide independent confirmation even when their introductory claim ultimately derives from the same Monograph. Whether this occurs, and whether contrary findings receive less attention, requires citation and funding analysis.

This is a normal feature of scientific agenda formation, not proof of misconduct. Authoritative syntheses properly direct attention to unresolved risks. But it creates a feedback problem. IARC evaluates the published literature; its classification stimulates a new published literature; and that literature is later treated as further evidence supporting the importance of the classification. The feedback is strongest when funders, journals and researchers reasonably prefer positive, policy-relevant questions. The result can be path dependence: once an agent enters the Monographs system and receives a prominent category, research resources accumulate around possible harm while comparative exposure, dose-response, substitution effects and opportunity costs receive less attention.

Citation also strips context. The full Monograph may distinguish pure substance from formulation, occupational from consumer exposure, cancer site from general cancer, and limited human evidence from strong animal or mechanistic evidence. A literature review may reduce this to one sentence. An advocacy report reduces it further to 'WHO says it causes cancer.' The words remain technically connected to the original document, but their policy meaning changes at each step.

From academic authority to public policy

IARC states that it does not recommend regulations or interventions, while acknowledging that public authorities use its evaluations when considering prevention. A classification can change the political and evidentiary environment without dictating a particular measure. The extent of that influence should be established from regulatory reasons, statutory mechanisms and public statements rather than inferred from timing alone.

The influence operates through several pathways. Some laws incorporate recognized carcinogen classifications directly or semi-automatically. Some regulators use IARC as one evidence source within a broader risk assessment. Occupational bodies and professional societies use Monographs to identify hazards and recommend exposure controls. Health charities reproduce IARC lists for public education. Legislators cite the classification in hearings and bills. Procurement officers, insurers and companies may change practice before formal regulation because the reputational and liability consequences of ignoring a cancer classification are asymmetric.

Transmission route	How IARC is used	Risk of distortion
Academic research	Background authority grant rationale study design and review synthesis	Repeated citation can look like independent confirmation
Public communication	Cancer lists charity guidance headlines and advocacy materials	Hazard becomes everyday risk and category becomes magnitude
Administrative policy	Evidence input or statutory listing trigger	A global category may displace local exposure and benefit analysis
Private governance	Procurement insurance warnings reformulation and product withdrawal	Decisions occur without transparent proportionality review
Litigation	General causation narrative notice warning theory and expert support	Institutional prestige can substitute for claimant specific evidence

The proposed transmission runs from an assessment through research, communication, policy and litigation, with possible feedback into research priorities. This describes mechanisms to investigate; it is not evidence of coordination.

Four examples of the authority chain

Glyphosate and California Proposition 65

California listed glyphosate under Proposition 65 effective July 7, 2017, following IARC's 2015 Group 2A classification. Listing and enforceable warning duties are separate legal questions. Subsequent litigation restricted the cancer-warning requirement, so the historical listing should not be described as an unrestricted current duty to warn. The episode illustrates how a classification can enter state law while remaining subject to additional constitutional and regulatory review (California OEHHA, 2017; National Association of Wheat Growers v. Bonta, 2023).

Aspartame and the visible hazard risk split

In 2023 IARC classified aspartame in Group 2B on limited evidence, while JECFA retained its acceptable daily intake. FDA disagreed with the IARC conclusion. The simultaneous assessments demonstrate why a hazard category cannot alone establish risk at permitted exposure levels. They also provide a useful test of whether later communication preserves the different questions and evidentiary limitations (WHO, 2023; IARC and JECFA, 2023; FDA, 2025).

Talc and the recycling of classification into litigation

IARC's 2024 evaluation classified talc as probably carcinogenic to humans while carefully distinguishing talc from asbestos-contaminated talc and describing limited evidence in humans alongside animal and mechanistic

evidence. In litigation, however, 'talc' covers materially different exposure and disease theories: ovarian-cancer claims based largely on epidemiology and mesothelioma claims alleging asbestos contamination. Academic and public writing that cites the broad category without preserving those distinctions can make heterogeneous claims appear to share one scientific foundation. The classification then returns to court as an authoritative summary of a literature partly shaped by the controversy itself.

Processed meat and public health messaging

The 2015 classification of processed meat as Group 1 illustrates how a hazard category can be mistaken for risk magnitude. Processed meat shares Group 1 with tobacco smoking because IARC judged the evidence that each can cause cancer to be sufficient, not because the exposures create comparable risks. Public reporting commonly flattened that distinction. The episode matters beyond food advice: it demonstrates that even accurate academic citation of a category can mislead policy debate when evidence strength is heard as effect size.

Litigation and policy as distinct pathways

A lawfare inquiry needs a sharper screen than asking which IARC agents are 'weak'. There are at least two distinct pathways. In the first, a classification is economically valuable because a concentrated and solvent producer or brand owner can be sued, a common disease supplies claimants and the label lowers the cost of constructing a common-causation case. In the second, the classification is politically valuable because it supplies an authoritative premise for regulation, restriction, procurement or a wider public-health narrative even where no attractive private defendant exists. Glyphosate and some other pesticides fit the first model; DDT and involuntary smoking are better studied through the second. The pathways can overlap, but they should not be conflated.

Alcohol and involuntary smoking illustrate how robust hazard findings can support prevention while leaving questions of dose, proportionality and implementation. Coffee illustrates revision: IARC moved it from Group 2B in 1991 to Group 3 in 2016. A fair institutional assessment must examine correction and justified regulation as well as contested classifications (IARC, 2004, 2012, 2016).

Selected agents illustrate different downstream pathways. Glyphosate supplies a prominent litigation case; aspartame a simultaneous hazard and risk comparison; involuntary smoking and alcohol cases with well-established carcinogenic hazards; and coffee an example of institutional revision. Their selection here is illustrative and cannot support an estimate of the prevalence of error or bias.

Nicotine and the problem of substitution

Restrictions on lower-risk nicotine products provide a potential test of category migration. Researchers should compare the stated evidence about each product with evidence about smoking, and assess adult switching, youth uptake, dependence and illicit supply. A ban does not establish that these considerations were ignored. That conclusion requires the actual legislative and regulatory record.

Comparing assessments without counting unlike labels

The five comparisons below examine how evidence, dates and decision rules can produce different institutional conclusions. Most compare IARC with EPA; aspartame also involves the international JECFA and FDA. They are purposively selected illustrations, not a representative sample or a completed systematic review. Coding expresses this paper’s interpretation of the cited assessments and remains open to revision.

Scope and limits of the comparison

No overall discrepancy rate is reported. An earlier draft's simple label count has been removed because it did not establish comparable agents, routes, cancer propositions, evidence periods and assessment questions. The examples distinguish substantive scientific differences, threshold and mandate differences, and mismatched evidence dates. These categories explain why a raw count would mislead.

Agent and assessments	Interpretation	Principal limitation
Glyphosate IARC 2015 and EPA historical reviews	Substantive scientific differences as well as risk-assessment differences	Formulations evidence dates and methods differ; EPA review is ongoing
Aspartame IARC and JECFA 2023 with FDA response	Different thresholds and mandates	JECFA is an international committee not a US regulator
Diazinon IARC 2015 and cited EPA reregistration record	Evidence-date mismatch	Do not treat an older label as a contemporaneous scientific rebuttal
2,4-D IARC 2015 and EPA 2012 petition order	Different integration of animal and mechanistic evidence	Historical assessments with imperfectly matched evidence
Malathion IARC 2015 and EPA 2016	Partial divergence in strength and interpretation	EPA also identified a possible carcinogenic signal

The five examples below support a method for comparing assessments. They do not estimate how often IARC differs from regulators across its full programme.

What a difference between assessments means

Glyphosate and 2,4-D illustrate differences that extend beyond hazard versus exposure-specific risk. Aspartame combines differences in evidentiary weight with different mandates. Diazinon illustrates the danger of comparing an older regulatory record with later evidence. Each conclusion concerns the identified historical assessments, not every current or future evaluation of the agent.

Malathion illustrates partial overlap: EPA identified suggestive evidence, so its position should not be coded as an affirmative finding of no carcinogenic potential. Cases involving different routes, formulations or statutory purposes require separate treatment. No binary classification should erase those distinctions.

Why the institutions can reach different answers

Four separable mechanisms must be coded. First, the evidence universe may differ. IARC generally relies on published or otherwise publicly reviewable evidence, whereas EPA can examine complete registrant study reports that were not publicly available in sufficient detail when a Working Group met. Second, the institutions may weight the same evidence differently, particularly modest observational associations, rare animal tumours, trend tests, historical controls and mechanistic assays. Third, their inference rules differ: IARC can classify a hazard from a specified combination of human, animal and mechanistic evidence without quantifying risk at ordinary exposure, while EPA can condition its conclusion on dose, route, mode of action and registered use. Fourth, cutoff dates differ. A later cohort update or animal experiment cannot explain what an earlier Working Group concluded, but it may determine whether that conclusion remains persuasive.

Glyphosate evidence reconstruction

EPA’s assessment is being updated. In September 2026 it announced additional work with HHS and extended comment on its open-literature search to November 23, 2026. The historical comparison below should not be read as a final account of the pending reassessment (EPA, 2026a, 2026b).

Glyphosate illustrates all four mechanisms. IARC's limited human evidence rested principally on positive associations with non-Hodgkin lymphoma in several case-control studies. It included the then-available Agricultural Health Study but regarded its follow-up as too short to outweigh those associations. EPA assigned greater weight to the cohort evidence and treated the case-control results as vulnerable to recall bias, exposure misclassification, confounding, inconsistent dose response and lack of reproducibility. A later Agricultural Health Study follow-up was not available to the March 2015 Working Group and therefore creates a genuine difference in evidence date rather than an interpretive disagreement about evidence both bodies possessed.

The animal evidence produces a more direct methodological conflict. IARC emphasized statistically significant dose trends, rare tumour types and findings occurring without overt toxicity, concluding that the evidence in experimental animals was sufficient. EPA placed more weight on pairwise comparisons with controls, historical-control ranges, consistency across studies, sexes and strains, and the possibility of chance findings generated by multiple tumour comparisons. The dispute is therefore partly about which statistical signal should govern: a positive trend and unusual tumour, or reproducible excesses surviving a broader weight-of-evidence review.

The evidence universe was also different. IARC excluded studies or summary tables that did not provide enough public detail for independent assessment. EPA could examine full registrant reports submitted under the pesticide programme. The trade-off is real. IARC's record is more publicly auditable, but it may omit well-powered negative studies. EPA's record may be more complete, but some of the evidence originates with the regulated producer and historically was difficult for outsiders to inspect. A balanced comparison must test both publication bias and sponsor bias rather than assuming that either public or industry evidence is intrinsically reliable.

Mechanistic evidence raises an agent-definition problem. IARC considered pure glyphosate, commercial glyphosate formulations and evidence from exposed populations, and found strong evidence of genotoxicity. EPA's regulatory synthesis placed greater weight on guideline studies and focused more narrowly on the active ingredient and registered products. Positive findings for a formulation may be highly relevant to real-world users while remaining insufficient to prove that glyphosate itself, rather than a surfactant or interaction among ingredients, produced the effect. This distinction matters legally because a claim may concern a marketed formulation even when the scientific label is attached to the active ingredient.

The conclusion is not that the disagreement disappears once hazard is separated from risk. EPA's own carcinogenic-potential conclusion differed from IARC's, and the bodies treated shared human, animal and mechanistic evidence differently. But the disagreement cannot be summarized honestly by placing Group 2A beside 'not likely' without displaying the different datasets, dates and inference rules. A reproducible comparison should publish the study inventory, inclusion decisions and evidence-stream matrix before reporting a discrepancy rate.

Aspartame evidence reconstruction

Aspartame is the strongest available test of whether different conclusions reflect different data or different interpretation. IARC and JECFA evaluated the agent within weeks of each other in 2023. Three JECFA members observed the IARC meeting; the secretariats attended both; and the joint WHO account says there was a large

degree of overlap in the evidence because studies submitted to EFSA had been made public. This is therefore not principally a case in which one body possessed a hidden positive or negative dataset.

The human signal was hepatocellular carcinoma. IARC identified three studies covering four large cohorts: EPIC; a pooled NIH-AARP and PLCO analysis; and Cancer Prevention Study II. All used artificially sweetened beverages as a proxy for aspartame rather than repeatedly measuring aspartame itself. Each reported a positive association with liver cancer overall or in an important subgroup. IARC called this limited evidence because chance, bias and confounding could not be ruled out. JECFA noted the same statistically significant findings but found no consistent association with a specific cancer and emphasized exposure misclassification, reverse causality and socioeconomic, lifestyle and dietary confounding. The human disagreement is thus mainly over what a recurring but fragile observational signal is entitled to establish.

The animal record was similarly shared. JECFA reviewed twelve oral carcinogenicity studies. All but the Ramazzini Institute studies by Soffritti and colleagues were negative. IARC treated increased tumours in rats and mice of both sexes across three published studies as limited animal evidence, while expressly identifying the absence of litter adjustment in prenatal studies, disputed lymphoma diagnoses and unresolved interpretation of liver and lung lesions. JECFA relied more heavily on the negative studies, the design and reporting limitations in the Ramazzini work, and the absence of a persuasive mode of action. The difference is one of evidentiary weight, not ignorance of the positive studies.

Mechanistic evidence exposes the decision threshold most clearly. Both bodies considered genotoxicity inconclusive. IARC nevertheless found consistent and coherent evidence of oxidative stress, with suggestive evidence of chronic inflammation and altered cell proliferation, cell death and nutrient supply in experimental systems. JECFA considered the oxidative-stress studies limited and observed that expected histopathological consequences were absent from other short- and long-term studies. It also emphasized that ingested aspartame is fully hydrolysed to phenylalanine, aspartic acid and methanol and does not circulate intact. IARC treated mechanistic clues as limited support for hazard; JECFA found no plausible mechanism strong enough to alter the risk assessment.

Exposure then separates the mandates. JECFA estimated mean intake at up to 10 mg/kg body weight per day in children and 5 mg/kg in adults, and high intake at up to 20 and 12 mg/kg respectively—below the retained acceptable daily intake of 40 mg/kg. IARC did not ask whether ordinary consumption exceeds a safe intake. Its Group 2B label followed from limited human evidence under the Monographs decision rules even though every evidence stream had acknowledged weaknesses. FDA went further and said the studies had significant shortcomings and did not support the possible-carcinogen classification.

The proper coding is therefore 'threshold and mandate divergence,' not simple scientific contradiction. There is a real interpretive disagreement about the weight of the liver-cancer cohorts, Ramazzini studies and oxidative-stress evidence. But the apparently stark public contrast—'possibly carcinogenic' versus 'safe within the ADI'—also joins two different propositions. For academic and policy analysis, the crucial transmission question is whether later papers cite Group 2B as evidence of ordinary-use risk without carrying forward the proxy-exposure limitations, alternative explanations and JECFA exposure assessment.

Diazinon evidence reconstruction

Diazinon demonstrates why assessment dates must be matched before labels are compared. IARC's Working Group met in March 2015. The United States cancer classification commonly placed against it derives from EPA's 2000 human-health assessment and the subsequent reregistration record. The most important IARC human evidence appeared after that EPA assessment. The cited EPA reregistration record is substantially

earlier than IARC’s evaluation. The apparent Group 2A versus 'not likely' contrast is therefore insufficient evidence that two bodies interpreted the same science differently.

IARC's limited human evidence rested on three cancer patterns. In the Agricultural Health Study, lung cancer showed a consistent exposure-response trend across successive updates, with adjustment for smoking and other pesticides. Non-Hodgkin lymphoma was positively associated with diazinon in two case-control datasets; the overall association was absent in the Agricultural Health Study, but follicular lymphoma and some small B-cell subtypes showed exposure-response signals. Leukaemia also rose monotonically with cumulative exposure in the Agricultural Health Study, although case numbers were limited. IARC acknowledged the small number of studies and could not fully exclude confounding by other pesticides.

The animal evidence was weak and largely consistent with the older regulatory view. One mouse study reported hepatocellular carcinoma in males only at the lowest dose. One rat study reported combined leukaemia or lymphoma in males only at the lowest dose, while a second rat study was negative. Neither positive finding showed the dose pattern normally expected of a causal effect, and IARC expressly said that the increases could not clearly be attributed to diazinon. It nevertheless categorized the animal evidence as limited. This is not where the sharp institutional difference lies.

Mechanistic evidence supplied the upgrading force. IARC found strong evidence of genotoxicity from DNA and chromosomal damage in experimental animals and human and animal cells. It also found strong evidence of oxidative stress across rodent tissues and cell systems, strengthened by experiments in which antioxidants reduced the effects. The Working Group concluded that both mechanisms could operate in humans. Much of this literature and the key-characteristics framework post-dated EPA's operative cancer assessment.

This is an evidence-date mismatch in the sources compared here. A current EPA synthesis might reach a similar or different conclusion, but that cannot be inferred from an older label. Diazinon therefore cannot contribute to a discrepancy count without a matched later assessment.

2,4-D evidence reconstruction

2,4-D is a cleaner test of interpretation than diazinon because EPA's 2012 petition order and IARC's 2015 evaluation considered substantially overlapping evidence. The labels are not exact opposites: EPA retained Group D, 'not classifiable as to human carcinogenicity,' while IARC assigned Group 2B, 'possibly carcinogenic to humans.' Yet the difference cannot be dismissed as hazard versus registered-use risk. EPA's order expressly revisited carcinogenicity, mutagenicity and oxidant effects before concluding that the evidence did not support a positive cancer finding. The comparison is historical and does not establish that these remain the latest assessments.

On human evidence, the institutions were close. IARC reviewed more than ten studies concerning non-Hodgkin lymphoma and found no strong or consistent increase. Some North American case-control analyses showed associations or exposure-response patterns, but other studies were null, adjustment for other pesticides moved estimates toward the null, and IARC's own meta-analysis found no association for ever exposure. EPA likewise concluded that the epidemiology did not establish a cause-and-effect relationship. IARC therefore called the human evidence inadequate rather than using it to drive Group 2B.

The animal record supplied limited positive evidence, but it was fragile. IARC identified an increase in reticulum-cell sarcoma in a female-mouse injection experiment with design limitations, increased multiplicity of urethane-induced lung adenomas in a co-carcinogenicity study, and a positive trend in male-rat brain astrocytoma that a later higher-dose study did not reproduce. Other mouse and rat studies were negative. EPA emphasized the two newer guideline bioassays with no statistically significant tumour response and later

histopathology that did not change the result. The disagreement is over whether isolated trends and non-standard experiments retain enough evidentiary value after negative guideline studies.

Oxidative stress is the decisive interpretive fork. IARC found strong evidence capable of operating in humans, drawing on effects in human erythrocytes in vitro, multiple exposed rat tissues and partial reversal by vitamin E. EPA had already confronted reported oxidant effects in 2012 but said they did not alter the classification because the primary rodent and human evidence was non-positive. This is the same mechanism performing different work: corroborative upgrading evidence for IARC, but an insufficient secondary signal for EPA.

The rest of the mechanistic record was comparatively weak. IARC found weak genotoxicity evidence because pure 2,4-D was largely negative and many positive studies involved formulations or mixtures. EPA similarly emphasized negative bacterial and in-vivo animal mutation assays, mixed mammalian-cell results and low mutagenic potential. IARC found moderate evidence of immunosuppression but described the findings as mixed, and only four of 265 relevant Tox21 or ToxCast assays were positive. These shared weaknesses sharpen the real dispute rather than resolving it: whether strong oxidative-stress evidence and limited animal signals may cross a hazard threshold when human evidence is inadequate and genotoxicity is weak.

The appropriate code is 'genuine divergence.' Some later papers and IARC's broader key-characteristics synthesis make the evidence sets imperfectly identical, but the core animal, epidemiological and oxidative-effect record was already before EPA. The 2,4-D comparison therefore shows that institutions can reach different conclusions mainly by assigning different upgrading force to the same type of mechanistic evidence—not only because regulators possess different data or answer an exposure-specific question.

Malathion evidence reconstruction

Malathion is a valid same-period comparison but not a clean contradiction. IARC's Working Group met in March 2015. EPA's 2016 human-health assessment incorporated epidemiology reviews completed in 2014 and 2016 and retained a cancer classification of 'suggestive evidence of carcinogenicity but not sufficient to assess human carcinogenic potential.' Both institutions therefore saw a possible cancer signal. They disagreed about how strong it was and how it should affect risk assessment.

IARC found limited human evidence based principally on non-Hodgkin lymphoma and prostate cancer. EPA reviewed more than two dozen cancer studies and described most results as null or statistically non-significant. It regarded the non-Hodgkin lymphoma findings as inconsistent and vulnerable to chance, recall bias and exposure misclassification. For prostate cancer, EPA distinguished the preliminary association with aggressive disease from total prostate cancer and said that external replication and a biological explanation were needed. The same observations crossed IARC's limited-evidence threshold but not EPA's hazard-identification threshold.

The animal disagreement is more concrete. IARC found sufficient evidence after considering liver tumours in mice and female rats, rare oral and nasal tumours in rats, mammary tumours after subcutaneous exposure and other findings. EPA treated the liver tumours as occurring only at excessive doses and regarded most rare oral and nasal lesions as indistinguishable from random occurrence. It also emphasized that malaoxon was not carcinogenic in its guideline mouse and rat studies. EPA therefore retained only suggestive evidence and noted that the tumour-producing doses were almost one hundred times higher than the points of departure used for risk assessment.

Mechanistic evidence widened the gap. IARC found strong evidence of genotoxicity from exposed humans, experimental animals and cells, notwithstanding negative bacterial mutagenicity tests. It also found strong evidence of oxidative stress, inflammation, receptor-mediated effects and altered cell proliferation, and concluded that these mechanisms could operate in humans. EPA's 2016 cancer synthesis said the mutagenicity

evidence did not support a mutagenic concern and did not use oxidative or receptor-mediated mechanisms to upgrade the classification. Instead it concluded that a nonlinear chronic reference dose would protect against carcinogenicity as well as other chronic effects.

This should be coded as 'partial scientific divergence.' The institutions considered substantially overlapping evidence and differed materially in causal inference, dose interpretation and mechanistic integration. But EPA did not find malathion non-carcinogenic. Its 'suggestive evidence' category points in the same direction as IARC, while expressing less confidence and allowing a threshold-based risk assessment. Counting malathion as a binary IARC-versus-EPA discrepancy would exaggerate institutional conflict; excluding it entirely would conceal a meaningful scientific disagreement over evidentiary strength.

From a comparison table to a testable political economy

The expanded dataset should include every IARC Group 1, 2A and 2B evaluation within a defined period, preferably 2010 through 2026, for which a United States federal assessment can be identified. Each row should preserve CAS number, agent or formulation, exposure route, cancer site, evidence cutoff date, human, animal and mechanistic evidence, United States agency, statutory question and review date. Concordance should then be reported twice: first as raw label agreement and again after excluding rows where the agencies asked materially different questions.

The explanatory variables are as important as the classification. Manufacturer concentration, market capitalization, Western ownership, claimant-pool size, product identification and latency should be recorded alongside foundation grants, NGO campaigns, academic-publication growth, media volume, Proposition 65 listing, plaintiff advertising, litigation finance, verdicts, settlements, warnings, taxes, bans and procurement changes. The central test is whether scientific divergence produces different downstream outcomes depending on the available end market. Concentrated solvent defendants should predict litigation. Diffuse defendants, public procurement or weak claimant-specific causation should predict policy campaigns. Reclassification cases such as coffee test whether the academic and policy ecosystem unwinds when the scientific signal weakens.

Three findings would be especially revealing. First, genuine IARC United States divergences may be more strongly associated with mass tort formation than aligned classifications because disagreement itself sustains expert conflict and trial uncertainty. Second, defendant concentration may explain which pesticide classifications become large cases while similarly classified agents remain primarily regulatory issues. Third, foundation and advocacy activity may cluster around policy-purpose agents even where no viable defendant exists. None of these relationships establishes improper motive. They would show where institutional authority has the greatest economic or political leverage and identify the records that require closer investigation.

Glyphosate shows how the gateway operates

In March 2015, IARC classified glyphosate as probably carcinogenic to humans, Group 2A. IARC reported limited evidence in humans, sufficient evidence in experimental animals and strong evidence of genotoxicity. Other regulatory bodies, considering exposure as well as hazard and sometimes a wider evidence base, reached different conclusions about risk. That institutional divergence did not prevent litigation. It created the central courtroom contest.

The IARC classification did at least four kinds of work for Roundup claims. First, it converted a disputed causal theory into one carrying the imprimatur of a WHO-affiliated scientific agency. Second, it supplied a common-causation foundation that could be reused across a large inventory of claims. Third, it supported the argument that Monsanto should have warned after 2015, notwithstanding the Environmental Protection Agency's

approved label. Fourth, it simplified the public story: an international cancer agency had said 'probably carcinogenic' while the manufacturer continued to sell without a cancer warning.

None of those propositions proves individual causation. Yet together they can move a case from speculative to financeable. Once claims can survive early motions and reach juries, a small number of very large verdicts affects the settlement value of the entire inventory. Bayer has paid billions of dollars to resolve Roundup claims while continuing to dispute the causal proposition. The process premium described in CGHA's companion paper, When Process Becomes Punishment, then becomes decisive: the expected cost of resistance includes legal expense, discovery, management time, reputational damage, financing effects and the risk that an exceptional verdict will reset negotiations across thousands of cases.

IARC does not write the legal test but it can set the litigation threshold

IARC does not control Federal Rule of Evidence 702 or the governing state rules. Courts must assess the expert's evidence and reasoning under the applicable admissibility standard, and claimants must establish the elements of their claims. In Monsanto Co. v. Durnell, the Supreme Court held that FIFRA preempted the failure-to-warn claim before it because it required a cancer warning different from the EPA-approved label. That legal holding does not resolve every scientific or liability question (Monsanto Co. v. Durnell, 2026).

The better description is that IARC helps set a de facto threshold for strategic litigation. Its label can supply sufficient scientific respectability to recruit clients, attract finance, retain experts, generate media attention and resist dismissal. Whether the claim ultimately wins may depend on later evidence. Whether a litigation industry can be built may depend on the classification itself.

Why the classification is useful to litigators

The practical value of an IARC classification is not confined to admissibility or the final verdict. It reduces the cost of constructing and communicating a case at several earlier stages. Its WHO association supplies recognition; its categorical language supplies a headline; its review supplies a map of favorable studies and mechanisms; and disagreement with regulators supplies an adversarial story in which a manufacturer can be portrayed as resisting an international cancer authority. These advantages exist even where the Monograph expressly stops short of exposure-specific risk or individual causation.

Litigation stage	Use of IARC	Immediate value	Question the court should preserve
Claim recruitment	A recognized cancer category makes a causal allegation intelligible in advertising and screening	Lowers acquisition and explanation costs	Was this claimant exposed at a relevant dose and during a plausible latency period?
Financing and aggregation	One classification supports a common theory across a large inventory	Makes portfolio valuation and outside finance possible	How many claims have product identification and medically plausible specific causation?
Expert development	The Monograph identifies studies mechanisms and cancer sites on which opinions can be built	Reduces the cost of constructing general causation testimony	Did the expert independently assess the record or merely borrow institutional authority?
Notice and warning	The publication date provides a visible point at which alleged knowledge can be framed	Supports a simple post-classification warning narrative	Could the defendant legally change the label and what did exposure-based regulators conclude?

Litigation stage	Use of IARC	Immediate value	Question the court should preserve
Trial narrative	WHO-linked categorical language is easier to communicate than dose response or uncertainty	Creates authority and moral asymmetry before a jury	Has hazard been presented as risk or as proof of this claimant's causation?
Settlement leverage	Survival of some claims plus a few large verdicts changes the expected cost of resistance	Raises the value of the full inventory before final merits resolution	Are weak and strong claims being priced together because process costs dominate?

The same classification can also assist defendants. It may define the limits of the evaluated agent, exposure route, cancer site and evidence category; confirm that the inquiry is hazard rather than risk; and reveal where the Working Group relied on animal or mechanistic evidence rather than persuasive human data. The legally important question is therefore not whether IARC may be mentioned. It is which proposition the classification genuinely supports and where an expert has supplied additional evidence for the remaining steps.

Poor science institutional capture or corruption

Critical accounts of Monograph 112 raise questions about expert relationships, subsequent litigation consulting, evidence selection and transparency. Those questions require dated disclosures, contracts, contemporaneous communications and the scientific record. Later consulting income alone does not prove that an earlier assessment was manipulated. IARC describes published safeguards for independent evaluation; an audit should test their application rather than presume either failure or success (IARC, 2019; Zaruk, 2025).

Four different findings should not be collapsed into the word corruption. Scientific disagreement exists when competent institutions weight substantially overlapping evidence differently, as the 2,4-D and malathion comparisons illustrate. Poor science exists when weak designs, selective endpoints, inadequate confounding control or implausible extrapolation receive more confidence than they warrant. Institutional capture exists when recurring networks, funding dependencies or ideological commitments systematically shape agendas, appointments, evidentiary choices or communications. Corruption is narrower and more serious: intentional misuse of office or scientific authority for an improper benefit, ordinarily requiring evidence of concealment, direction, exchange or knowing manipulation.

Possible finding	Evidence that could support it	What does not establish it
Scientific disagreement	Transparent differences in causal inference dose treatment or mechanistic weighting	A different final category by itself
Poor or selective science	Omitted contrary evidence unstable analyses inconsistent inclusion rules or claims stronger than the underlying studies	A hazard classification that differs from an exposure-based risk assessment
Institutional capture	Persistent network concentration undisclosed dependencies one-sided agenda setting or procedures that protect a preferred conclusion	Shared professional views membership or foundation support alone
Corruption	A quid pro quo concealed financial interest directed manipulation fabricated record or knowing abuse of authority for improper benefit	Consulting income earned later unless linked by evidence to an earlier official act

The evidence discussed here justifies examination of transparency and inference; it does not establish corruption. Claims of intentional manipulation require records connecting conduct to the disputed assessment

and improper benefit. A demand for disclosure should identify that missing evidence, rather than present the allegation as a conclusion.

The correct accountability standard is symmetrical. Corporate ghostwriting, selective publication and attempts to pressure journals are relevant and should be disclosed. So are plaintiff-side consulting, litigation-oriented research, foundation support, activist coordination and communications designed to produce a legal or political outcome. Disclosure does not decide whose science is correct. It allows readers and courts to test whether study selection, analytic choices or public claims changed with the source and intended use of funding.

Foundation funding can build the ecosystem around the classification

Foundation influence need not take the crude form of purchasing a scientific conclusion. Funding can select the problem, assemble a network, support particular research questions, amplify selected findings and connect academic work with advocacy, media, lobbying and strategic litigation. This is a general institutional mechanism familiar from agenda-setting and knowledge-production research, not an idea attributable to one writer. Zaruk's recent essays provide a pointed contemporary account of how he believes professionalized philanthropy operates across NGOs, fiscally sponsored projects, research, media and legal campaigns; they are cited here as examples of that argument, not as its origin (Zaruk, 2026a; 2026b).

A classification may become influential through multiple organizations that research, communicate, advocate and litigate around the same risk. Common funding can align activity, but parallel action can also reflect independent agreement. Whether a particular funding strategy coordinated those outputs is an empirical question for grant terms, communications and disclosures.

This is a hypothesis to be tested, not a license to infer coordination from common views. Parallel activity may reflect genuine agreement about a danger. The empirical questions are concrete: Which grants were earmarked? What deliverables were required? Did funders support both research and litigation organizations working on the same target? Were communications or media placements coordinated? Did authors disclose the intended legal use of their work? Did contrary findings receive comparable support and attention? Were fiscal sponsors or donor-advised funds used in ways that obscured the ultimate donor?

Climate attribution when litigation demand reaches upstream into science

Climate attribution provides a useful comparison precisely because the underlying scientific proposition need not be denied. Anthropogenic greenhouse-gas emissions warm the climate. The legal problem begins when that general proposition is translated into a chain of increasingly specific claims: human influence on climate; climate influence on the probability or intensity of a particular event; the share of that influence attributable to a class of emitters; the share attributable to a named defendant; and finally the monetary damages legally caused by that defendant. Confidence at the first link does not automatically transfer to the last.

The Firebreak's September-October 2026 climate series argues that strategic litigation, philanthropic funding, communications campaigns and attribution research increasingly form one ecosystem. Its strongest contribution is not the rhetorical claim that climate science has been corrupted. It is the narrower institutional question: whether actors financing climate litigation also finance research, communications or assessments that supply the causal propositions those lawsuits require, and whether courts and the public can see those relationships clearly enough to judge independence and uncertainty.

That question is especially important in event attribution. Rapid attribution studies can estimate how modeled probabilities or intensities differ between a factual climate and a modeled counterfactual climate without the anthropogenic forcing under study. Such analyses can be scientifically informative. They are also unusually

portable into headlines and pleadings because they produce event-specific statements while public attention is concentrated on the disaster. The combination creates an incentive to distinguish carefully between what the model estimates, what assumptions drive the counterfactual, what uncertainty remains, and what further evidence would be required for defendant-specific legal causation.

Scientific material prepared for judges deserves clear disclosure of relevant authorship, funding and litigation relationships. A funder supporting both research and its synthesis does not establish a purchased conclusion, and consulting work does not by itself invalidate scholarship. Evaluation should focus on methods, contrary evidence, committee selection, disclosed interests and the precise legal uses proposed.

This comparison clarifies the difference among disagreement, weak science, capture and corruption used earlier in this paper. Climate attribution may be methodologically sound yet communicated beyond its warranted scope. A funding network may shape the questions asked without dictating answers. An assessment process may be institutionally captured even if individual researchers act in good faith. Corruption is a still narrower allegation requiring evidence of intentional manipulation, concealment or exchange. Collapsing those categories makes the critique easier to dismiss; separating them makes it testable.

The resulting authority loop parallels IARC but adds a further feedback mechanism. IARC begins with a classification that can later be used in litigation. In climate cases, litigation itself can increase demand for event- and source-attribution research capable of supporting causation arguments. The loop can therefore run in both directions: research creates legal leverage, and legal strategy creates a market for particular forms of research. That does not invalidate the research. It means independence, preregistration where feasible, transparent model choices, sensitivity testing, replication, funding disclosure and clear limits on legal inference become central accountability safeguards.

Why this belongs in the CGHA lawfare programme

CGHA's lawfare research asks when legal process moves from adjudicating a plausible wrong to obtaining policy, commercial or reputational objectives through leverage. IARC sits further upstream. It helps determine which causal propositions acquire authoritative status in academic writing and public policy before a complaint is filed. A classification can affect research funding, journal framing, warnings, insurance, investment, product availability and settlement strategy even where exposure-specific risk and individual causation remain untested.

The institutional sequence may connect funding, assessment, communication, policy and claims. At every stage legitimate activity is possible, and the sequence need not occur in every case. An audit should establish which links exist and whether an evidentiary burden changed as a proposition moved between institutions.

A research test for the IARC authority chain

Stage	Minimum evidence	Question to answer
Classification	Working group composition, declarations, drafts, evidence inclusion rules and post-meeting changes	Did conflicts or procedural choices materially affect the classification?
Funding network	Grant agreements, donor-advised funds, fiscal sponsors, regrants and common contractors	Was formally separate activity strategically coordinated?
Academic translation	Citation mapping across original studies reviews grant rationales and later papers	Did one classification become an apparently independent academic consensus?

Stage	Minimum evidence	Question to answer
Policy translation	Regulatory records legislative hearings warnings procurement and guidance	Where did hazard become risk or a policy instruction without new evidence?
Claim formation	Advertising, claimant screening, medical records, expert retention and litigation finance	Did common evidence substitute for claimant-specific support?
Court treatment	Rule 702 rulings, jury instructions, verdicts, appeals and reversals	How much legal weight did the IARC label actually receive?
Behavioral effects	Warnings, withdrawals, insurance, research changes, settlements and market exits	What changed before the merits were finally resolved?

A proportionate reform agenda

IARC should retain the ability to identify carcinogenic hazards. The solution is not to make an international scientific agency answer legal questions it was not designed to decide. It is to prevent its answer to one question from silently deciding several others.

Priority setting should disclose nominations, supporting organizations and reasons for selecting or declining agents, so agenda formation can be distinguished from evidence evaluation.

IARC should publish complete and usable conflict declarations covering current and reasonably anticipated expert and litigation work, together with the financial relationships of relevant institutions.

Draft histories, evidence inclusion decisions and material post-meeting changes should be preserved and disclosed, subject to narrow protections for personal or genuinely confidential data.

Academic papers citing an IARC category should state whether it is being used as background, evidence or a policy premise, and should preserve the agent, exposure and cancer-site limits of the underlying Monograph.

Policies relying on IARC should separately document exposure, absolute and relative risk, benefits, feasible substitutes and the consequences of error. Classification alone should not complete that assessment.

Courts should instruct clearly on the difference between hazard classification, exposure-specific population risk and claimant-specific causation. An IARC classification should be relevant but never conclusive.

Expert reports should state precisely where they depart from the classification and what additional evidence connects it to dose, latency, disease subtype and alternative causes.

Foundations, fiscal sponsors, NGOs, media organizations and litigation groups should disclose grants and coordination material to the campaign at issue, including the ultimate source of donor-advised funds where legally possible.

Claimant screening and controlling legal questions should be addressed early, before inventories and discovery costs create settlement pressure detached from the merits.

Conclusion

IARC's formal authority is narrower than its practical influence. It identifies carcinogenic hazards; it does not calculate ordinary-use risk, choose proportionate policy or decide individual causation. But its classifications can shape the research agenda, become a premise in academic writing, alter regulatory burdens and provide

the common scientific authority around which a mass tort is recruited, financed and tried. That makes IARC a critical institution in the political economy of knowledge as well as litigation.

The right response is neither automatic deference nor automatic dismissal. IARC classifications should be scrutinized with the same seriousness applied to corporate studies, paid experts and litigation-funded research. Academics should preserve the distinction between a classification and new empirical evidence. Policymakers should show the exposure, benefit and substitution analysis that converts hazard into intervention. Courts should insist that each inferential step to individual causation is proved. The aim is not to protect products from liability. It is to ensure that a hazard label opens an inquiry rather than predetermining research, policy and legal results. The climate-attribution comparison reinforces the same rule: confidence in a broad scientific proposition should not be allowed to substitute for evidence at each narrower step from event to source to defendant to damages.

Relationship to the series

Paper 2 supplies the series' account of how evidence becomes institutional authority. IARC shows how an authoritative classification can travel downstream into policy and tort litigation; climate attribution adds the reverse pressure, in which litigation demand may travel upstream and shape the production, funding and communication of scientific evidence. Paper 1 provides the process test, Paper 3 examines professional enforcement, and Paper 4 asks how similar authority networks operate in international governance. Paper 5 examines incentives for the persistence of campaigns across these settings.

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