Research Article

Latency, Persistence, and the Duration of Regulatory Delay: A Comparative Analysis of Seven Agrochemical and Industrial Chemical Disasters, 2001–2026

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Abstract

Background. Chemical exposure disasters differ enormously in how quickly harm becomes visible, yet comparative scholarship rarely treats that variation as an object of study. We ask whether the interval separating the first credible signal of population harm from an effective institutional remedy is governed by the strength of the available evidence or by features of institutional design. Methods. We conducted a structured comparative case analysis of seven chemical exposure settings whose documented public health burden occurred or peaked between 2001 and 2026, selected purposively for maximum variation in agent class, outcome latency, and governance jurisdiction. Cases were characterised along five pre-specified dimensions and the primary epidemiological studies appraised using ROBINS-E domains. The design is comparative rather than systematic, and no pooled estimate was computed. Findings. Delay ranged from seventeen months to more than three decades and showed no consistent relationship with evidentiary strength. The chlordecone evidence base was the most rigorous in the sample and its delay among the longest; the Tianjin hazard involved no scientific uncertainty and the institutional failure was nonetheless total. Latency instead tracked two institutional features: whether a body held both jurisdiction and a duty to publish, and whether entitlement required proof of individual causation. Compensation outcomes varied by an order of magnitude under comparable evidence, according to whether eligibility was presumptive or adjudicated. Interpretation. For agents combining environmental persistence with long-latency developmental or endocrine endpoints, the conventional sequence of authorisation, observation, and withdrawal cannot operate within the period during which harm accrues. We set out four institutional instruments addressed to the interval itself.

Keywords: endosulfan; chlordecone; persistent organic pollutants; environmental justice; precautionary principle; chemical governance

1. Introduction

Synthetic chemistry underwrites modern agriculture and modern industry alike. The benefits are measurable in yield, in vector control, and in material throughput. The costs are measurable too, but they accrue on a different timetable, and the asymmetry between those two timetables is the subject of this paper.

Environmental health scholarship conventionally distinguishes acute industrial catastrophe from chronic environmental degradation. The methyl isocyanate release at Bhopal in 1984 remains the archetype of the former: a discrete event, an identifiable plume, mass casualties within hours. Much of the harm documented since 2001 fits that template poorly. Exposure is diffuse, so no single moment of injury can be located. Latency is long, so outcomes manifest after exposure has ceased and often after the responsible entity has reorganised. And the relevant endpoints are multifactorial, which permits any individual case to be attributed to something else.

Each of those difficulties is epistemically genuine. Taken together, however, they produce a regulatory environment in which inaction carries the lowest institutional cost, and that asymmetry, more than the toxicology, is what this paper takes as its object.

The endosulfan episode in Kasaragod district, northern Kerala, supplies an unusually complete record of the pattern. From 1978 the state-owned Plantation Corporation of Kerala applied endosulfan to cashew holdings across eleven village panchayats, moving to helicopter application in the early 1980s and continuing until aerial spraying was halted in 2001. The terrain was poorly suited to the method: undulating ground, numerous water bodies, and dwellings inside and immediately adjacent to the plantation boundary.

Clinical reports of congenital anomaly, neurological disability, delayed sexual maturation, and reproductive morbidity accumulated locally through the 1990s before entering the national record. Investigating at the request of the National Human Rights Commission, the National Institute of Occupational Health concluded in its final report that endosulfan was a causative factor in the disorders observed, finding significantly higher prevalence of neurobehavioural disorder, congenital malformation, and male reproductive abnormality in the exposed village relative to a reference population (NIOH, 2002). The associated peer-reviewed analysis reported that sexual maturity rating and serum testosterone in exposed male schoolchildren were inversely related to aerial exposure after adjustment for age (Saiyed et al., 2003). What followed is as instructive as the epidemiology.

India prohibited endosulfan under Supreme Court direction in 2011; the Conference of the Parties to the Stockholm Convention listed technical endosulfan and its related isomers in Annex A the same year, with time-limited crop-pest exemptions (UNEP, 2011). The Supreme Court directed in January 2017 that ₹5 lakh be paid to each identified survivor within three months. Nine years later the direction remains partially unexecuted, with a further 977 persons identified in 2017 receiving sanction orders only in February 2026 (Jose, 2026).

Whether this sequence is particular to one Indian district or structurally characteristic has not been established comparatively. The relevant literatures remain separated by discipline. Toxicological reviews address mechanism without governance; legal scholarship examines liability without exposure science; environmental justice scholarship documents distributional inequity, frequently without the toxicokinetic detail needed to explain why these particular harms evade detection. Agrochemical and industrial-release events are seldom placed in a single analytic frame, although both are governed by the same regulatory logic of permitted risk.

We therefore pursue three objectives. The first is to compare the toxicological, epidemiological, and ecological profiles of seven chemical exposure settings whose burden occurred or peaked between 2001 and 2026, using Kasaragod as the reference case. The second is to identify the institutional mechanisms generating delay in each setting, and to test whether delay varied with evidentiary strength. The third is to derive from that comparison a governance framework specified at the level of implementable instruments.

2. Methods

2.1 Design and its rationale

This is a structured comparative case analysis. We state that designation precisely because the alternative labels carry commitments this study does not meet.

A systematic review in the PRISMA sense requires exhaustive retrieval against a pre-registered protocol, duplicate screening, and a reported flow of records from identification to inclusion (Page et al., 2021). Our research question is not of that form. We are not estimating an exposure–outcome association across comparable studies; we are comparing the institutional trajectories of a small number of heterogeneous events, for which the unit of analysis is the setting rather than the study. Reporting a PRISMA flow diagram over a purposive sample of seven cases would convey a completeness the design does not possess.

We accordingly report the search and selection procedure in full, so that it is auditable, while making no claim of exhaustive retrieval. Section 7 states the consequences of that choice for the inferences available.

2.2 Search procedure

MEDLINE, Scopus, and Web of Science Core Collection were searched for records indexed between 1 January 2001 and 30 June 2026. Strings combined agent terms (endosulfan, chlordecone, Kepone, glyphosate, paraquat, 2,4,5-T, TCDD, vinyl chloride, sodium cyanide, ammonium nitrate) with outcome terms (congenital anomal*, neurotoxic*, endocrine disrupt*, prostate cancer, Parkinson*, chronic kidney disease, preterm birth) and setting terms (aerial spray*, plantation, derailment, industrial accident).

Institutional documents were retrieved from the World Health Organization, the United Nations Environment Programme, the Secretariat of the Stockholm Convention, the National Academies of Sciences, Engineering, and Medicine, the United States National Transportation Safety Board, and the Indian National Institute of Occupational

Health. Judicial material was obtained from the Supreme Court of India and the Paris Administrative Court of Appeal. Reference lists of included records were hand-searched. Languages were restricted to English and French.

2.3 Eligibility

A setting was eligible where an agrochemical or industrial chemical was released outside controlled containment; a defined civilian or occupational population was exposed; the documented public health burden occurred or peaked between 2001 and 2026, which admits historical exposures whose outcome literature falls within the window; and a governmental, intergovernmental, or judicial response was documented, permitting institutional comparison.
Single-individual poisoning, deliberate chemical weapons use, pharmaceutical adverse events, and exposure wholly contained within a sealed industrial process were excluded.

2.4 Case selection and the latency gradient

Seven settings were retained from the eligible pool by purposive selection for maximum variation. Variation was sought across agent class, governance jurisdiction, income classification of the exposed population, and—centrally for this study—outcome latency.

The last criterion requires explanation, since it determines the shape of the comparison. Latency here denotes the interval between exposure and the clinical manifestation of the characteristic outcome. It ranges in this sample from hours to generations, and the cases were chosen to span that range instead of clustering at one end.

Four settings sit at the long-latency pole, where the characteristic endpoint appears years to decades after exposure: endosulfan in Kasaragod, chlordecone in the French West Indies, paraquat in occupational agriculture, and dioxin in southern Viet Nam. Two occupy an intermediate position, with mixed acute and chronic presentation: glyphosate in Colombian aerial eradication and in South Asian paddy cultivation, and the East Palestine derailment, where acute irritant symptoms were immediate and the carcinogenic concern is latent by one to two decades. One setting, the Tianjin Port explosions, sits at the acute pole, with harm complete within minutes.

The acute cases are included by design, and the reason is methodological. A comparison drawn only from slow-onset events cannot distinguish institutional failures specific to latency from failures common to chemical governance generally; without contrast cases, every observed failure is confounded with the latency of the sample. Tianjin in particular functions as a double control: harm was acute, and the relevant hazard knowledge was entirely uncontested. Where a failure mode appears at the acute pole as well as the chronic one, latency is not its explanation.

2.5 Extraction and appraisal

Each setting was characterised along five pre-specified dimensions: toxicological mechanism at the level of molecular target; dominant exposure pathway; acute and chronic outcome profile; environmental persistence, expressed where available as measured soil or tissue half-life; and institutional response, operationalised as the interval in years between the first peer-reviewed or official report of population-level harm and the first binding national restriction, together with compensation status at the close of the window.

Primary epidemiological studies supporting each case were appraised against the domains of the ROBINS-E tool for non-randomised studies of exposure. Domain-level and overall judgements are reported in Table 2. Two of the included sources—an environmental monitoring study and a hypothesis-generating paper—report no exposure–outcome analysis, and ROBINS-E does not apply to them; they are listed in Table 2 with that designation, and are not omitted, since their evidentiary status bears directly on the weight given to the corresponding cases.

3. Comparative Case Analysis

Cases are ordered from the long-latency pole to the acute. Mechanistic detail is given where it explains the latency or the specificity of the observed outcome, those being the properties that make this class of harm difficult to regulate.

3.1 Endosulfan in Kasaragod, Kerala (application 1978–2001; outcomes 2001–2026)

Endosulfan is a cyclodiene organochlorine acting principally as an antagonist at the γ-aminobutyric acid type A receptor chloride ionophore. Blockade of GABA-gated chloride influx removes the dominant inhibitory constraint on neuronal firing, producing the hyperexcitability and seizure activity characteristic of acute intoxication.

Two further properties matter at population scale. The technical product comprises α- and β-isomers that oxidise environmentally to endosulfan sulfate, a metabolite of comparable toxicity and greater persistence than either parent. Beyond its ionotropic action, the compound interferes with steroidogenesis and androgen receptor signalling, which supplies a mechanism for the reproductive endpoints observed in exposed populations.

Experimental work has characterised the developmental neurotoxicity in some detail. Gestational and lactational exposure in mice reduced striatal dopamine transporter and tyrosine hydroxylase expression in male offspring, with the deficit amplified by subsequent MPTP challenge, indicating that early exposure sensitises the nigrostriatal dopaminergic system to later insult (Wilson et al., 2014). That finding predicts a latent and progressive neurological course in place of an immediately apparent one—precisely the pattern that registration review based on acute toxicity is not designed to detect.

The human evidence rests on the NIOH investigation and its peer-reviewed output. Saiyed et al. (2003) compared male schoolchildren aged 10–19 in exposed Padre village with children from a matched control area, reporting that sexual maturity rating for pubic hair, testicular, and penile development and serum testosterone were each negatively associated with aerial exposure in multiple regression ($p < 0.01$), with elevated luteinising hormone—a configuration consistent with interference in testicular steroid synthesis rather than central hypogonadism. Congenital anomalies of the male reproductive tract, including cryptorchidism, were more prevalent among the exposed.

The study carries high risk of bias on ROBINS-E (Table 2), chiefly from ecological assignment of exposure without individual dose reconstruction. Three features nonetheless constrain the plausible confounding: the gradient across exposure categories, the internal consistency of the hormonal pattern, and the concordance with the independent NIOH prevalence findings in the same population (NIOH, 2002). We treat the association as credible while noting that the design cannot establish the magnitude of effect.

Persistence sustained exposure beyond the end of application. Residues were recovered from drinking water and human blood ten months after the final spray (Saiyed et al., 2003). Bulk quantities were subsequently found to have been disposed of in a disused well within the plantation area, a point source whose groundwater contribution went unassessed for two decades before becoming the subject of proceedings before the National Green Tribunal.

Measured from first local clinical reporting in the early 1990s, the interval to national prohibition in 2011 approaches two decades. Compensation was ordered in 2017 and remained incomplete in 2026 (Jose, 2026). One structural feature deserves note for the accountability analysis below: the applicator was an instrumentality of the same state that later became the compensating authority.

3.2 Chlordecone in Guadeloupe and Martinique (application 1972–1993; outcomes 2001–2026)

Chlordecone, an organochlorine applied against the banana root borer, was prohibited in the United States in 1976 on toxicity grounds. Authorisation for sale and use in the French West Indies was renewed repeatedly thereafter, and application continued until 1993 (Paris Administrative Court of Appeal, 2025). The compound is a well-characterised endocrine disruptor with estrogenic and progestogenic activity, classified by IARC as possibly carcinogenic to humans. This is the strongest evidence base in the sample, because exposure is measurable in plasma and the affected population is served by a health system capable of cohort follow-up.

The Karuprostate case-control study of 623 men with prostate cancer and 671 controls found risk rising with plasma concentration (OR 1.77, 95% CI 1.21–2.58 for the highest tertile above the limit of detection; p for trend = .002) and with a cumulative exposure index (OR 1.73, highest quartile), with effect modification by family history and by chlordecone reductase genotype (Multigner et al., 2010).

In the Timoun mother-child cohort, maternal plasma chlordecone at delivery across 818 pregnancies was associated with shortened gestation (−0.27 weeks per $\log_{10}$ increase; 95% CI −0.50 to −0.03) and with a 60% increase in preterm birth risk (95% CI 10–130%), independently of p,p′-DDE and PCB-153 and irrespective of whether labour was spontaneous or induced (Kadhel et al., 2014). The specificity of the association to chlordecone, where the co-measured organochlorines showed none, strengthens the mechanistic inference, given the bearing of estrogenic and progestogenic activity on myometrial quiescence.

Persistence here is effectively generational. Applying the WISORCH first-order desorption model to French West Indian soils, Cabidoche et al. (2009) found it valid for andosol without any degradation term, indicating no detectable physicochemical or microbial breakdown; lixiviation was the only meaningful depletion pathway, and projected contamination endured for decades to centuries according to organic carbon content. Prior deep tillage had already dispersed residues through the profile, making mechanical removal unrealistic.

The institutional sequence is the clearest case in the sample of harm produced by delay where the knowledge already existed: seventeen years separated prohibition in the reference jurisdiction from withdrawal in the overseas departments. On 11 March 2025 the Paris Administrative Court of Appeal held the French state at fault for authorising sale, permitting prolonged use, and failing to assess the resulting pollution, awarding up to €10,000 for anxiety-related moral injury to eleven of approximately 1,300 claimants; the state appealed. An earlier criminal action was dismissed on limitation grounds in 2023.

3.3 Paraquat in occupational agriculture (2001–2026)

Paraquat dichloride offers the clearest contemporary instance of litigation outpacing regulation. The bipyridylium herbicide generates superoxide through redox cycling and is taken up selectively by substantia nigra dopaminergic neurons via the dopamine transporter, producing the oxidative mitochondrial injury underlying its association with Parkinson's disease.

Latency between occupational exposure and clinical parkinsonism is measured in decades, which places the compound at the long-latency pole alongside the organochlorines, notwithstanding its acute lethality on ingestion.

Paraquat is prohibited in the European Union and numerous other jurisdictions and remained registered in the United States throughout the study window. Facing claims from more than six thousand Parkinson's patients, the manufacturer signed a letter agreement in April 2025 and a master settlement agreement later that year, resolving claims while maintaining that no peer-reviewed analysis has established causation (Gillam, 2025).

The epistemic position is worth stating plainly. A civil standard of proof has produced a substantial transfer of resources to claimants; the regulatory standard, applied to the same body of evidence, has not produced withdrawal. The divergence arises from how the burden of proof is allocated; the underlying science before each forum is the same.

3.4 Dioxin and the legacy of Agent Orange in southern Viet Nam (dispersal 1961–1971; outcomes 2001–2026)

Agent Orange, a 1:1 formulation of 2,4-D and 2,4,5-T, was contaminated during manufacture with 2,3,7,8-tetrachlorodibenzo-p-dioxin. The exposure closed in 1971; inclusion rests on the eligibility criterion that the outcome literature and remediation response fall within the window, and on the case's value as the limiting instance of persistence.

TCDD is a high-affinity ligand of the aryl hydrocarbon receptor. Binding promotes nuclear translocation, dimerisation with the AhR nuclear translocator, and transcriptional activation at dioxin response elements, altering expression of xenobiotic-metabolising enzymes, growth factors, and hormone-responsive genes. Because AhR signalling participates in normal developmental patterning, receptor-mediated toxicity provides a route by which ancestral exposure may influence development in later generations. The compound is lipophilic and metabolically resistant, with a human elimination half-life of years and a subsurface soil half-life considerably longer.

Dwernychuk et al. (2002) measured TCDD in soil, fish fat, duck fat, pooled human blood, and breast milk collected between 1996 and 1999 at A So village in the Aluoi Valley, site of a former United States Special Forces base used for Agent Orange storage and sprayed between 1965 and 1970. Concentrations were markedly elevated relative to surrounding areas and to urban background. The authors concluded that TCDD can be mobilised from subsurface reservoirs decades after deposition through ordinary soil disturbance and re-enter the human food chain. A former storage site thus functions as a chemical reservoir with no natural expiry, converting a historical military decision into a continuing agricultural exposure hazard.

Health outcomes are synthesised biennially by the National Academies under the Agent Orange Act of 1991. The eleventh update graded candidate outcomes into categories of association, retaining sufficient evidence of association for soft-tissue sarcoma, non-Hodgkin lymphoma, chronic lymphocytic leukaemia, and chronic hypertension, with limited or suggestive evidence for a longer list (National Academies of Sciences, Engineering, and Medicine, 2018).
That series carries an analytic significance beyond its findings. It demonstrates that sustained, funded, statutorily mandated evidence synthesis for a legacy exposure is achievable; its absence in the other settings examined here therefore reflects political choice, the method being available to any of them.

Compensation remains structurally asymmetric. United States veterans access presumptive service connection, under which listed conditions require no individual proof of exposure causation. Vietnamese civilians exposed to the same agent in the same places have no equivalent entitlement, and remediation has proceeded as bilateral project aid at specific airbase hotspots.

3.5 Glyphosate in Colombian aerial eradication and South Asian paddy cultivation (2001–2026)

This setting differs from the preceding ones in that the exposure takes the form of a sustained regulatory posture with no bounded terminating event, and it is retained because the question of how evidence accumulates against a currently licensed product is answered differently here than in the historical cases.

The Colombian aerial eradication programme under Plan Colombia sprayed glyphosate formulations over coca-growing municipalities, generating a natural experiment in population-level herbicide exposure. Exploiting variation in spraying across municipality and time within a panel of individual health records, Camacho and Mejía (2017) found that increased spraying raised consultations for dermatological conditions and the incidence of miscarriage, with results robust to individual fixed effects and to control for the extent of coca cultivation. Evidence for respiratory outcomes was weaker.

The fixed-effects design substantially reduces the confounding that limits most pesticide epidemiology, and its ROBINS-E appraisal is correspondingly more favourable than for the cross-sectional designs elsewhere in the sample (Table 2). Its principal limitation lies in the outcome measure, since consultation counts capture care-seeking behaviour alongside morbidity.

In South Asia, the epidemic of chronic kidney disease of unknown aetiology among paddy farmers in Sri Lanka's North Central Province, reported from the mid-1990s and absent the conventional risk factors of diabetes and hypertension, prompted the hypothesis that glyphosate acquires nephrotoxicity through metal chelation. Jayasumana et al. (2014) proposed that the compound forms stable complexes with the calcium, magnesium, strontium, and iron of locally hard groundwater and with arsenic and cadmium, these complexes resisting degradation and delivering metal to renal tubular tissue; the correspondence between groundwater hardness and disease distribution was offered in support.

That paper is explicitly hypothesis-generating and must be weighted as such. It is mechanistically coherent and epidemiologically suggestive; it does not establish causation, and ROBINS-E does not apply to it. A subsequent case-control study in Padavi-Sripura found occupational herbicide exposure and well-water consumption associated with the disease, though with high risk of bias from self-reported exposure (Jayasumana et al., 2015). Parallel epidemics in Andhra Pradesh and Mesoamerica suggest a shared aetiological structure in which heat stress, dehydration, and agrochemical exposure interact.

3.6 The East Palestine derailment, Ohio (2023; outcomes 2023–2026)

On 3 February 2023, thirty-eight cars of Norfolk Southern train 32N derailed at East Palestine, Ohio; five tank cars contained vinyl chloride monomer. Three days later the local incident commander authorised a vent-and-burn of those cars, releasing over a million pounds of vinyl chloride and its combustion products.

Vinyl chloride is a genotoxic human carcinogen. Hepatic cytochrome P450 2E1 oxidises it to chloroethylene oxide, a reactive epoxide forming DNA adducts and underlying the association with hepatic angiosarcoma and hepatocellular carcinoma. Combustion adds hydrogen chloride, phosgene, and carbonyl compounds including acrolein.

Independent exposure assessment began within weeks. Combining Environmental Protection Agency stationary monitoring with mobile proton-transfer-reaction time-of-flight mass spectrometry, Oladeji et al. (2023) reported mean concentrations of benzene, toluene, xylenes, and vinyl chloride below Agency for Toxic Substances and Disease Registry minimal risk levels for intermediate and chronic exposure, while nine of fifty measured species exceeded local baseline. Acrolein was most prominent, reaching up to six times background near the site during daytime with nocturnal return to baseline; non-targeted analysis identified further compounds above background.

The study is methodologically strong for exposure characterisation and explicit about its limits: short-duration measurement cannot establish longitudinal exposure, and health-relevant concentrations for some species fall below conventional detection limits. Outcome research has accordingly moved to biomarkers and organ-specific longitudinal designs, with federally funded cohorts examining hepatic and thyroid function and a dedicated research programme office established in 2026 to coordinate follow-up. Given a latency of one to two decades for the cancers of concern, the principal outcome question cannot be answered within this window.

The institutional findings are unusually direct. The National Transportation Safety Board determined that an overheated wheel bearing caused the derailment; that the train was not classified as a high-hazard flammable train although the resulting fires resembled the scenarios those rules were designed to prevent; and that the vent-and-burn was unnecessary to prevent a polymerisation-induced tank failure, the incident commander having acted on incomplete and misleading information from the carrier and its contractors, with the tank car owner's assessment of low explosion risk never reaching him (NTSB, 2024).

Two features distinguish this case from the long-latency settings. Definitive public findings issued seventeen months after the event, the shortest interval in the sample. And a class action settlement of approximately $600 million was reached within roughly fifteen months, before the outcome science had matured at all. The determinant of both was the existence of an investigative body holding jurisdiction together with a statutory duty to publish.

3.7 The Tianjin Port explosions, China (2015; outcomes 2015–2026)

On 12 August 2015 a fire at a hazardous materials warehouse operated by Ruihai International Logistics in the Dongjiang bonded port area produced two explosions thirty-three seconds apart, the second involving detonation of approximately 800 tonnes of ammonium nitrate (Zhao, 2016). The official toll was 173 dead, 720 injured, and 70 missing, the fatalities falling overwhelmingly on firefighters who had applied water to a fire involving calcium carbide and other water-reactive materials. Seventeen thousand housing units were damaged and six thousand residents displaced.

The stored inventory included sodium cyanide alongside ammonium, potassium, and sodium nitrate and calcium carbide. Within a week the municipal environmental authority reported cyanide in the adjacent watercourse and in coastal waters at the edge of the evacuation zone exceeding tolerance thresholds by a wide margin. Cyanide binds the ferric iron of cytochrome c oxidase, arresting mitochondrial electron transport and producing histotoxic hypoxia; its environmental behaviour, however, inverts that of the organochlorines, degrading and dispersing over days to weeks. Tianjin thus serves as the sample's persistence-null and latency-null comparator: catastrophic acute harm, modest chronic legacy, and a hazard profile dominated by blast and by responder exposure.

The failure lay in siting and licensing. The warehouse held hazardous materials in quantities and combinations exceeding its authorisation, at a separation distance from residential buildings well below that required by national rules. No scientific uncertainty attended any element: the incompatibility of water-reactive materials with conventional firefighting, the detonability of bulk ammonium nitrate, and the required separation distances were all settled knowledge. The outcome was determined before the fire began.

4. Cross-Case Findings

Table 1 arrays the seven settings across the comparison dimensions; Table 2 reports the risk-of-bias appraisal. Three findings follow from the comparison, and we state them here without restating the case material.

First, delay did not vary with evidentiary strength. The two variables run in opposite directions across much of the sample. Chlordecone has the most rigorous evidence base and among the longest delays; Tianjin involved no contested science and a complete institutional failure; East Palestine produced peer-reviewed exposure assessment within five months and definitive accident findings within seventeen. The distinguishing feature of the rapid cases is the presence of a body holding both jurisdiction and a duty to publish. Because this pattern appears at the acute pole as well as the chronic one, latency alone does not explain it.

Second, latency and persistence together determine whether prohibition ends exposure. Chlordecone showed no detectable degradation in andosol (Cabidoche et al., 2009); TCDD remained mobilisable from subsurface reservoirs after three decades (Dwernychuk et al., 2002); endosulfan and its sulfate persisted in water and blood ten months after the final spray and at an uncontrolled disposal point two decades later. For these agents, withdrawal terminates the source while leaving the exposure in place—which has direct implications for how compensation and remediation should be designed. Cyanide at Tianjin, degrading within weeks, marks the opposite limit.

Third, the design of entitlement predicts compensation outcomes more reliably than the strength of the scientific case. Where eligibility is presumptive, defined by geography, period, and a schedule of conditions, recovery is routine: United States veterans under the Agent Orange Act, and East Palestine residents under a class settlement reached before the outcome science matured. Where eligibility requires adjudication of individual causation, recovery is rare: eleven of approximately 1,300 chlordecone claimants, and a Kerala compensation order enforced through contempt proceedings and still being executed in tranches nine years on. The evidentiary strength underlying the chlordecone claims exceeds that underlying the veterans' presumptions by a wide margin, and the outcomes run the other way.

Table 1
Comparative profile of seven agrochemical and industrial chemical exposure settings, 2001–2026, ordered by outcome latency.

Setting, period, and latency class	Chemical agent and mechanism	Primary health impacts	Ecological damage and persistence	Institutional response and interval
Kasaragod, Kerala, India. Application 1978–2001; outcomes 2001–2026. Long latency.	Technical endosulfan and endosulfan sulfate. $GABA_a$ receptor chloride-channel antagonism; interference with steroidogenesis and androgen signalling.	Higher prevalence of neurobehavioural disorder, congenital malformation and male reproductive abnormality than reference population (NIOH, 2002); delayed sexual maturation and reduced serum testosterone in exposed boys (Saiyed et al., 2003).	Residues in groundwater, surface water and human blood 10 months after final spray; uncontrolled bulk disposal in a disused well; 11 panchayats affected across approx. 4,700 ha.	Aerial spraying halted 2001 by court order; national ban 2011; Stockholm Annex A listing 2011. Compensation ordered 2017, incomplete in 2026 after contempt proceedings. Interval approx. 20 years.
Guadeloupe and Martinique, France. Application 1972–1993; outcomes 2001–	Chlordecone (Kepone). Estrogenic and progestagenic endocrine disruption; IARC Group 2B.	Prostate cancer risk rising with plasma concentration (OR 1.77, 95% CI 1.21–2.58) (Multigner et al., 2010);	No detectable degradation in andosol; lixiviation the sole depletion pathway; contamination projected over	Prohibited in the USA 1976; authorisation renewed in the French West Indies until 1993. State held at fault 11 March 2025; €10,000 awarded to 11 of

2026. Long latency.		shortened gestation and 60% increased preterm birth risk (Kadhel et al., 2014).	decades to centuries (Cabidoche et al., 2009). Detectable in over 90% of the adult population.	approx. 1,300 claimants; state appealed. Interval 17 years after reference-jurisdiction ban.
Americas, Asia and Europe, occupational, 2001–2026. Long latency.	Paraquat dichloride. Redox cycling generating superoxide; dopamine-transporter-mediated uptake into substantia nigra neurons.	Parkinson's disease among long-term agricultural applicators; over 6,000 US claimants at the time of settlement.	Strong soil adsorption limiting mobility; acute lethality on ingestion a substantial rural mortality burden.	Banned in the EU and numerous jurisdictions; still registered in the USA through 2026. Master settlement agreement signed 2025 without admission of causation. Interval unresolved.
Southern Viet Nam. Dispersal 1961–1971; outcomes and remediation 2001–2026. Multigenerational latency.	TCDD contaminant of 2,4-D / 2,4,5-T formulation. High-affinity aryl hydrocarbon receptor agonism altering developmental gene expression.	Soft-tissue sarcoma, non-Hodgkin lymphoma, chronic lymphocytic leukaemia and chronic hypertension graded sufficient evidence of association; further outcomes limited or suggestive (National Academies, 2018).	TCDD reservoirs in former base soils mobilisable after three decades and re-entering the food chain via fish, poultry and breast milk (Dwernychuk et al., 2002).	Spraying ended 1971. Presumptive compensation for US veterans under the Agent Orange Act 1991; no equivalent entitlement for Vietnamese civilians. Hotspot remediation as bilateral project aid.
Colombia (aerial eradication); Sri Lanka and Andhra Pradesh (paddy cultivation), 2001–2026. Mixed latency.	Glyphosate formulations; hypothesised nephrotoxic metal–glyphosate complexes in hard groundwater.	Increased dermatological consultations and miscarriage incidence with spraying intensity (Camacho & Mejía, 2017); chronic kidney disease of unknown aetiology among paddy farmers (Jayasumana et al., 2014, 2015).	Soil and groundwater residues; co-occurrence with arsenic and cadmium in endemic areas; drift beyond target parcels.	Colombian aerial programme suspended 2015 then contested; glyphosate remains registered in most jurisdictions. No compensation mechanism. Interval unresolved.

East Palestine, Ohio, USA. 3 February 2023; outcomes 2023–2026. Mixed latency.	Vinyl chloride monomer and combustion products (hydrogen chloride, phosgene, acrolein). CYP2E1 oxidation to chloroethylene oxide forming DNA adducts.	Acute headache and respiratory, dermal and ocular irritation; hepatic angiosarcoma and hepatocellular carcinoma the latent concerns driving ongoing cohort follow-up.	Over one million pounds of vinyl chloride released by vent-and-burn; soil and watercourse contamination; indoor residues detectable over two years later. Short environmental half-life relative to organochlorines.	NTSB (2024): overheated wheel bearing; train not classified high-hazard flammable; vent-and-burn unnecessary and based on incomplete and misleading carrier information. Class settlement of approx. $600m within 15 months. Interval 17 months to findings.
Port of Tianjin, China. 12 August 2015; outcomes 2015–2026. Acute; no meaningful latency.	Ammonium nitrate (approx. 800 t detonated), sodium cyanide, calcium carbide, nitrates. Cyanide inhibition of cytochrome c oxidase; blast overpressure.	173 deaths, 720 injuries, 70 missing, predominantly firefighters; blast and burn trauma; acute cyanide exposure risk in the evacuation zone (Zhao, 2016).	Cyanide in the adjacent watercourse and coastal waters far above tolerance thresholds; 17,000 housing units damaged, 6,000 residents displaced. Rapid environmental degradation.	Criminal prosecution of company and local officials; national revision of hazardous storage siting rules. Failure located in licensing and inspection. No scientific uncertainty involved.

Note. Intervals are measured from the first peer-reviewed or official report of population-level harm to the first binding national restriction, and are approximate where the date of first credible signal is contested (see Section 7). OR = odds ratio; CI = confidence interval; IARC = International Agency for Research on Cancer; NTSB = National Transportation Safety Board; TCDD = 2,3,7,8-tetrachlorodibenzo-p-dioxin.

Table 2

Risk-of-bias appraisal of the primary studies supporting each case, by ROBINS-E domain.

Study and design	Confounding	Exposure measurement	Participant selection and missing data	Outcome measurement	Overall judgement

Saiyed et al. (2003). Cross-sectional, exposed vs. reference village, male children aged 10–19.	Some concerns. Age adjusted; residual socio-economic and nutritional confounding not excluded.	High. Exposure assigned at village level; no individual dose reconstruction, though residues confirmed in blood and water.	Some concerns. Reference village selected by investigators; participation not fully reported.	Low. Sexual maturity rating applied by trained examiners with assayed serum hormones.	High risk of bias. Direction of association credible and internally consistent; magnitude not reliably estimable.
Multigner et al. (2010). Hospital-based case-control, 623 cases and 671 controls, with genotyping.	Low. Adjusted for age, family history and known covariates; genotype interaction examined.	Low. Plasma chlordecone concentration plus cumulative exposure index.	Some concerns. Hospital-based control selection may not represent the source population.	Low. Histologically confirmed prostate cancer.	Some concerns. The most robust exposure–outcome evidence in the sample.
Kadhel et al. (2014). Prospective mother-child cohort, 818 pregnancies (Timoun).	Low. Adjusted for principal obstetric and socio-demographic covariates; co-pollutants measured.	Low. Maternal plasma concentration at delivery, with co-exposures quantified.	Some concerns. 818 of 1,068 enrolled contributed; attrition not fully characterised.	Low. Gestational age from clinical records; preterm birth objectively defined.	Some concerns. Effect estimates precise and biologically coherent.
Camacho & Mejía (2017). Panel of individual health records with municipality-level exposure and individual fixed effects.	Low. Individual fixed effects compare the same person across exposure levels; coca cultivation controlled.	Some concerns. Municipality-level spraying intensity proxies individual exposure.	Some concerns. Restricted to individuals presenting to the health system.	High. Consultation counts conflate morbidity with care-seeking behaviour.	Some concerns. Strong internal validity for the design; outcome measure is the limiting factor.
Jayasumana et al. (2015). Case-control, agricultural workers, Padavi-Sripura.	High. Limited adjustment for heat stress, dehydration and co-exposures.	High. Self-reported herbicide use and water source.	Some concerns. Recruitment via clinical services.	Low. Serum creatinine and biopsy-supported case definition.	High risk of bias. Consistent with the hypothesis; not confirmatory.
Jayasumana et al. (2014).	Not applicable.	Not applicable.	Not applicable.	Not applicable.	ROBINS-E not

Hypothesis paper proposing a metal-chelation mechanism.					applicable: no original exposure–outcome analysis. Treated as hypothesis-generating.
Dwernychuk et al. (2002); Oladeji et al. (2023). Environmental and ambient-air monitoring.	Not applicable.	Low. Direct analytical measurement in environmental and biological media.	Not applicable.	Not applicable.	ROBINS-E not applicable: exposure characterisation without health outcome analysis.

Note. Appraisal performed by the authors using the domains of the ROBINS-E tool for non-randomised studies of exposure. Judgements are reported at domain level and summarised overall. ROBINS-E does not apply to studies reporting no exposure–outcome association; these are listed for completeness because their evidentiary status bears on the weight given to the corresponding cases.

5. Regulatory Failure and Environmental Justice

5.1 The allocation of the burden of proof

The pesticide registration systems examined here share a structural feature. A licensed product may remain in commerce until harm is demonstrated to an administrative standard, and the burden of that demonstration falls on parties other than the licensee.

The practical consequences follow from the properties of the agents. Detecting a latent multifactorial endpoint at population scale requires a sizeable exposed cohort, individual exposure reconstruction, and years of follow-up. In an agricultural district of northern Kerala, or a banana-growing commune in Martinique, that capacity does not exist locally. It must be imported, and it typically arrives only once harm has become visible enough to generate political attention.

The interval during which the burden cannot be discharged is therefore not neutral waiting time. It is the period in which exposure continues, and it constitutes a substantial share of the eventual harm.

Scientific uncertainty is the normal condition of toxicology. Under this allocation of the burden, that normal condition operates as an asset to the party favouring continued registration. The paraquat record illustrates the mechanics: internal corporate documents obtained and published by journalists indicated awareness of research linking the compound to Parkinson's disease and efforts to shape the scientific and public record, while the public position maintained that no peer-reviewed analysis had established causation (Gillam, 2025). Both statements may be literally accurate at once, and the standard that permits this is the proper object of criticism.

5.2 Accountability where the regulator is the polluter

The settings divide instructively on the identity of the responsible actor. In two—paraquat and East Palestine—a private corporation was the proximate agent, and in both the mechanism that produced results was civil litigation, with regulatory enforcement playing no material part.

In three—Kasaragod, the French West Indies, and Viet Nam—an organ of the state was the applicator or authorising authority, which generates a conflict the compensation records make plain. In Kerala the spraying entity was a state-owned corporation and the compensating entity was the state; the 2017 direction required contempt proceedings against the Chief Secretary, and sanctions were still issuing in tranches in 2026. In France the authorising ministry and the defendant before the Paris Administrative Court of Appeal were the same state, which appealed the 2025 finding

against it.

Where polluter and regulator coincide, the polluter-pays principle reduces to a transfer between departments, and the claimant's remedy depends on judicial willingness to compel an unwilling executive.

Tianjin presents a third configuration: a private operator holding authorisations that did not cover its actual inventory, in a jurisdiction whose inspection regime had not detected the discrepancy. Criminal prosecutions of company and local officials followed. Prosecution after the fact, however, does not address the mechanism of death, which was the absence of accurate inventory information at the moment of response.

5.3 Distributional patterns

The affected populations were not a random sample of their respective states. Kasaragod's exposed group comprised agricultural labourers and smallholders in a district peripheral to Kerala's economic centres, who required three decades of organised mobilisation—including a hunger strike before the State Secretariat in 2022—to secure implementation of an existing judicial order.

Guadeloupe and Martinique are overseas departments whose populations are predominantly of African descent, and in which a pesticide prohibited in the metropolitan reference jurisdiction remained authorised for a further seventeen years. The Colombian spraying programme covered municipalities inhabited substantially by smallholders. Sri Lanka's disease burden falls on paddy farmers. Vietnamese civilians have no analogue of the entitlement available to veterans of the state that dispersed the agent. East Palestine is a village of roughly 4,700 people, and Tianjin's casualties fell on firefighters and on residents of housing built within a kilometre of a hazardous materials warehouse.

Consistency of this kind across seven settings, four continents, and five legal systems indicates something more systematic than coincidence. Two mechanisms suffice to generate it without any need to posit intent.

Hazardous activity is sited according to land and labour costs, which places it where the political capacity to object is weakest. And the capacity to discharge the burden of proof described above—to commission epidemiology, retain counsel, and sustain litigation across decades—is distributed in proportion to the resources of the exposed population. A formally uniform standard of proof therefore delivers substantively unequal protection, which is the operative definition of structural environmental injustice.

5.4 The reach of international instruments

The Stockholm Convention is the principal multilateral instrument addressing persistent organic pollutants, and its listing of technical endosulfan in Annex A by decision SC-5/3 was consequential (UNEP, 2011). Three limits are visible in these cases.

Listing operates prospectively. It restricts production and use while creating no remediation obligation for contaminated media and no compensation entitlement for those already exposed. Specific exemptions permit continued use for designated crop-pest complexes, extending exposure precisely in the jurisdictions least able to fund substitution; the accompanying work programme recognised the need for cost-effective alternatives without financing their deployment. And the Convention reaches agrochemicals meeting persistence and bioaccumulation criteria while leaving the high-volume industrial chemicals implicated at East Palestine and Tianjin to national transport and storage regimes of widely varying rigour.

The resulting architecture addresses the agents of the 1970s with reasonable effect and the exposure pathways of the 2020s considerably less so.

6. A Framework Addressed to the Interval

The comparison locates the harm in a specific place: the interval between the appearance of a credible signal and the operation of an effective remedy. The four instruments below are specified accordingly, with attention to the actor that would carry each. They are offered for deliberation and none is presented as a costed programme, since the comparison supports the diagnosis more strongly than it supports any particular remedy.

6.1 Sentinel biomonitoring in exposed agricultural districts

Every long-latency case in this review was detected by clinicians, parents, and local journalists. No surveillance system identified any of them. Formalising that detection capacity is the highest-yield intervention available.

We propose a district-level sentinel network in areas of intensive pesticide application, combining three elements. The first is a congenital anomaly register with mandatory reporting from all delivery facilities, including a standard panel of endocrine-sensitive endpoints such as hypospadias, cryptorchidism, and anogenital distance. The second is periodic residue monitoring of groundwater, surface water, and breast milk at fixed sampling points. The third is a defined statistical trigger—exceedance of expected anomaly prevalence or of residue reference values—obliging the public health authority to open an investigation within a stated period and to publish the result irrespective of finding.

The third element carries most of the weight. Our cross-case finding was that speed depended on an institution holding jurisdiction together with a publication duty; a monitoring system without a mandatory trigger would reproduce the discretion that generated the delay.

Breast milk monitoring merits particular emphasis. It integrates maternal body burden over time, requires no invasive procedure, and samples the exposure route most relevant to the developmental endpoints at issue. It is also the measurement that would have detected the Kasaragod exposure years before it was detected.

6.2 Reversing the burden for persistent bioaccumulative agents

The precautionary principle is widely endorsed and seldom operationalised. A workable formulation for this agent class would provide that where a compound satisfies defined persistence and bioaccumulation criteria—a soil half-life exceeding one year, say, or a bioconcentration factor above a specified threshold—registration for open environmental application requires affirmative demonstration of developmental and endocrine safety by the registrant, at the registrant's expense, as a condition of licence.

This is already the standard applied to pharmaceuticals. The asymmetry between the two regimes is difficult to justify on grounds other than historical accident.

Two corollaries follow from the cases. Aerial application over terrain with human habitation, open water, or significant relief should require site-specific authorisation with an affirmative suitability finding: the Kasaragod exposure was produced as much by method and terrain as by molecule, and a state committee reached exactly that conclusion in 2001, after two decades of spraying. Registration decisions should also be subject to periodic review on a fixed cycle with automatic sunset absent renewal, converting the default from continued authorisation to lapse. Repeated administrative renewal maintained chlordecone authorisation for seventeen years after the reference jurisdiction had prohibited it; a sunset default would have forced an affirmative decision to continue, with the accountability that entails.

6.3 Presumptive, portable entitlement for long-term rehabilitation

Our third cross-case finding identifies entitlement design as the dominant predictor of compensation outcomes, and the framework should follow that finding directly.

Four features appear necessary. Eligibility should be presumptive, defined by residence within a mapped exposure zone during a defined period together with a diagnosis on a published schedule of conditions, the schedule revised as evidence develops. Entitlement should run to continuing care in place of a single payment, since neurological disability, developmental impairment, and renal failure require lifetime management and a lump sum discharges the payer's obligation without meeting the recipient's need.

The fund should sit outside the annual budget of the department that authorised the exposure, financed by levy on the relevant product class, so that payment does not depend on the fiscal position or the willingness of the responsible agency. The Kerala sequence, in which a judicial order required contempt proceedings and remained in partial execution nine years later, is the direct argument for this feature.

Caregiver recognition is the fourth. The care burden in affected households falls predominantly on women, is unremunerated, and is omitted from every scheme examined here. A scheme that supports the patient and ignores the carer understates household cost substantially.

6.4 Financed agroecological substitution, and two measures for industrial release

Prohibition without an available alternative displaces risk instead of reducing it, and the specific exemptions in the endosulfan listing exist because substitutes were neither identified nor affordable at the point of decision. Effective substitution requires that the alternative be developed and financed before prohibition takes effect.

The instruments are known: integrated pest management with biological control agents and pheromone-based monitoring, extension services equipped to support transition, and transition payments covering the yield risk of the first cycles. The binding constraint is finance. One plausible mechanism is a levy on production of listed and candidate compounds, hypothecated to substitution finance in jurisdictions holding exemptions, which aligns the cost of transition with the revenue from delay.

The industrial cases imply two further measures. Tianjin was a siting and inventory failure, which argues for mandatory separation distances enforced through land-use law and not through facility licensing alone, and for real-time hazardous inventory data accessible to responding fire services at dispatch. East Palestine was a failure of information transfer between carrier and public incident commander, which argues for emergency decisions of that class to require documented independent technical concurrence before execution—addressing the specific gap identified when the tank car owner's low-risk assessment never reached the decision-maker (NTSB, 2024).

7. Limitations

Four limitations bear on the inferences available from this design.

The sample is purposive rather than exhaustive. Seven settings were selected for variation across agent class, latency, and jurisdiction, and they are not representative of the population of chemical exposure events in any statistical sense. The cross-case findings should be read as hypotheses carrying comparative support, and not as estimated frequencies. A formal systematic review with registered protocol and reported screening flow would be required to establish prevalence, and we have not attempted one.

Selection favoured documented cases, which introduces a predictable bias. A setting enters this sample only if a scientific or institutional record exists, so the cases are systematically drawn from the subset in which some institutional response eventually occurred. Events producing comparable harm without generating a record are invisible to this design, and their omission almost certainly understates the typical duration of delay.

The delay interval is measured imprecisely. Its start point—the first credible signal of population-level harm—is contested in several cases, particularly where local clinical observation preceded formal publication by years, as at Kasaragod. We have used the earliest documented report in each case and flagged the contested instances, but the measure should be treated as ordinal rather than cardinal, and the comparison is accordingly drawn between magnitudes rather than between point estimates.

Finally, the evidentiary quality of the underlying studies is uneven, as Table 2 records. The chlordecone findings rest on biomarker-based designs with low risk of bias; the Kasaragod findings rest on an ecological exposure assignment carrying high risk of bias; the Sri Lankan nephropathy hypothesis rests on no exposure–outcome analysis at all. We have weighted the cases accordingly in the narrative, and the cross-case findings do not depend on the weakest studies. Readers should nonetheless treat the Kasaragod effect magnitudes, in particular, as indicative.

8. Conclusion

Seven settings differing in agent, mechanism, geography, and legal system converge on one finding. The magnitude of harm was determined less by the toxicity of the compound than by the duration of the interval between credible signal and effective response, and that interval was governed by institutional design.

The implication is that the precautionary principle is better understood as an institutional claim than an epistemic one. Stated as a rule for deciding under uncertainty, it attracts the standing objection that it would paralyse innovation. A narrower formulation survives that objection. For compounds that persist in environmental media, bioaccumulate in

human tissue, and act on developmental or endocrine pathways with latencies measured in years to generations, the conventional sequence of authorisation, observation, demonstration, and withdrawal cannot operate within the period during which harm accrues. For this bounded class, the observation phase is the exposure, and the burden of affirmative safety demonstration belongs with the party seeking authorisation.

The entitlement finding generalises further, and it is the most actionable result reported here. Compensation outcomes across these cases varied by an order of magnitude under comparable or inverted evidentiary strength, according to a single design feature: whether eligibility was presumptive or required adjudication of individual causation. That feature is within the gift of any legislature, requires no scientific advance, and could be altered in a single statute.

The populations in this review were, with striking consistency, agricultural, low-income, or constitutionally peripheral to the authority that permitted their exposure. They were also, with equal consistency, the first to identify the harm. At Kasaragod, detection ran from parents and local clinicians through a regional journalist to a national institute, to the Supreme Court of India, and finally to the Conference of the Parties—a chain that took roughly two decades to traverse and that a functioning sentinel system would have compressed to months.

Building such a system is a technical problem of modest difficulty. The reasons it has not been built in the districts that need it lie elsewhere, and naming them accurately is the first step toward a chemical governance regime in which the interval between knowing and acting is measured in months rather than generations.

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