INDIAN INSTITUTE OF SCIENCE

BENGALURU

INTRODUCTION TO
INTEGRATED ONE HEALTH - PERMACULTURE FRAMEWORK

Under the Guidance of

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Acknowledgements

This internship at the Indian Institute of Science, Bengaluru, has been one of the most enriching experiences of my academic journey, and it would be incomplete without acknowledging the people who made it so.

I am grateful to IISc and the Office of Research and Grants for the opportunity to undertake this internship, which allowed me to step outside my medical training and engage with a genuinely interdisciplinary research environment.

I owe my deepest gratitude to Prof. Sai Siva Gorthi sir, my Principal Investigator, for welcoming me into the lab, for trusting me with a project as ambitious and interdisciplinary as the One Health and for his constant guidance, patience, and encouragement throughout my time here. His willingness to let a medical intern wade into environmental data science and AI — and to genuinely engage with the ideas that came out of it — shaped this internship into something far more valuable than I could have planned for myself.

I am sincerely thankful to all the PhD scholars and project associates of the lab, who patiently answered my endless questions, helped me navigate everything from literature databases to the finer points of building an AI-based tool, and made sure I never felt out of place despite coming from a very different academic background. Their openness in sharing their own work, experiences, and knowledge made this project more memorable.

I would also like to thank the RRC-HTA team for their valuable guidance and support, which helped sharpen the health technology assessment dimensions of this work.

I gratefully acknowledge the AI tools used in generating the diagrammatic representations of key concepts in this report, which helped translate complex ideas into visuals for easier understanding.

Finally, my thanks to everyone else in the lab and at the department. This report is as much a product of their collective support as it is of my own effort.

My Experience at IISc

If you had told me before this internship that I would spend my days debating the finer points of pollutants, sources & impacts and arguing with a chatbot about whether it was giving accurate OneHealth advice, I would not have believed you. And yet, here we are.

IISc's campus deserves its reputation before anything else about the work is mentioned. Tree-lined avenues, the calm campus devoid of any chaos, it is very hard to feel stressed about a deadline when your walk to the department involves such wonderful sceneries. I quickly learned that "I'll be there in five minutes" on this campus should always be mentally revised to fifteen, on account of its sheer size alone. As a newbie, I also learned the hard way that the campus is nearly impossible to navigate without Google Maps open at all times — I lost count of how many times I confidently walked in the wrong direction with the full conviction of someone who definitely knew where they were going.

The people, though, are what actually made this internship memorable. The lab had exactly the mix I didn't know I needed: PhD students, Co interns who could explain a complex instrumentation concept in one breath and recommend the best food place on campus in the next.

There was a particular joy in the range of what a single day could involve here — one afternoon spent cross-referencing WHO and CPCB permissible limits for Air pollutants, the next spent analysing how much pollutants might possibly be in my organ systems after finding the extent of pollution among which we live in.

Somewhere between the pollutant tables, the trend graphs, and the chatbot sessions, I found that I had genuinely started to enjoy the research- knowing about the resultant pollutant of our activities how it adversely impacts a wide range of biodiversity including ourselves.

More than anything, this internship taught me that public health and environmental science are not neighbouring disciplines that occasionally wave at each other — they are the same conversation, to be viewed from the same rooms.

ABSTRACT

Environmental pollution is increasingly understood not as a set of isolated contaminant problems but as a disturbance to a single, interconnected biological system spanning humans, animals, plants, and the physical environment. This report builds its argument through a deliberate conceptual progression: it first establishes health, at the individual level, as a dynamic equilibrium, with disease as its disturbance and symptoms as its language; it then extends this same equilibrium logic to the planet as a whole, arriving at the One Health principle that human, animal, and environmental health form one interconnected system rather than three separate domains. Permaculture is the integrated planetary equivalent of the body's healing process, framing regenerative, closed-loop resource use as the practical treatment (the planetary analogue of medicine for humans) that follows from a One Health diagnosis. Using this combined One Health–Permaculture lens, the report synthesises current evidence on several major environmental pollutant classes, examining their sources, environmental transport, and documented effects on human health, wildlife, plant physiology, soil microbial communities, and aquatic and climate systems. This synthesis is supplemented by component-contaminant matrix as well as a decadal and multi-decadal trend analyses of major air, water, soil, climate, and health-related contaminants in India, set against corresponding global patterns from peer-reviewed literature and international monitoring data. Building on this evidence, the report proposes a Planetary Health Value Assessment (PHVA) framework, extending conventional Health Technology Assessment through the convergent, "binocular" lenses of One Health and Permaculture, and outlines a prototype- an Ai Chatbot – "EcoAdvisor"- a public-facing digital tool designed to translate this evidence into accessible form. The recurring pattern across the evidence — persistence, bioaccumulation, and multi-systemic harm resistant to single-domain solutions — reinforces the report's central premise that planetary and individual health follow the same underlying logic, and that restoring one increasingly depends on restoring the other.

INTRODUCTION:

Health, at its most basic, can be understood as a dynamic equilibrium: a state the body maintains and restores on its own, disturbed by disease and expressed through symptoms rather than imposed from outside by medicine alone. Antibiotics , painkillers etc, each assists a self-correcting process that the body already carries out by default. This same logic extends naturally to the planet. Just as the human body maintains equilibrium across its organs and systems, the earth maintains its own equilibrium across climate, soil, water, plants, and animals, functioning as a single interconnected whole rather than a collection of separate concerns. Disturbance of this planetary equilibrium through deforestation, industrial contamination, chemical-intensive agriculture, and the accumulation of persistent pollutants - produces symptoms in much the same way

disease does in the body: rising temperatures, biodiversity loss etc, are the planet's symptoms of its Dis-ease. This is the foundational insight of the One Health principle: that human, animal, and environmental health are not separate systems but one interconnected whole, disturbed at one point and expressed at another.

If One Health supplies the diagnosis, Permaculture supplies the corresponding logic of treatment. As a regenerative, closed-loop design philosophy in which every output becomes an input for another part of the system, Permaculture does not seek to override the earth's natural cycles but to work with them - assisting the planet's own capacity for repair rather than replacing it with external intervention. Read together, One Health and Permaculture forms a complete vision of the problem: one eye locates where ecological and biological disturbance occurs and how it propagates across species and systems, the other identifying how sustainable, self-renewing design can restore the equilibrium that disturbance has displaced. And the PHVA along with One Health Testing acts like the Binocular Lenses in which the former Evaluates and the latter acts as the Evidence .

Operationalising this dual perspective requires moving from concept to instrument, which is the motivation behind the Planetary Health Value Assessment (PHVA) framework outlined in this report — an approach to evaluating pollutants, products, and activities simultaneously through their One Health impact and their alignment with regenerative, closed-loop principles, extending existing sustainability and health-technology assessment models to capture both dimensions together.

The Research Question:

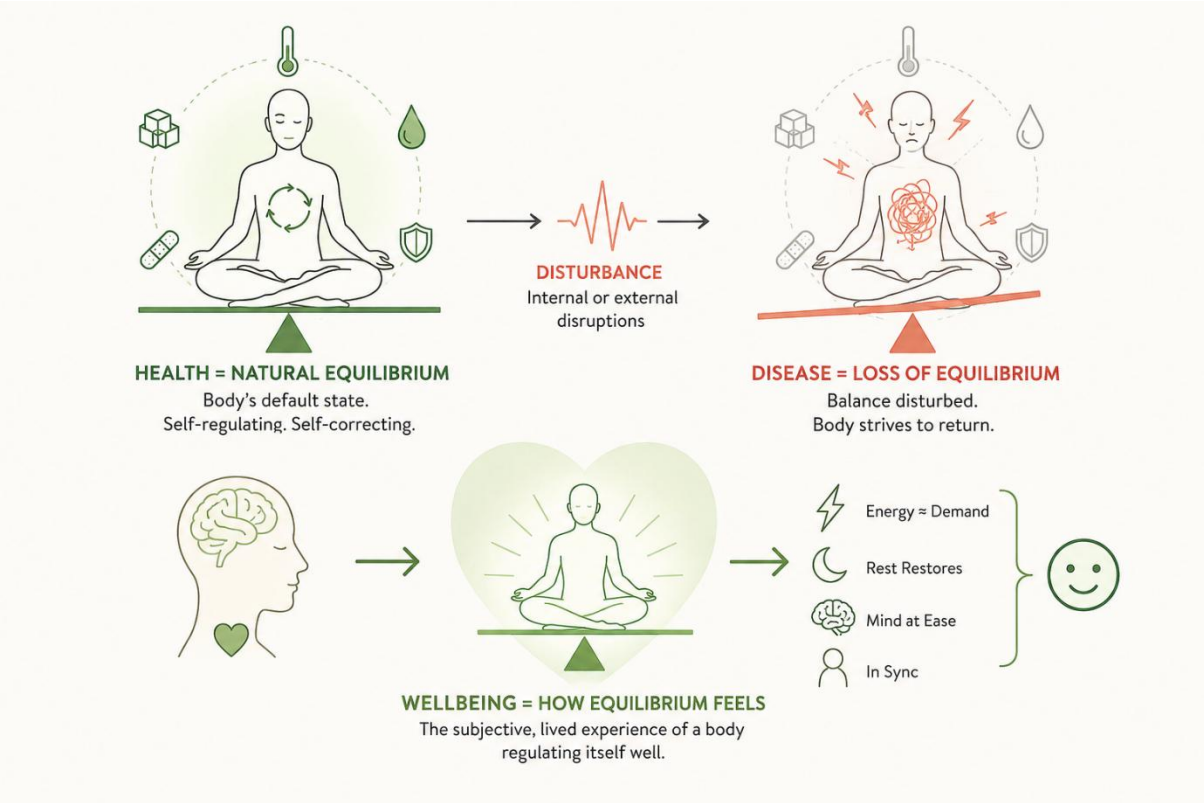
Before going into the pollutant tables, trend graphs, analysis and matrices, lets try to answer a one basic question — one that sits underneath the entire perspective of what we are focusing on - What is health, actually? Why does the world which used to be so rooted in finding the so called “cure” for the emerging and prevalent “dis-eases” end up talking about animals, soil, plants, and climate even? The answers to all these lie under the logic of One Health which only really makes sense once the logic of ordinary, individual health is made explicit first.

The Body's Natural Equilibrium:

The World Health Organization defines health as a state of complete physical, mental, and social well-being, rather than just the absence of disease or infirmity.

Thus, Health is often defined, by default, as the absence of disease — but that is a definition built backwards, from illness toward its opposite. A more useful & logical starting point is to think of health as a natural state of equilibrium: a dynamic, continuously self-correcting balance that the body maintains on its own, without instruction. Temperature is regulated, blood sugar is buffered, wounds close, infections are contained — all of this happens as the body's default operating state, not as something supplied to it from outside. Disease, in this view, literally means Dis-ease - simply a disturbance of that equilibrium; and the body's overwhelming tendency, given half a chance, is to return to it.

Wellbeing too, is a relative feeling - it is what equilibrium feels like from the inside. It is the subjective, lived experience of a system that is regulating itself well: energy that matches demand, rest that actually restores, a mind that is not constantly fighting its own body.



Symptoms: The Body's Language:

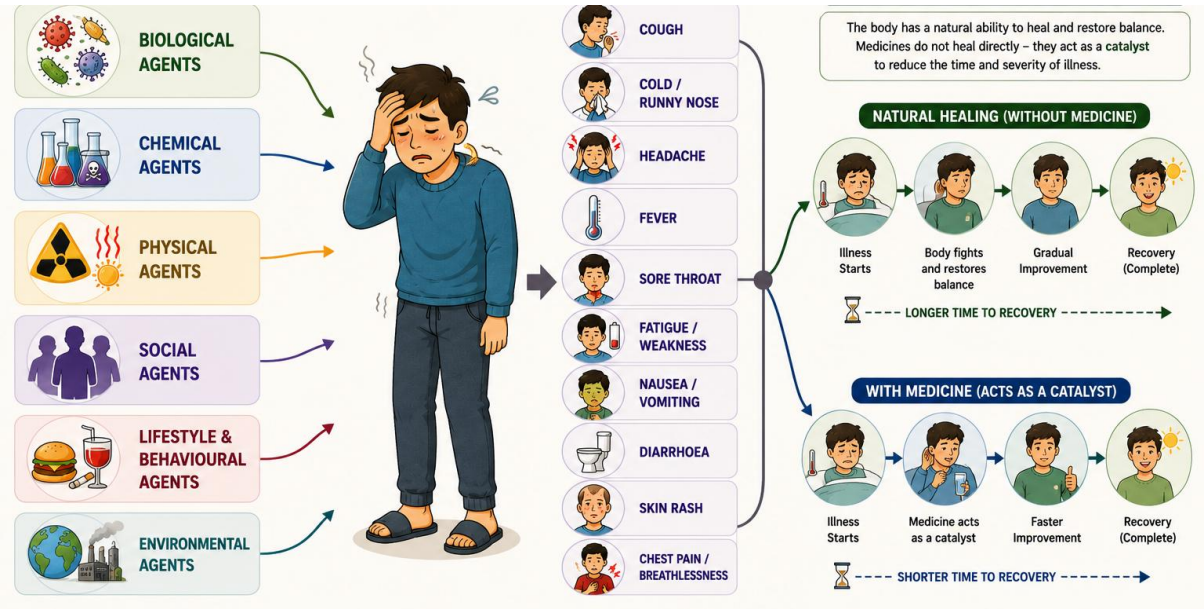
If health is equilibrium, then symptoms are what the body produces when that equilibrium is disturbed and it is trying to express itself. A fever is not a malfunction to be silenced immediately— it is a deliberate natural defensive action which rises the temperature that makes the body a more hostile environment for the pathogens. Cough, inflammation, and fatigue during illness

are not due to the disease as such ; they are largely the immune system's chosen strategy, made uncomfortable on purpose so that it cannot be ignored.

Healing-Do Medicines cure The Disease?

When someone recovers from illness, is it medicine that cures the Disease or is it their own body that heals, with medicine simply acting as a catalyst ? Antibiotics do not personally hunt down every last bacterium — they reduce the pathogen load enough that the immune system, still doing the actual work, can finish the job. A plaster cast does not knit a fractured bone back together — it holds the pieces still while the body's own cells rebuild it over weeks. Painkillers do not repair damaged tissue — they manage the symptom of pain while the underlying repair continues regardless, on its own.A vaccine doesn't fight/destroy the pathogen by itself — it pre-trains the immune system to recognize it, so the body's own defenses can respond faster and harder when the real infection actually shows up.

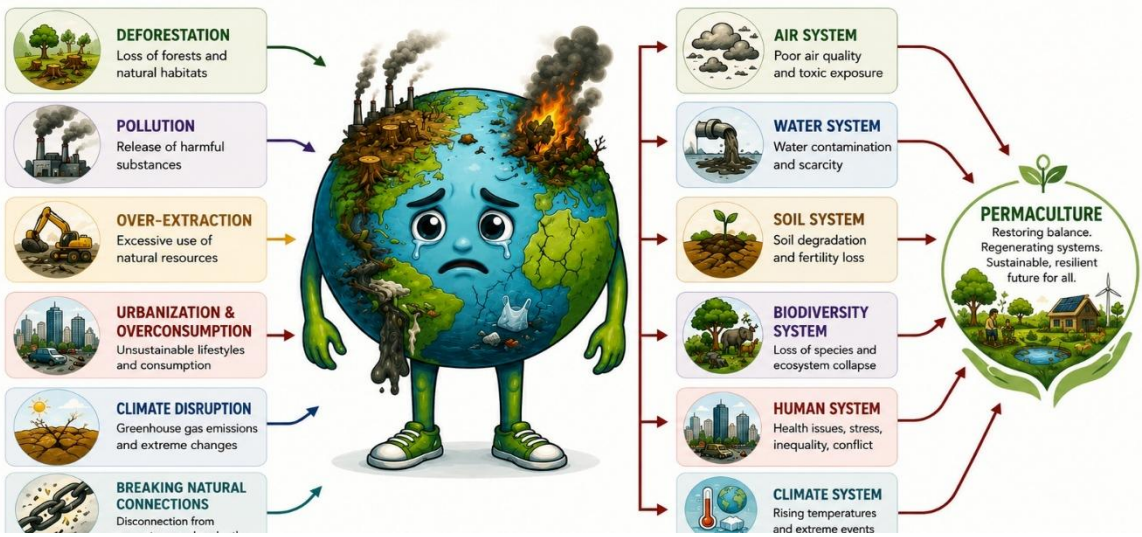
Healing, in other words, is the body's natural, default trajectory back toward equilibrium. Medicine's most honest role is not to replace that process but to assist it: to remove obstacles, to buy time, to correct what the body genuinely cannot correct on its own, basically acting as a catalyst in the process of healing and let the underlying biology do what it already knows how to do.



From One Body to One Planet:

Now let’s humanize the earth and consider it as a single being- Just as the human body maintains a natural equilibrium across its organs and systems, the earth

maintains its own equilibrium across plants, animals, environment, & all functioning together as one interconnected whole. Health, in this sense, is simply what that equilibrium looks like when it is intact. The earth's version of ‘dis-ease’, then, is not any single crisis in isolation, but the cumulative disturbance caused by man-made activity — the deforestation, pollution, and extraction that interrupt its natural healing process: the quiet, continuous cycle of resource replenishment through which the planet regenerates what is used and restores what is lost.



What’s “One Health”?

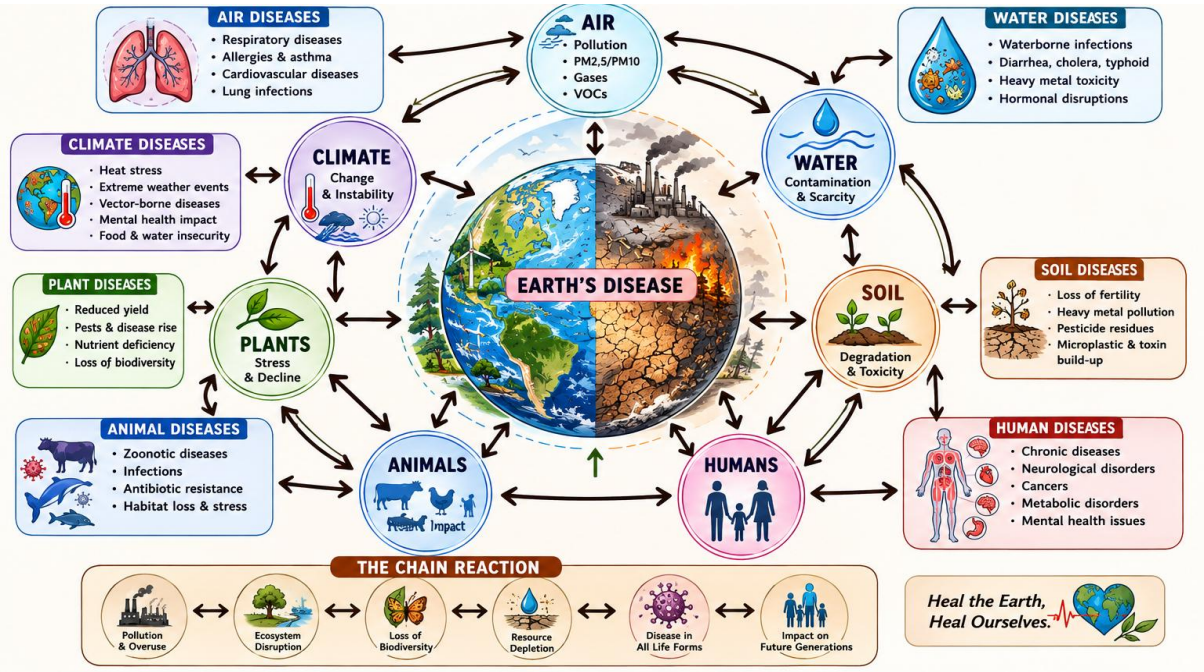
Thus, One Health, at its core, is the recognition that human health, animal health, and environmental health are not three separate systems-instead they are one interconnected system, in the same way that a person's organs are part of a single physiology. A pathogen that jumps from an animal reservoir to a human, A pesticide sprayed on a field doesn't just affect the crop; it can affect soil microbes, water sources, animals that graze nearby, and eventually the humans who eat the food or drink the water. — they're the same underlying system, disturbed at one point and expressed at another.

Why ONE HEALTH -A Selfless Act?:

Humans are, in general, a fairly self-interested species, and it would be a stretch to claim that the sudden interest in planetary health is motivated primarily by selfless concern for plants, soil bacteria, or coral reefs , climate change, etc. Looked at closely, One Health still tends to be human-centred in its foundational background: we care about zoonotic spillover because it threatens human populations; we care about antimicrobial resistance in livestock because it threatens human treatment options; we care about pesticides effect on plants

because it threatens human food security. What gets described as finding a “cure” for our external environment is, more precisely, an attempt to cure every component that makes up OUR external environment, because a sick planet eventually, unavoidably, makes for a sick human.

So, it is more apt to say that we have moved on from a narrower era of medicine, one preoccupied only with building an individual's ‘internal’ immunity, into an era where we now work to build immunity for our ‘external’ environment as well — for the animals, plants, and ecosystems we share the planet with.



Permaculture as the Earth's Medicine

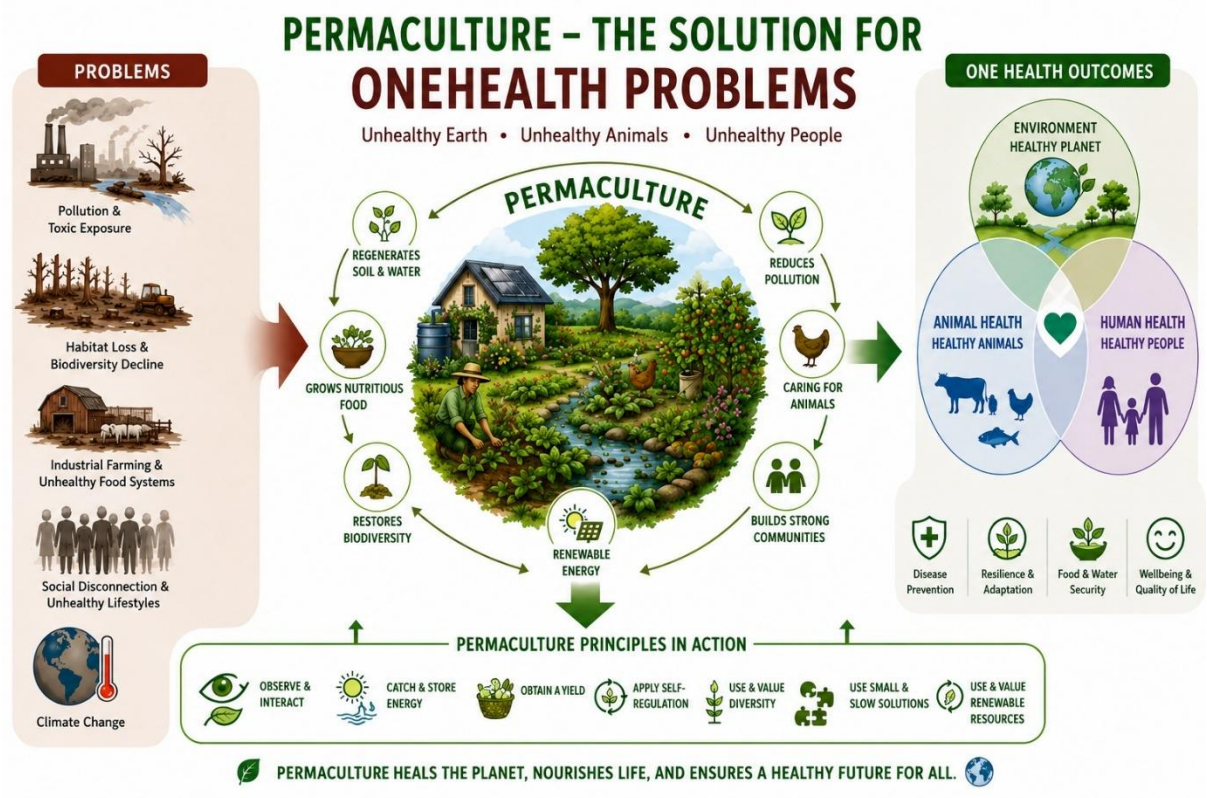
Once health is understood this way- as a property of an interconnected system, not of any one part of it in isolation - a further question becomes almost unavoidable: if a human body has a natural equilibrium that can be disturbed and can heal on its own without relying completely on an external factor, does something similar apply at a planetary scale? Can the earth itself with its own version of health, has its own capacity to heal, given the chance?

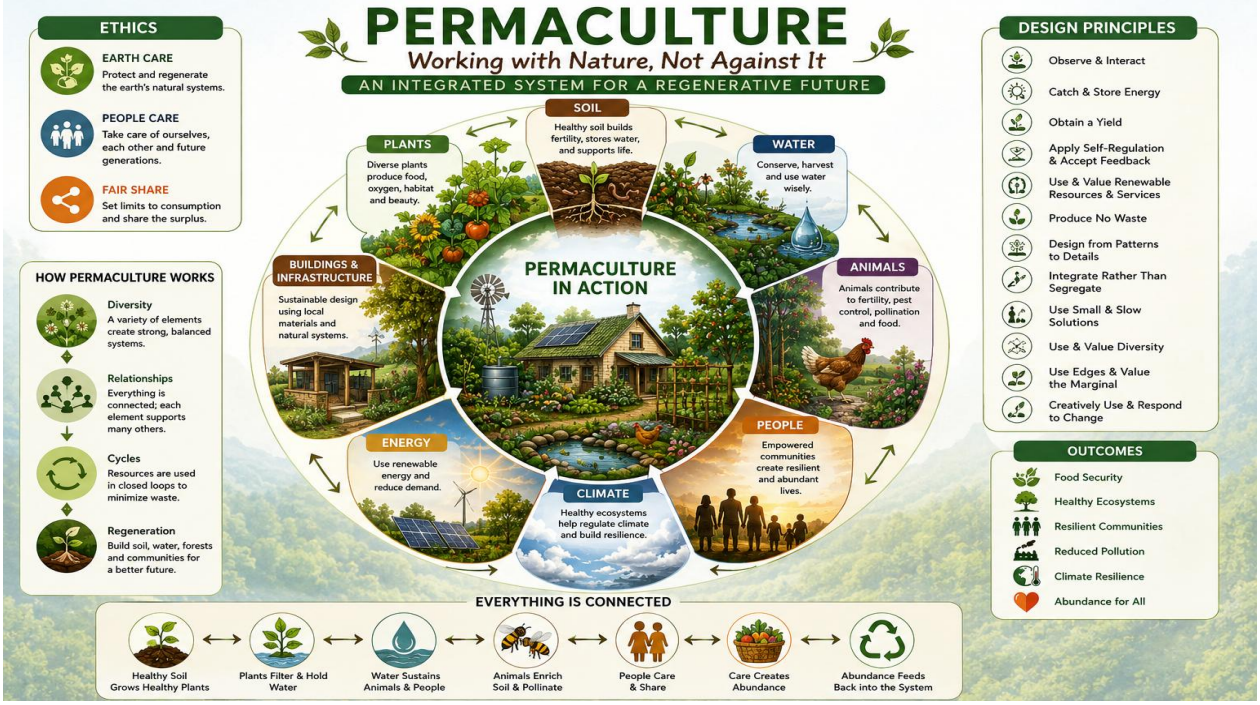
The earth, too, has a natural equilibrium: stable climate patterns, self-regulating nutrient cycles, biodiversity that keeps any one species from overwhelming the rest, etc. Much of recent human activity — deforestation, monoculture agriculture, fossil-fuel combustion, chemical-intensive farming, unmanaged waste - behaves, like a chronically persistent pathogen: not a single acute injury,

but a sustained disturbance that the planet's regulatory systems are increasingly straining to compensate for.

And just as a human body produces symptoms when its equilibrium is disturbed, the earth appears to be doing the same — rising global temperatures as its fever, extreme weather events as its pain response, mass species die-offs as a kind of organ failure- visible, hard-to-ignore signs that something underneath has shifted. These are not separate environmental “issues” to be managed separately,instead when, read through a One Health lens, they are symptoms of a single planetary “dis-ease”.

If that is the diagnosis, Permaculture is one of the more coherent answers to the question of treatment. Permaculture does not propose to override the earth's systems— it proposes to work with them: a Zero Waste Generative Solution, a Self Sustainable Closed-Loop Resource Use; A Regenerative design that restores natural cycles . It is not an immediate short term remedy, but rather it aims at a gradual long term planetary health. Permaculture does not cure the earth. It aids the earth in curing itself.





Alternate treatment options?:

Is Permaculture the only treatment for the earth's "dis-ease" ? Is there no other alternate treatment options? Why not use the already existing treatment plans? If bad air quality is the symptom, then use face masks, air purifiers, If Industrial Wastage seepage into the water bodies the issue, then treating the industrial effluents should be enough, right ? Why Permaculture? Why not ordinary environmental management? Why isn't sustainability enough?

These problems have already been viewed and an evolution of the "treatment plan" has taken place. From Stable approach to sustainable approach to regenerative system approach.

A **stable solution** creates medicine for human disease, pesticide for plant disease, a purifier for dirty water - treating each "planetary component" separately, the way traditional control theory treats a system: dampen the disturbance, return to set point, and stop there. It works, but it only ever sees the one signal it was built to correct, never the living system generating that signal. It gives a very narrow perspective into the problem.

A **sustainable solution** widens the lens slightly, it still fixes the same symptom, but now also tries not to create a new one while doing so. This is real progress - closer to the “reduce impact, conserve resources, sustain over time” goal of the Sustainability stage in the control-systems progression — but it is still reactive: minimizing harm at the edges of a problem it hasn't addressed at the root. It give a broader yet a superficial view into the problem.

A regenerative solution asks a different question entirely: not “how do I clean this air” but “how do I restore the Earth's own, already-existing capacity to keep its air clean” planting the trees and restoring the wetlands that let the planet do, for itself, what the purifier was standing in for. This is precisely why the PHVA framework needs two eyes rather than one field's lens: One Health supplies the diagnostic evidence of where the “dis-ease” shows up across species and systems, and Permaculture supplies the regenerative design logic for treating the root cause rather than the symptom — together forming the PHVA “binocular” evaluation that no single discipline could produce alone. It give a broader, deeper view into the problem and aims to find a long lasting solution.

	STABLE SOLUTION	SUSTAINABLE SOLUTION	REGENERATIVE SOLUTION
CORE LOGIC	Treat the symptom of each symptom separately, in isolation.	Treat the symptom, while also trying to minimize the harm caused by it in associated systems.	Restore the system’s own capacity to prevent/heal the problem on its own.
CONTROL-THEORY PARALLEL	Traditional control: dampen disturbance, return to setpoint.	Sustainability: reduce impact, conserve resources, do less harm.	Regenerative control: build capacity, generate surplus, net-positive impact.
SCOPE OF VIEW	Narrow — one component treated alone (air alone, water alone, plant alone).	Broader — accounts for downstream/side effects of the fix itself.	Systemic — treats the component as part of one living, interconnected whole.
RELATIONSHIP TO THE PROBLEM	Reactive; addresses the effect after it appears.	Reactive-preventive; reduces future harm at the margins.	Proactive; addresses the root cause generating the effect.
WHAT IT ASSUMES ABOUT THE SYSTEM	The system is a passive, isolated machine to be corrected from outside.	The system is a resource to be managed and conserved responsibly.	The system is a living, self-regenerating whole capable of healing itself if enabled to.
IN ONE HEALTH / PHVA TERMS	Curing one component’s “dis-ease” without asking why it got sick.	Curing the component while not poisoning a neighbouring one in the process.	Aiding the Earth’s own healing process (its planetary “immune system”) the way medicine aids the human body’s.
REPRESENTATIVE INTERVENTION	External corrective technology.	Cleaner technology with reduced impacts.	Restoration of ecological process that self-regulates the problem.
UNDERLYING LIMITATION	Never questions why the disturbance keeps recurring.	Still fundamentally corrective, not root-cause; harm is reduced, not eliminated.	Slower, harder to operationalise, and harder to measure — but addresses the actual “dis-ease”.

For instance, let’s take air pollution as an example and see its solution under the above mentioned three approaches.

Stable approach

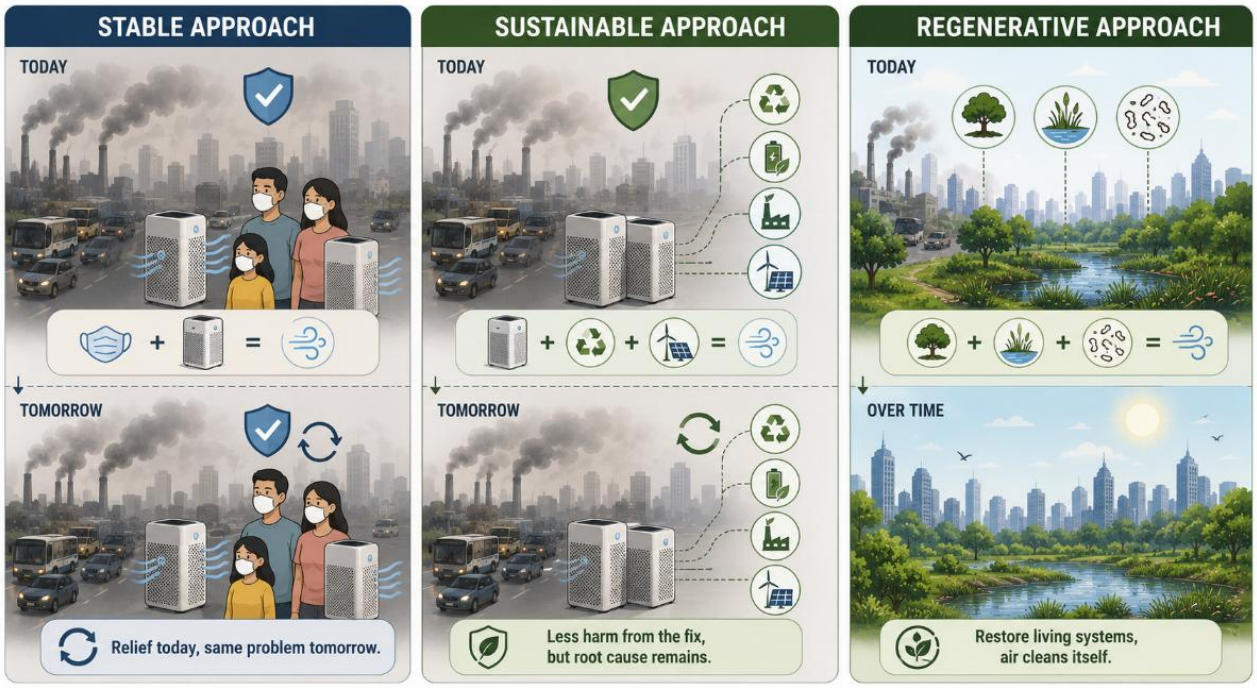
A city installs large-scale air purifiers or asks residents to wear masks. The immediate symptom — bad air quality on a given day — is corrected. But the sources (vehicle emissions, industrial combustion, biomass burning) remain untouched, so the disturbance recurs the next day, and the purifiers must run indefinitely just to hold the line.

Sustainable approach

The city still installs air purifiers, but now also ensures the purifiers' filters, batteries, and manufacturing waste are properly treated and recycled rather than dumped elsewhere, and perhaps switches the electricity powering the purifiers to a renewable source. The harm of the “fix” itself is minimized — but the fundamental cause of the polluted air is still being managed at arm's length, not resolved.

Regenerative approach

Instead of (or alongside) purifiers, the city invests in urban forestry, wetland restoration, and soil-microbiome recovery — living systems that absorb particulate matter and pollutant gases as an ordinary part of their own metabolism, the same way lungs clear themselves without external instruction. Over time, the air cleans itself, because the underlying living system's own regenerative capacity has been restored rather than substituted for.



Why Evidence, If the Treatment Is Already Known?

At this point, a reasonable objection presents itself. If Permaculture is already established as the earth's answer to its dis-ease — regenerative, closed-loop, working with the planet's own capacity to heal rather than against it — why does this report, or any report, need to marshal pollutant matrices, trend graphs, and pages of citations before saying so? When a patient is unwell, they do not demand a systematic review before taking the tablet the doctor prescribes. They go to the hospital, they are given the medicine, they take it. Why should the planet's medicine be any different — why not simply adopt Permaculture at scale and be done with the research?

The answer is that the analogy holds, but only if it is followed all the way through. The patient does not see the evidence at the point of taking the tablet — but the tablet itself would not exist, would not be trusted, and would not be prescribed by that doctor in that dose for that condition, without decades of trials, pharmacological evidence, and regulatory scrutiny sitting invisibly behind it. The absence of evidence at the bedside is not the absence of evidence — it is evidence that has already done its work, upstream, so that the moment of treatment can be simple. What looks like an evidence-free act of taking medicine is actually the end product of a long evidentiary chain; it is not the model for skipping that chain, it is the reward for having already completed it.

From Ultimate Rule to Ultimate Choice: The Need for a Rigorous Evidentiary Framework

This is the distinction this report insists on: the evidence must be collected not so that Permaculture is endlessly deferred, but precisely so that Permaculture can become the ultimate *choice* — reached for as naturally and unreflectively as a patient reaches for a prescribed medicine — rather than remaining the ultimate *rule*, imposed uniformly and asked to be taken on faith. A rule adopted without evidence is fragile: it cannot be scaled, defended against competing claims, adapted to local conditions, or trusted by the institutions — governments, funders, health systems — whose adoption is what would actually move it from principle to practice at planetary scale.

This is also why the comparison to Health Technology Assessment made later in this report is not incidental. HTA does not exist to slow down the delivery of medicine; it exists so that medicine's benefit, once demonstrated, can travel — so that a treatment validated in one hospital can be confidently prescribed in another, funded by another country's health system, and written into another region's clinical guidelines, all without each new setting having to re-derive its efficacy from scratch. One Health and Permaculture need the same evidentiary spine. Without it, Permaculture risks the fate of many indigenous and traditional ecological practices that are, in truth, sound and often quietly effective, but that never accumulated a rigorous, portable evidence base — and so remain confined to the regions, communities, and specific circumstances where they originated, unable to be scaled, funded, or mandated elsewhere, however genuinely they might help there too.

Evidence and evaluation, in this sense, function like the two railings of a train track. Either one alone goes nowhere: a railing without its twin is just a bar of metal lying in a field. It is only when evidence — the pollutant matrices, the trend analyses, the systematic reviews assembled in the chapters that follow — runs parallel to evaluation — the PHVA framework's structured judgment of what that evidence means and what should be done with it — that the track can bear weight. And it is only once that track is laid, continuously, from grassroots field trials up through national policy to international guideline, that the train of Permaculture implementation can actually run: smoothly, at scale, and somewhere.

This is, ultimately, why the report that follows does the work it does before it allows itself to simply recommend a solution already known.

Diagnostic Tests for the Planet:

Now, we know the Causative agent as well as the treatment-so, since the diagnosis is done, is the job also done ? If so, then having health physicians and specialists in this era would be pointless, since we could just diagnose and treat all our problems by ourselves with just the help of AI technologies . Because, probable Diagnosis of the disturbance is only half the job. Knowing that a patient has an infection is not the same as knowing what infection it is, which parts it affects, to how much extent it affects, what treatment to give, at what dose, for how long etc — that translation from diagnosis to action requires the help of the diagnostic tests ordered by trained health care personnel. In human health, this bridge between evidence and action is formalized through Health Technology Assessment (HTA) — a structured process that takes raw clinical and diagnostic data and converts it into a coherent, actionable plan.

The question, then, is: what performs this role for the planet? Planetary health needs, and is building, its own equivalent. This is exactly the role played by the various One Health Testing — water quality assessment, air quality assessment, climate assessment, soil and plant nutritional assessment, and others like them. Each functions as a evidence of the disease (a diagnostic test) in its

own right: water quality assessment is the planet's blood panel, climate assessment its vital signs chart, soil and plant nutritional assessment its tissue biopsy. Individually, each tells us how deeply the "disease" — the cumulative disturbance of deforestation, pollution, and extraction — has invaded one particular system.

PHVA-THE TREATMENT PROTOCOL PLANNER:

The diagnosis broken into parts is not yet a plan for the whole. Imagine a sick patient, being sent from orthopedics to cardiology to pathology — undergoing separate consultations, separate tests, separate treatment plans for what is in fact one underlying cause — and then being made to repeat this entire circuit again and again just to monitor progress. It is not just inconvenient; it is unacceptable, inaccessible, and unethical to treat this way. The fragmented care of an interconnected system is not really care at all.

The same logic holds, at planetary scale, with even higher stakes. The various dimensions of planetary health cannot be assessed in isolation, by different bodies, in different places, using disconnected methods — the errors compound, the costs multiply, the time lost is time the planet's equilibrium cannot afford. What is needed is a single, unifying framework that can take in all these separate determinant-level assessments and return one integrated protocol: a diagnostic & monitoring system of the whole, along with a tool that gives us a holistic view and idea about the management plan for restoring it. This is precisely the space that PHVA (Planetary Health Value Assessment) occupies — the planetary equivalent of a unified diagnosis-to-treatment planner, translating fragmented environmental data into one coordinated plan of action for healing the earth without inflicting further harm on it in the process

The Project: A Planetary Health Value Assessment Framework:

Thus, this is the central goal of the proposed Planetary Health Value Assessment (PHVA) Framework — an approach that extends conventional Health Technology Assessment (HTA) and existing sustainability-scoring models by adopting what we came to call a binocular lens approach- between One Health & Permaculture

The idea is simple to state but, harder to operationalise: most existing frameworks look at health impact or environmental impact, but rarely both, and almost never through a systems lens that spans humans, animals, plants, and the physical environment together-while also simultaneously looking it from a self-sustainable(Permaculture) aspect. The PHVA framework proposes viewing every activity, product, or exposure through these two convergent "eyes":

- **One Health — the interconnectedness of human, animal, and ecosystem health,.**
- **Permaculture — a Regenerative design philosophy rooted in working with self-sustainable natural systems, long-term ecological sustainability & a closed-loop resource use, wherein every output is an input for another part of the system.**

These two perspectives converge through the binocular view into the One health testing (The Evidence) and the PHVA (The Evaluation). Ultimately the instrumentation systems and AI modelling and those that orginate as a convergent resultant of these act as the practical machinery that lets us actually measure, validate, and simulate the interdependencies across ecosystems.

A fuller specification of this framework has since been developed as a standalone research and policy proposal, positioning PHVA explicitly alongside the frameworks it draws from and extends — One Health, Planetary Health, the planetary-boundaries science, Doughnut Economics, Health Technology Assessment and the Quality-Adjusted Life Year etc,.so that PHVA is understood as an interoperability layer rather than a rival to any of them. The proposal organizes evidence into eight domains (environmental/ecological effectiveness, resource/material efficiency, ecological/health safety, equity and distributional justice, resilience and adaptive capacity, knowledge and research contribution, cultural/social-cohesion value, and economic value) each mapped to a validated measurement instrument and operationalized through a four-tier process.

This process keeps a disaggregated dashboard mandatory while treating any single composite metric ("Planetary Health Years") as strictly optional, subject to the same disability- and equity-focused scrutiny that exposed the QALY's documented discriminatory failures, before any higher-stakes use is considered. This deliberate refusal to collapse the evidence prematurely into one number is what allows the binocular view of One Health and Permaculture to be evaluated at the same resolution as the diagnostic evidence it is built on, rather

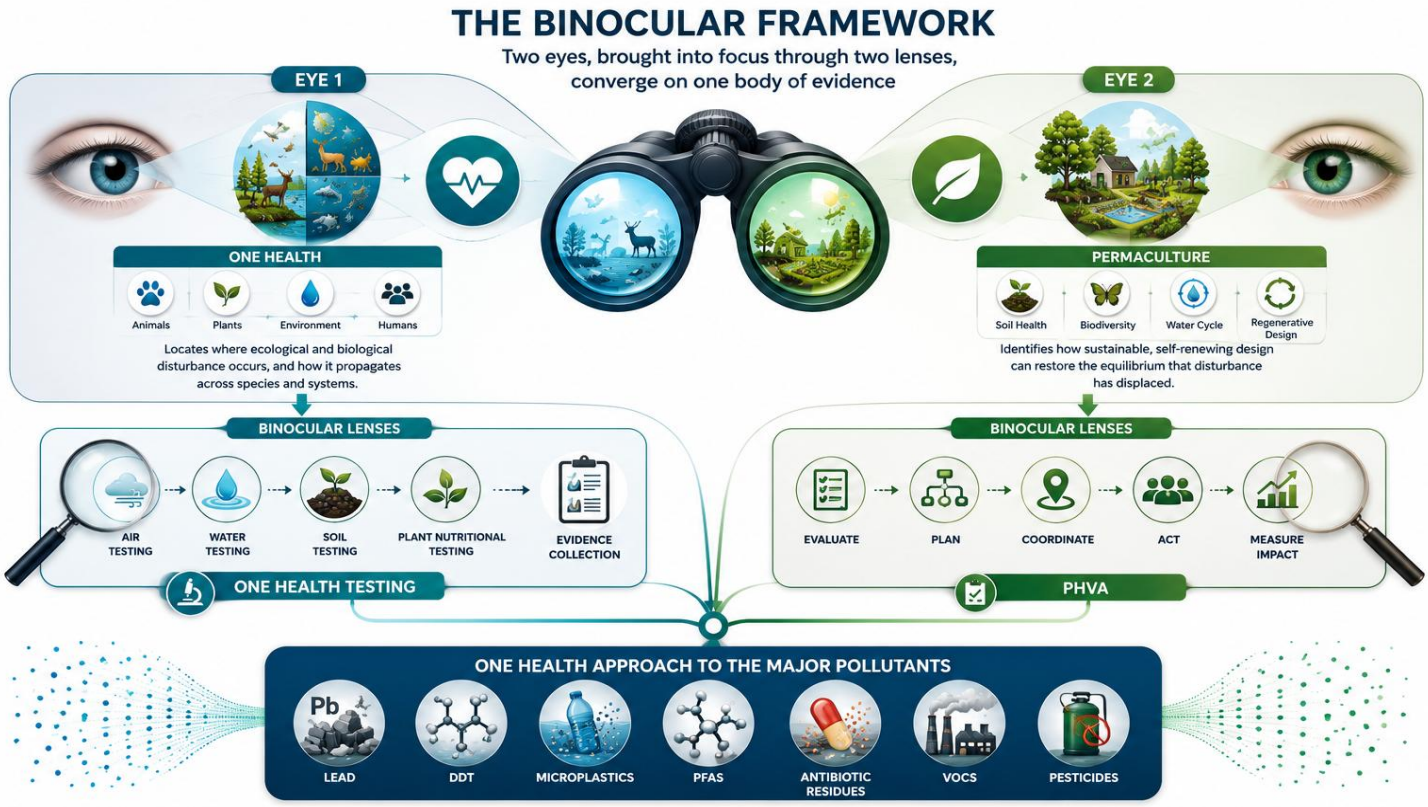
than being flattened into a single score that could hide exactly the trade-offs — between, say, environmental effectiveness and equity — that a genuine planetary diagnosis needs to surface. For more details, please refer the full report of my fellow intern Dr. Rohit on the Introduction to PHVA Framework.


RESEARCH AND POLICY PROPOSAL

Planetary Health Value Assessment (PHVA)

A Multi-Criteria Framework for Evaluating the Planetary Health Impact and Value of Human Actions, Products, and Services

Reference: Planetary_Health_Value_Assessment_PHVA_Proposal (1).docx



One health approach to the major environmental pollutants:

Having established the perspective of view as to how to view each substance/activity that acts as a “Dis-ease” causing agent of the planet , the report now applies it to individual pollutant classes:

ONE HEALTH APPROACH TO LEAD POLLUTION

A Systematic Review of Sources, Exposure Pathways, and Impacts on Human, Animal, Plant, Aquatic, and Ecosystem Health

TABLE OF CONTENTS

Abstract

Lead (Pb) is a naturally occurring heavy metal and a non-biodegradable environmental pollutant with no known biological function. Human-driven mobilization of this metal over the past three thousand years has made it one of the most widespread toxic pollutants on Earth.

Past achievements in decreasing the use of leaded gasoline, lead-based paint, and adulterated turmeric demonstrate that reducing lead exposure is indeed possible. Nevertheless, contemporary challenges such as electronic waste (e-waste) and ammunition, combined with persistent legacy contamination, remain significant unmet concerns.

Within a One Health framework, this review offers a thorough synthesis of current evidence regarding lead's sources, environmental transport, exposure pathways, and its multi-systemic impacts on human health, wildlife, birds, plants, and aquatic and ecosystem vitality.

Lead exposure in humans leads to a wide range of effects—neurodevelopmental deficits, renal dysfunction, reproductive toxicity, immune dysregulation, hematological toxicity, etc. It also disrupts plant growth, photosynthesis, and nutrient uptake, and thus threatens wildlife, aqualife, and birds indirectly via bioaccumulation and biomagnification, as well as by direct exposure—through its impact at the cellular level, the impact of oxidative stress, epigenetic modifications and the divalent cation analogy via which it causes all these effects. The review also highlights the emerging interaction between climate change and lead pollution, whereby climate-driven wildfires and other environmental disturbances remobilize legacy lead deposits, thus increasing lead exposure risks.

Ultimately, this review examines contemporary strategies, regulatory frameworks, and interventions, while underscoring persistent gaps, particularly in low- and middle-income countries. Consequently, lead toxicity should not be regarded merely as a toxicological challenge, but rather as a complex environmental, ecological, and public health crisis that demands a synchronized, multidisciplinary strategy to preserve the integrity of the global ecosystem.

1. INTRODUCTION:

Lead is a soft, bluish-grey heavy metal (chemical symbol Pb, from the Latin plumbum) that occurs naturally in the Earth's crust but has been extracted, refined, and dispersed by human activity since antiquity. Historical records indicate that lead was widely used in ancient societies for pipes, cookware, cosmetics, and wine storage, practices that contributed to some of the earliest documented cases of environmental contamination and human poisoning. [5] Unlike organic pollutants, lead is not biodegradable: once released, it persists indefinitely in soils, sediments, water, and biological tissues, redistributing between environmental compartments rather than breaking down. The WHO identifies lead as one of ten chemicals of major public health concern and states that there is no level of lead exposure known to be without harmful effects, and that the health impacts of lead exposure are entirely preventable. [1] Lead is a highly poisonous metal that can affect almost every organ system in the body thus being the lead among heavy metals that cause or contribute to death.

The Institute for Health Metrics and Evaluation estimates that more than 3.5 million deaths globally are attributable to lead exposure, a substantial rise from an earlier WHO estimate of roughly 1 million deaths per year. [1,31] A 2023 World Bank modelling study published in The Lancet Planetary Health independently estimated 5.5 million adult cardiovascular deaths attributable to lead exposure in 2019 alone — about six times higher than earlier Global Burden of Disease estimates — alongside 765 million IQ points lost in children under five worldwide and a global economic cost of roughly US$6 trillion. [3] What distinguishes lead from many other pollutants is the breadth of its footprint: it is simultaneously an occupational hazard, a domestic and dietary contaminant, an agricultural and soil problem, a wildlife toxicant of global conservation concern, an aquatic contaminant, and a component of the long-range atmospheric and cryospheric record of human industrial activity stretching back over two millennia. [4] This review attempts a 360-degree synthesis of that Lead footprint, moving systematically from sources and exposure pathways through effects on humans, animals (including birds), plants, aquatic systems, soil biota and biodiversity, and finally its more indirect relationship with climate change, before summarising remediation and policy responses documented in the literature.

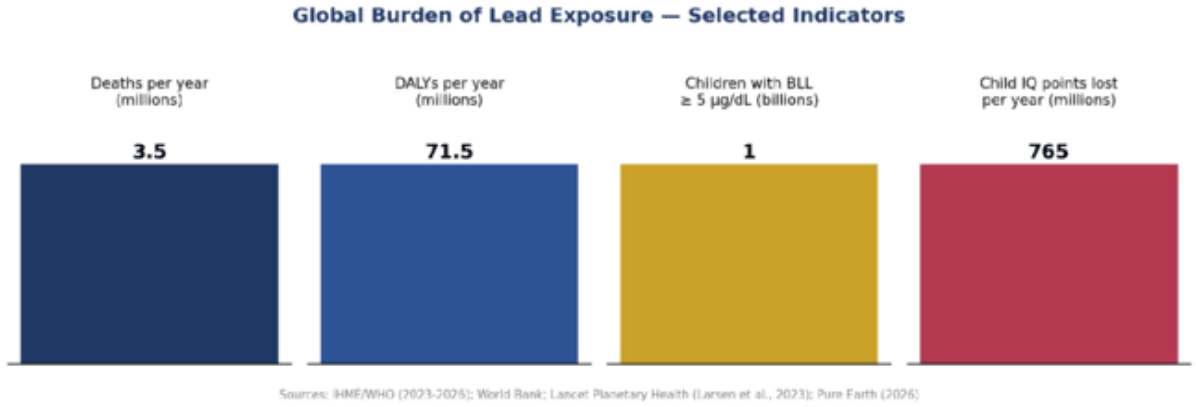


Figure 1. Global burden of lead exposure — selected indicators [1,2,3].

2. METHADOLOGY

This review synthesises findings from freely accessible, peer-reviewed articles and systematic reviews, drawn from PubMed/MEDLINE, Scopus-indexed journals

(ScienceDirect, Springer Nature, Frontiers, MDPI, PLOS), and Google Scholar, supplemented by authoritative institutional sources (WHO, UNEP, US EPA, Pure Earth). Search terms combined "lead pollution," "lead toxicity," "Pb exposure," and domain-specific qualifiers (e.g., "plants," "fish," "birds," "soil microbiome," "climate," "children," "cardiovascular"). Priority was given to systematic reviews, meta-analyses, and recent primary research with open-access full text.

3. SOURCES OF LEAD POLLUTION

Lead pollution arises from a mixture of legacy contamination (historical uses whose residues persist in soil, dust, and infrastructure) and ongoing emissions from current industrial, occupational, and consumer activities. The relative importance of each source varies markedly between high-income countries (HICs), where legacy paint, plumbing, and industrial soil contamination dominate, and low- and middle-income countries (LMICs), where informal recycling, adulterated consumer products, and weak regulatory enforcement play a larger role.

3.1 Lead in acid battery production and recycling

Lead-acid battery manufacture and recycling are by far the largest single use of lead worldwide. UNEP and WHO sources estimate that approximately 85–86% of global lead consumption goes into the production of lead-acid batteries, chiefly for motor vehicles, energy storage, and back-up power supplies. [6,7] Much of this demand is met by recycled lead, and lead recycling is itself an important cause of environmental contamination and human exposure. [7] When recycling is conducted informally — batteries broken open by hand in homes and backyards, a practice common in many LMICs — it becomes one of the most concentrated sources of human and environmental lead exposure; recent investigative reporting has documented substandard used-lead-acid-battery (ULAB) recycling operations poisoning entire worker and family populations near recycling sites. [9]

3.2 Mining, smelting, and industrial manufacturing

Primary and secondary mining, ore smelting, and metal manufacturing/recycling operations remain major point sources of soil, air, and water contamination, particularly around active or legacy industrial sites. Global consumption of lead through battery production alone increased from roughly 0.6 million tonnes in 1960 to over 10 million tonnes by 2012, a trend driven by rising numbers of motor vehicles and growing demand for standby and renewable-energy storage batteries. [8]

3.3 Lead-based paint

Residential lead-based paint, especially in buildings more than 20 years old, remains a major exposure source; one recent mechanistic review attributes approximately 20.6% of documented exposure cases to such residential/paint-related factors. [5] Many countries still permit the manufacture and sale of paint containing high lead levels, predominantly LMICs, prompting the UNEP/WHO-led Global Alliance to Eliminate Lead Paint, which works with governments and industry to draft and enforce lead-paint laws and has supported paint reformulation pilots that cut lead content in tested products by orders of magnitude. [15]

3.4 Adulterated spices, cosmetics, and traditional products

An emerging and increasingly well-documented source is deliberate adulteration of consumer products. Turmeric adulterated with lead chromate (used to intensify colour) has been documented at scale across South Asia, with studies in Bangladesh, India, Pakistan, Sri

Lanka, and Nepal — countries producing over 80% of the world's turmeric and home to 1.7 billion people — finding widespread contamination and modelling elevated child blood lead levels in affected regions. [10] A targeted, multi-year intervention in Bangladesh combining media campaigns, public education, and enforcement reduced the share of turmeric mills adding lead chromate from 30% to 0% and cut market contamination from 47% to undetectable levels, demonstrating that this source is highly tractable to policy action. [11] Traditional kohl/eye cosmetics have separately been identified as the second most common attributable source of paediatric lead poisoning in one clinical case review, accounting for roughly 23.5% of cases. [5]

3.5 Electronic waste (e-waste)

Lead is the most prevalent heavy-metal contaminant in electronic waste, and informal e-waste recycling — dismantling, open burning, and acid leaching of circuit boards, solder, and cathode-ray-tube glass — is a rapidly growing exposure source, particularly for children living near recycling sites in LMICs such as Guiyu, China. [12]

3.6 Ammunition, fishing weights, and lead shot

Lead ammunition (shot and bullets) and fishing sinkers introduce metallic lead directly into terrestrial and aquatic environments and into the food chain via game meat. Shooting is estimated to release tens of thousands of tonnes of lead ammunition into the European environment annually, and this constitutes a well-documented and largely distinct exposure pathway for wildlife, discussed further in Section 6. [13,14]

3.7 Legacy leaded gasoline, plumbing, and other residual sources

Although leaded automotive gasoline has been phased out in almost all countries, decades of historical use left substantial lead residues in urban soils and roadside dust that continue to contribute to background exposure; isotopic studies have confirmed that fire- and flood-remobilised lead in some regions is chemically consistent with historical leaded-gasoline emissions rather than natural background sources. [29,30] Other residual and minor sources identified in the literature include lead pipes and solder in ageing plumbing, lead crystal glassware, ceramic glazes, and legacy industrial deposits in urban infrastructure.

3.8 Summary of relative contributions

Because exposure pathways differ so much by setting, the literature does not converge on one universal percentage breakdown. Table 1 and Figures 2–3 summarise the recurring figures identified across the sources reviewed here; these should be read as illustrative, order-of-magnitude findings from specific studies rather than fixed global constants.

Figure 2. Global end-use of refined lead metal [6,7]

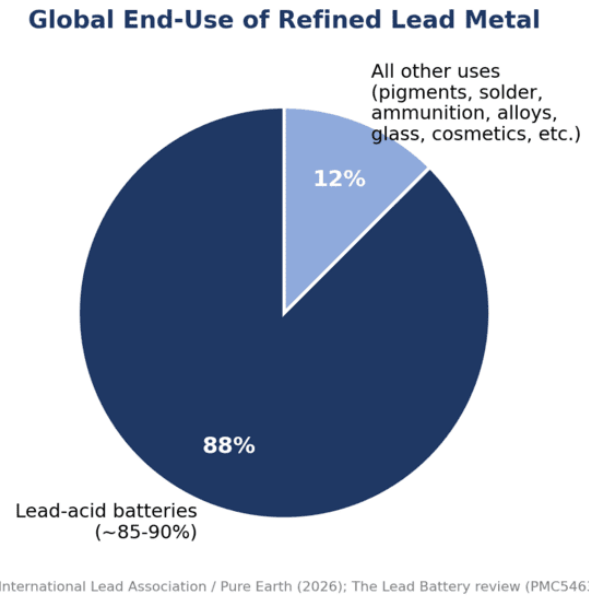


Table 1. Major documented sources of lead pollution and their reported relative contributions.

Source category	Reported contribution / significance	Ref.
Lead-acid battery production & recycling	~85–86% of global refined lead consumption; informal recycling linked to a large share of exposure-related disease burden	[6,7,9]
Mining, smelting & industrial manufacturing	Major point-source soil/air/water contamination; global lead mine production has risen sharply since the 1990s	[8]
Lead-based paint	~20.6% of documented exposure cases (residential, buildings >20 yrs); still legally sold in many countries	[5,15]
Adulterated cosmetics & spices (kohl, turmeric)	Kohl: ~23.5% of paediatric poisoning cases in one review; turmeric lead-chromate adulteration widely documented in South Asia	[5,10,11]
Electronic waste (e-waste)	Most prevalent heavy-metal contaminant in e-waste; informal recycling is a growing paediatric exposure source	[12]
Ammunition, fishing weights & lead shot	Direct introduction into terrestrial/aquatic food chains and wildlife	[13,14]
Legacy leaded gasoline & plumbing	Residual urban soil/dust contamination from historical use; wildfire and flood remobilisation ongoing concern	[29,30]

Figure 3. Illustrative source attribution from a clinical case review of paediatric lead poisoning [5].

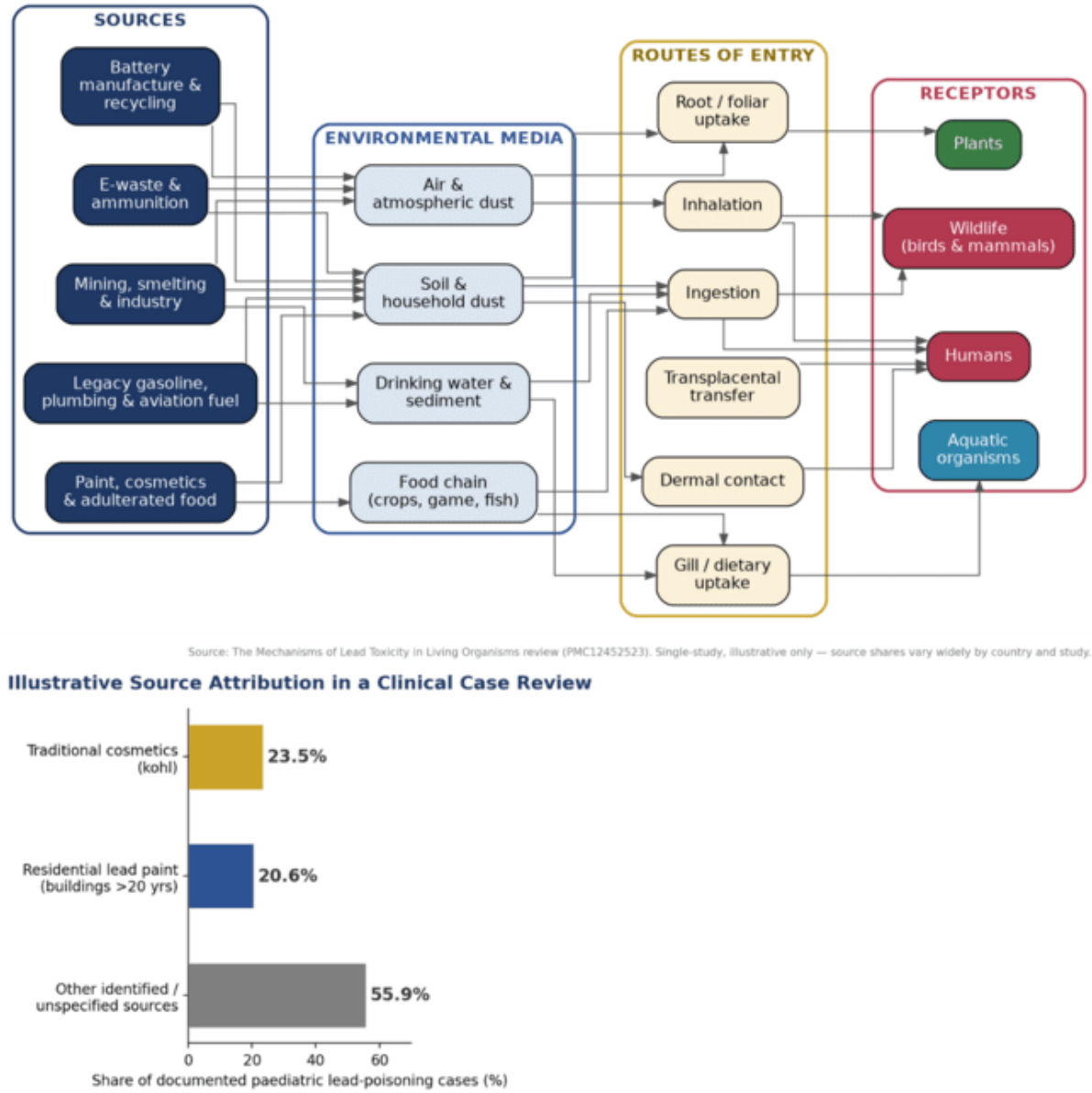


Figure 4. Lead exposure pathway: sources → environmental media → routes of entry → receptors.

4. MODES OF ENTRY INTO THE ENVIRONMENT, BIODIVERSITY, AND HUMAN BODY

Once released, lead moves between air, soil, water, dust, and food through physical and biological processes, and enters organisms via several distinct routes, summarised in Figure 4 and Table 2.

4.1 Environmental transport

Airborne lead particulates from smelting, battery manufacturing, and recycling settle onto surrounding soil and household dust, where they persist for decades because lead binds strongly to soil organic matter and mineral surfaces. Lead can also contaminate drinking

water directly through ageing lead pipes, solder, and fittings. [1,17] Long-range atmospheric transport disperses industrial and combustion-derived lead across oceans and thousands of kilometres, as demonstrated by lead's presence in polar ice cores far from any point source — measurements at the South Pole detected substantial industrial lead pollution as early as 1889, more than two decades before polar explorers first reached the pole. [4]

4.2 Routes of entry into the human body

Human exposure occurs mainly through inhalation of lead particles and fumes from burning or processing lead-containing materials, and ingestion of lead-contaminated dust, water, or food, with additional exposure via dermal contact and transplacental transfer, since lead stored in maternal bone is mobilised into blood during pregnancy and lactation and crosses the placenta. [1,17]

4.3 Routes of entry into wildlife and biodiversity

Terrestrial wildlife and birds are exposed chiefly through direct ingestion of spent lead shot or bullet fragments mistaken for grit or found in shot carcasses, and secondarily through consumption of contaminated vegetation, invertebrates, or prey in contaminated habitats. [13]

Aquatic organisms take up lead from water and from contaminated sediment and diet, with uptake mechanisms differing between freshwater and marine/seawater habitats. [23]

Plants absorb lead passively through root systems from soil pore water and, to a lesser extent, through foliar deposition of airborne particulates onto leaf surfaces via the cuticle and stomata. [18]

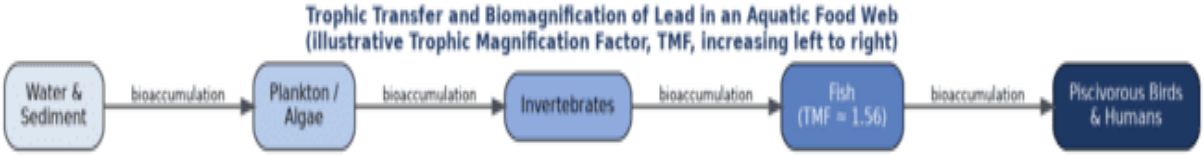
4.4 Trophic transfer and biomagnification

Because lead is persistent and bioaccumulates in tissues, it can move up food chains from sediment and plankton to invertebrates, fish, birds of prey, and ultimately humans who consume contaminated fish, game, or crops. Fish sit at or near the top of most aquatic food chains and are consequently among the most susceptible aquatic organisms to lead's toxic effects, accumulating it via both waterborne and dietary routes. [21,23]

Table 2. Principal routes of lead entry by receptor type.

Receptor	Principal routes of entry	Ref.
Humans	Ingestion (food, water, soil, dust, adulterated products); inhalation; dermal contact; transplacental transfer	[1,17]
Terrestrial wildlife / birds	Direct ingestion of used shot/bullet fragments; consumption of shot carcasses; contaminated vegetation	[13,14]
Aquatic organisms	Waterborne uptake across gill membranes; dietary uptake from contaminated sediment/prey	[23]
Plants	Passive root uptake from soil pore water; foliar deposition via cuticle/stomata	[18]
Soil microorganisms	Direct contact within soil matrix; bioavailability governed by pH, organic matter, and speciation	[25]

Figure 5. Trophic transfer and biomagnification of lead through an illustrative aquatic food web [21,23,24].



5. EFFECTS ON HUMAN HEALTH

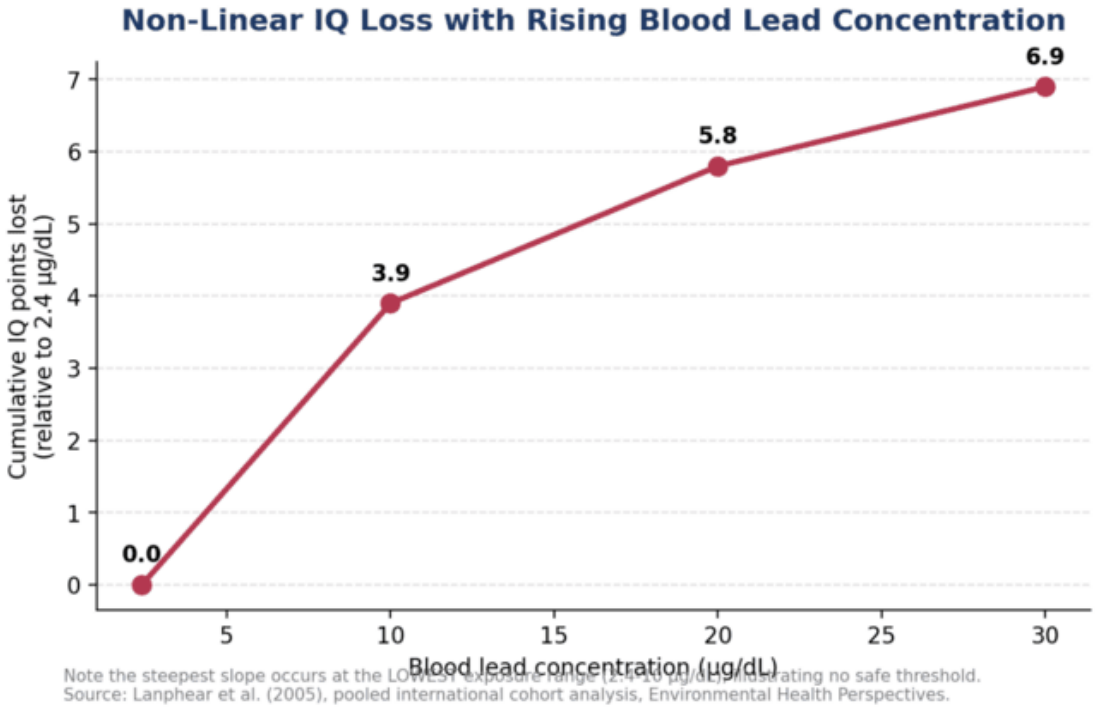
Human toxicology is the most thoroughly studied dimension of lead pollution, and the evidence base has driven a progressive downward revision of what is considered a "safe" blood lead level (BLL), now understood to have no lower threshold. [1]

5.1 Neurodevelopmental and cognitive effects

The nervous system is one of the primary targets of lead toxicity, with the brain being the most sensitive organ to lead exposure (Cleveland et al., 2008). Lead is a confirmed neurotoxin, and its effects on the developing brain are considered irreversible. [27] It markedly disrupts synapse formation within the cerebral cortex and also interferes with the normal development of neurochemicals, including neurotransmitters, and alters the organization and function of ion channels. Furthermore, lead poisoning causes demyelination, reduces the number of functional neurons, disrupts neurotransmission, and impairs neuronal growth (Pearson & Schonfeld, 2003). These structural and functional abnormalities contribute to persistent neurological dysfunction.

A landmark international pooled analysis of 1,333 children from seven longitudinal cohort studies found an inverse, non-linear relationship between blood lead concentration and IQ score: children whose maximal blood lead level stayed below 7.5 µg/dL still showed measurable intellectual deficits, and the steepest IQ loss occurred at the very lowest exposure levels rather than the highest, with no evidence of a safe threshold. [26] Cumulative global impact is substantial: the 2023 World Bank/Lancet Planetary Health modelling study found that children under five years old worldwide lost 765 million IQ points in 2019 alone, 729

million of them in LMICs — an estimate roughly 80% higher than earlier calculations. [3]



5.2 Cardiovascular disease

In adults, cardiovascular disease is now recognised as the dominant cause of lead-attributable mortality. The 2023 World Bank modelling study estimated 5.5 million adult cardiovascular deaths attributable to lead exposure in 2019 — about 6 times higher than the previous Global Burden of Disease estimate — putting lead's combined cardiovascular and cognitive burden on a par with fine-particulate ($PM_{2.5}$) air pollution. [3]

5.3 Renal, haematological, reproductive, and immune effects

Lead is a cumulative toxicant affecting multiple body systems; documented health effects in children include anaemia, hypertension, renal impairment, immunotoxicity, toxicity to the reproductive organs, and reduced intelligence, while in adults lead causes long-term harm including increased risk of high blood pressure, cardiovascular problems, and kidney damage, and lead exposure during pregnancy can cause reduced fetal growth and preterm birth. [1,17]

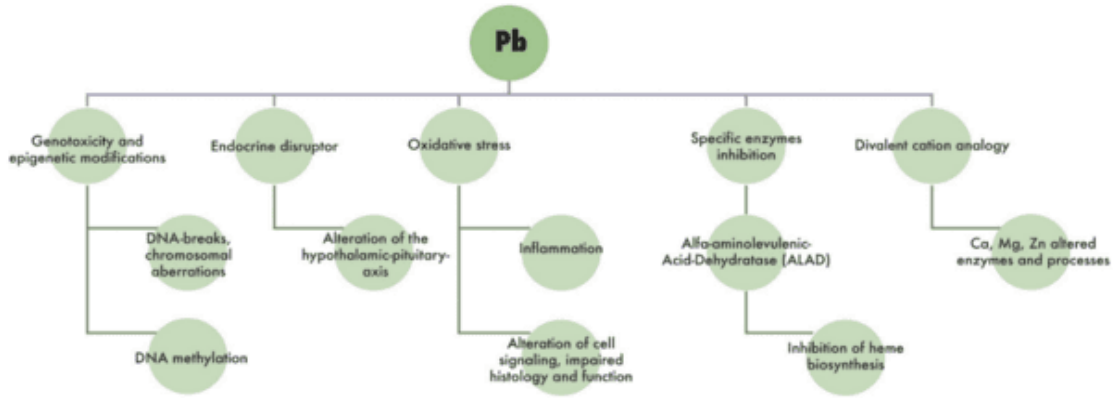
Wedeen et al. (1975) investigated the effects of occupational lead exposure and reported tubular dysfunction in patients who underwent kidney biopsy. These findings provided early evidence that lead nephropathy represents a significant occupational health hazard

In males, blood lead levels exceeding 40 µg/dL have been associated with a reduction in sperm count as well as alterations in semen volume. In addition, important sperm characteristics, including motility and overall morphology, are significantly impaired at these exposure levels (Navas-Acien et al., 2007)

Lead exposure also has profound effects on the female reproductive system, with reproductive complications often being more severe. Elevated lead levels have been associated with adverse pregnancy outcomes, including miscarriages, premature delivery, low birth weight, and impaired childhood development (Park et al., 2008). These findings highlight the heightened vulnerability of women and the developing fetus to lead toxicity.

5.4 Cellular and molecular mechanisms

At the molecular level, lead exerts toxicity chiefly through ion mimicry: because its ionic properties resemble those of calcium, roughly 90% of the body's lead burden is stored in bone tissue for decades, while circulating lead disrupts cellular processes by displacing calcium and other essential metals in enzymes and proteins, leading to mitochondrial dysfunction, oxidative stress, DNA damage, and epigenetic modifications. [5] Increased oxidative stress and modulation of cellular thiols play a prominent role in the disease manifestations seen across the haematopoietic, renal, reproductive, and central nervous systems. [27]



5.5 Global disease burden

Taken together, the global health-economic burden of lead exposure has been estimated to rival that of ambient and household air pollution combined, with a total global cost of lost IQ and cardiovascular mortality estimated at approximately US$6 trillion in 2019. [3]

Table 3. Blood lead level (BLL) reference points and associated documented effects in humans.

BLL threshold	Documented association / regulatory meaning	Ref.
≥ 3.5 µg/dL	Associated with decreased intelligence, behavioural difficulties, and learning problems in children; no lower threshold identified	[17]
≥ 5 µg/dL	WHO action / clinical-management threshold; roughly a third of the world's children estimated to exceed this level	[1,2]
2.4–10 µg/dL	Steepest IQ-loss slope of any exposure range in the Lanphear pooled analysis	[26]
10–30 µg/dL	Continued, though flatter, IQ decrement per unit increase in blood lead	[26]
Any detectable level	No blood lead concentration has been identified as free of harmful effects	[1]

6. EFFECTS ON WILDLIFE:

Wildlife toxicology provides some of the clearest population-level evidence of lead's ecological cost, distinct from and complementary to human health data.

6.1 Birds:

Avian lead poisoning from ammunition sources has been recognised for over a century and remains one of the best-documented wildlife contaminant issues globally. [13] Birds are poisoned in two principal ways: direct ingestion of spent lead gunshot mistaken for grit, especially by waterfowl and other gizzard-grinding game birds, and ingestion of lead bullet or shot fragments embedded in shot carcasses or offal, which particularly affects raptors and scavengers such as eagles, vultures, hawks, falcons, and owls. [13]

A comprehensive review and update found that both the number of species affected and the geographical spread of documented poisoning cases have increased over time, and that sub-lethal poisoning has been detected at blood lead concentrations lower than previously reported, suggesting earlier effect-level thresholds should be revised downward or abandoned. [13] Lead poisoning is estimated to kill approximately one million wildfowl per year in Europe alone and cause sub-lethal poisoning in at least three million more. [13] An economic and welfare analysis further found that shooting continues to release tens of thousands of tonnes of lead ammunition into the European environment annually, contaminating soil and water and putting at risk both wildlife and the people who consume game meat containing embedded lead fragments. [14]

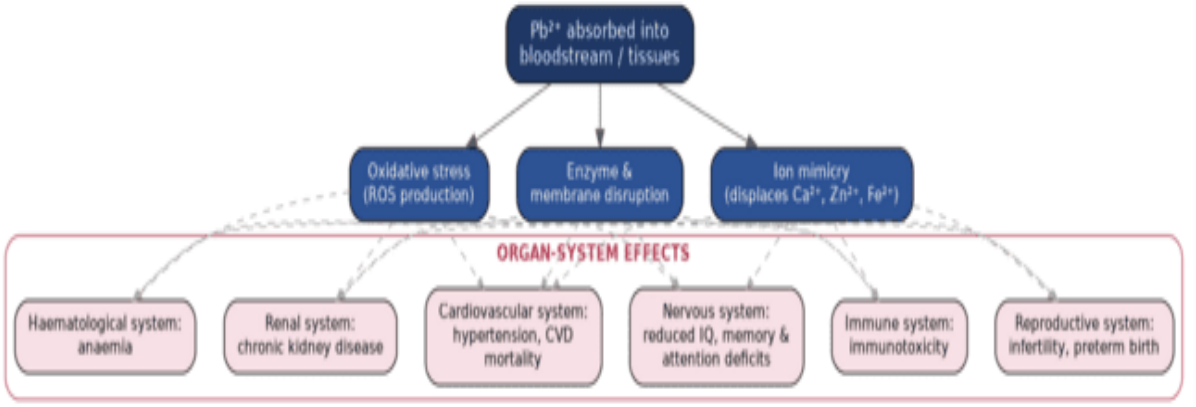


Figure 7. Mechanism of lead toxicity: from ion mimicry and oxidative stress to organ-system effects, applicable across vertebrate taxa including humans, birds, and mammals [5,27].

6.2 Terrestrial mammals and other wildlife

Fragments of lead derived from ammunition used to kill game birds and mammals are often present in the edible tissues of harvested animals, representing a potentially significant dietary exposure pathway for both scavenging wildlife and people who frequently consume game meat. [14] Across taxa, documented sub-lethal and lethal effects of lead include anaemia, lethargy, muscle wastage, loss of coordination, and neurological signs such as leg paralysis or convulsions, consistent with the toxicity mechanisms described for humans in Section 5.4. [13]

6.3 Policy responses for wildlife

In response to this evidence, some jurisdictions banned lead gunshot for waterfowl hunting decades ago, and international bodies have subsequently called for broader phase-outs of lead

ammunition across terrestrial and wetland habitats; nonetheless, current regulations in most of the world remain limited to specific hunting contexts and do not adequately control the wider risks to non-target wildlife and human consumers documented in the literature. [14]

7. EFFECTS ON PLANT HEALTH AND NUTRITION

Lead has no known biological or nutritional role in plants, yet it is readily taken up and can severely disrupt plant physiology, ultimately threatening food security in contaminated agricultural soils.

7.1 Uptake and translocation

Plants absorb lead predominantly through root systems, a process controlled by cation exchange capacity, soil pH, particle size, root nature, and other physio-chemical factors. [18] Because lead binds strongly to soil constituents, only a limited fraction is typically bioavailable for root uptake; once absorbed, lead is transported via xylem vessels but is largely immobilised at the root endodermis by negatively charged cell-wall components, which restricts translocation to above-ground edible tissue in most (though not all) plant species. [18]

7.2 Germination, growth, and yield

Lead toxicity obstructs seed germination, reduces root and shoot length, retards overall plant growth, and reduces final crop yield. [18] Lead shares about 10% of total pollution produced by heavy metals, and excess lead accumulation has been reported to reduce root growth by up to 42% in some studies. [19]

7.3 Nutrient uptake and water relations

Lead disrupts nutrient uptake through roots, alters plasma membrane permeability, and disturbs chloroplast ultrastructure, triggering changes in both respiration and transpiration that impede the movement of water and nutrients from soil to shoot. [18]

7.4 Photosynthesis and biochemistry

Lead impairs photosynthesis, disrupts water balance and mineral nutrient status, changes hormonal status, and alters membrane structure and permeability, with oxidative stress identified as a central mechanism linking lead exposure to these physiological disruptions across a wide range of plant species. [18,20]

7.5 Consequences for food safety and nutrition

Because a portion of absorbed lead can be translocated from roots to edible above-ground tissues in some species, contaminated agricultural soils create a direct, if partially restricted, pathway for lead to enter the human food chain, compounding the dietary exposure already caused by product adulteration described in Section 3.4. [18]

Table 4. Summary of documented phytotoxic effects of lead.

Physiological domain	Documented effect	Ref.
Germination & growth	Obstructs seed germination; reduces root/shoot length (up to ~42% root growth reduction in some studies); retards overall growth and final crop yield	[18,19]

Physiological domain	Documented effect	Ref.
Nutrient uptake	Disrupts nutrient uptake through roots and alters plasma membrane permeability	[18]
Water relations	Disturbs respiration and transpiration; disrupts water balance and mineral nutrient transport	[18]
Photosynthesis	Impairs photosynthesis and disrupts chloroplast ultrastructure	[18,20]
Oxidative & hormonal stress	Triggers oxidative stress; alters hormonal status and membrane structure/permeability	[18,20]
Food safety & nutrition	Root uptake and limited translocation to edible tissue create a pathway for lead to enter the human food chain	[18]

8. EFFECTS ON AQUATIC HEALTH

Lead is a persistent contaminant of freshwater and marine ecosystems, entering aquatic systems via industrial effluent, atmospheric deposition, urban runoff, mining discharge, and legacy sediment contamination.

8.1 Bioaccumulation and trophic position

Fish are at the top of the food chain in most aquatic environments and are the most susceptible to the toxic effects of lead exposure; toxicity is induced primarily by bioaccumulation in specific tissues, with accumulation mechanisms varying depending on habitat (freshwater or seawater) and pathway (waterborne or dietary exposure). [21,23] Because fish are also a major route of human dietary exposure, they serve a dual role as both bioindicators of aquatic contamination and a vector connecting aquatic pollution to human health outcomes. [21]

8.2 Physiological and cellular effects

Bioaccumulated lead causes oxidative stress in fish tissues due to excessive reactive oxygen species (ROS) production; this oxidative stress in turn induces synaptic damage and neurotransmitter malfunction, producing measurable neurotoxicity. [21] As a divalent cation, lead can also substitute for calcium at multiple levels, affecting various cell-signalling pathways, and documented effects extend to immune-system alterations and histopathological lesions across fish organs. [22,23]

8.3 Broader heavy-metal context in fish

Reviews synthesising evidence across multiple heavy metals confirm that metal bioaccumulation in fish organs causes structural lesions, functional disturbances, oxidative stress, histopathological manifestations, and altered transcriptional gene regulation, with cellular dysfunction capable of disturbing broader ecological equilibrium. [22,24]

8.4 Ecosystem-level implications

Heavy-metal contamination including lead threatens the sustainable development of aquaculture through impaired growth, reproduction, and physiology of farmed fish, and

reviews describe cascading impacts on aquatic ecosystem structure and function across diverse habitats, underscoring the need for improved water management and pollution-control strategies to protect both aquatic ecosystems and the humans who depend on them for food. [24]

9. EFFECTS ON SOIL MICROBIOME

9.1 Soil microbial communities

Lead contamination measurably suppresses soil microbial activity. A controlled field study using Biolog EcoPlate technology found that lead pollution above 300 mg/kg significantly reduced soil microbial biomass carbon and nitrogen and reduced catalase activity, while increasing invertase activity and suppressing the general ability of soil microorganisms to metabolise carbon sources — changes with direct implications for nutrient cycling and, in turn, plant productivity (Section 7). [25]

9.2 Ecosystem-level and biodiversity impacts

Lead poisoning has documented implications across multiple levels of ecological organisation, from suppressed soil microbial function up through disrupted food chains to species-level conservation concern. [25] The wildlife evidence summarised in Section 6 demonstrates that lead poisoning is a recognised threat to numerous bird species, including species of conservation concern, meaning lead pollution is not merely a diffuse background stressor but can act as a direct driver of population-level harm in vulnerable, long-lived taxa with slow reproductive rates. [13]

10. LEAD POLLUTION AND CLIMATE CHANGE: A TWO-WAY RELATIONSHIP

Lead is not a greenhouse gas and is not considered a direct driver of climate change in the way carbon dioxide or methane are; however, the literature documents a genuine bidirectional relationship between lead pollution and the climate system, in two distinct senses, summarised in Figure 8.

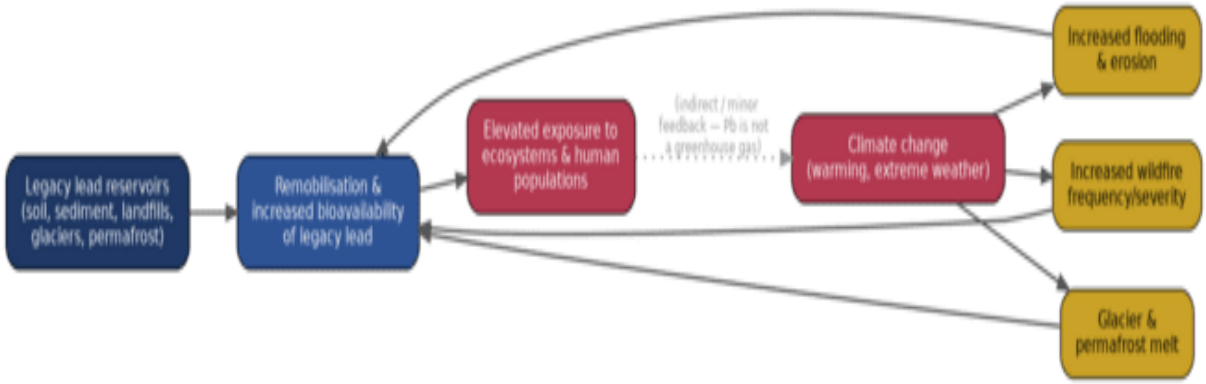


Figure 8. The climate–lead remobilisation relationship: legacy lead reservoirs, climate-driven remobilisation pathways, and resulting exposure [4,29,30].

10.1 Lead as a paleo-archive of industrial and climatic history

High-resolution ice cores from Antarctica preserve an extraordinarily detailed record of atmospheric lead deposition: an array of 16 ice cores found substantial toxic lead pollution had reached the South Pole and spread across Antarctica by 1889 — more than 22 years before Amundsen and Scott's polar expeditions — with an estimated 660 tonnes of industrial

lead deposited over Antarctica during the subsequent 130 years, traced by isotopic fingerprinting to mining and smelting at Broken Hill, Australia. Unlike the Arctic, where lead pollution peaked in the 1970s, Antarctic lead pollution was as high in the early twentieth century as at any time since industrialisation, and it persists into the present. [4]

10.2 Climate change as a driver of lead remobilisation

A growing body of literature documents how wildfires- whose frequency and severity are increasing under climate change -remobilise legacy lead that had been deposited in soils and vegetation decades earlier. A study of the 2012 Williams Fire in the Angeles National Forest, California, found elevated, isotopically distinct lead concentrations in fire ash, with isotope ratios falling between those of natural background lead and historical leaded-gasoline emissions, confirming that the fire had remobilised anthropogenic legacy lead; the authors explicitly warned that such pyrogenic remobilisation of toxic metals will increase with projected increases in wildfire frequency and intensity under climate change. [29]

Independent isotopic analysis of ash from multiple Australian wildfires corroborated this pattern, finding that legacy leaded-petrol depositions contributed between roughly 5% and 73% of the lead measured in different fire-affected sites, and concluding that remobilisation of legacy industrial lead by wildfire makes it a persistent and problematic contaminant in contemporary environmental systems. [30]

10.3 Implications

Taken together, this evidence indicates that while lead pollution does not meaningfully contribute to greenhouse forcing, climate change is likely to exacerbate lead exposure risk by reactivating decades- to centuries-old contamination that would otherwise have remained relatively immobile — an emerging concern for regions with substantial legacy industrial contamination and increasing exposure to wildfire. [29,30]

11. ENVIRONMENTAL EFFECTS: AIR, WATER, AND SOIL

Beyond the specific biological domains discussed above, lead pollution has broad environmental footprints worth summarising discretely.

11.1 Atmospheric contamination

Smelters, lead battery manufacturers, and recycling plants emit airborne lead particulates that settle onto soil and dust in surrounding areas; because low background atmospheric concentrations and distinct isotopic signatures make lead an effective tracer of industrial pollution, its atmospheric dispersal has been documented reaching even the most remote regions on Earth. [4]

11.2 Water contamination

Surface water and groundwater become contaminated via industrial effluent, mining discharge, urban and agricultural runoff, and, in drinking-water distribution systems specifically, corrosion of ageing lead pipes, solder, and fittings — a pathway of particular concern in older urban infrastructure in both HICs and LMICs. [1,17]

11.3 Soil contamination

Historically, the use of lead in urban infrastructure — water pipes, roofing, and household paint — resulted in significant accumulation in soil and dust, especially in densely populated or industrialised regions; in the United States, legacy contamination from such uses has led to

persistent soil lead levels exceeding standard thresholds, particularly in urban neighbourhoods with older housing stock, a pattern echoed in industrial regions internationally. [5]

12. REMEDIATION, REGULATION, AND SUSTAINABLE SOLUTIONS

The literature describes a range of policy-based and technical interventions aimed at reducing both existing contamination and future emissions, summarised in Table 5.

12.1 Regulatory and institutional responses

International coordination includes the WHO's clinical-management guidance recommending that the source of lead exposure be identified and exposure reduced or terminated for anyone with a blood lead level above 5 µg/dL. [1] The UNEP/WHO Global Alliance to Eliminate Lead Paint supports countries in drafting and enforcing lead-paint regulations, has grown the number of countries with mandatory lead-paint limits substantially since 2012, and coordinates the annual International Lead Poisoning Prevention Week alongside partners including the US EPA and the International Pollutants Elimination Network. [15] The US EPA participates in the Lead Paint Alliance, works with ASTM International on new testing standards for lead in paint, and has organised G7 and G20 expert webinars on reducing lead pollution and exposure in low- and middle-income countries. [16]

12.2 Source-specific interventions

Targeted interventions have shown that individual sources are highly tractable: a multi-faceted media, education, and enforcement campaign in Bangladesh reduced turmeric mills adding lead chromate from 30% to 0% of sites tested and cut worker blood lead levels by roughly 24–30%. [11] Formalizing and enforcing standards around used-lead-acid-battery recycling—shutting down unsafe operations and aligning market incentives with safety—has produced measurable progress in countries including Brazil, China, and parts of Southeast Asia. [9]

12.3 Persistent gaps and challenges

Despite these efforts, the literature identifies persistent enforcement gaps in LMICs, continued legality of lead ammunition for non-waterfowl hunting in most jurisdictions, and the emergence of newer exposure pathways - e-waste, adulterated spices and cosmetics , that have not yet received the same sustained policy attention as legacy sources such as paint and gasoline. [12,15]

Table 5. Selected regulatory and source-specific interventions for lead pollution.

Intervention	Description / effect	Ref.
Lead-paint regulation & reformulation	Global Alliance to Eliminate Lead Paint; supports drafting/enforcing national lead-paint laws and industry reformulation pilots	[15]
Leaded-gasoline phase-out	Historically the most successful lead-reduction intervention to date; template for the paint phase-out	[16]
Turmeric/spice adulteration enforcement	Targeted media, education, and enforcement campaign cut adulteration and blood lead levels sharply in Bangladesh	[11]

Intervention	Description / effect	Ref.
ULAB recycling formalisation	Shutting down unsafe informal recycling and enforcing environmental law reduced exposure in countries such as Brazil and China	[9]
International coordination	WHO clinical-management guidelines; G7/G20 expert webinars on lead; EPA-ASTM XRF testing standards	[1,16]

13. CONCLUSION

The literature reviewed here gives a consistent picture: lead is a uniquely persistent, non-biodegradable, and multi-domain pollutant with no biological function and no demonstrated safe exposure threshold in humans or wildlife. [1] Its sources span heavy industry (mining, smelting, battery manufacture and recycling) to everyday consumer products (paint, cosmetics, spices) to recreational activity (ammunition), and its effects radiate across virtually every level of biological organisation — from oxidative stress at the cellular level, through impaired cognition and cardiovascular disease in individual humans, to measurable population-level harm in vulnerable wildlife, avians, plants and disrupted nutrient cycling at the ecosystem scale. [3,13,25]

While lead is not a direct driver of climate change, the two problems are increasingly intertwined, as wildfires associated with a warming climate remobilise legacy lead contamination that had been considered environmentally locked away. [29,30] The uneven global distribution of exposure - concentrated overwhelmingly in low- and middle-income countries and in the most vulnerable populations, particularly young children and pregnant women — frames lead pollution as inseparably an environmental, public-health, and environmental-justice issue. [2,3]

Encouragingly, targeted interventions such as the historical elimination of leaded gasoline, the ongoing global campaign against lead paint, and successful source-specific enforcement campaigns against adulterated turmeric demonstrate that ambitious, coordinated reduction of lead exposure is achievable; sustaining and extending that momentum to emerging sources such as e-waste and ammunition remains the central challenge identified across the literature synthesised in this review. [11,15,16]

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Vancouver style. Numbers correspond to in-text citation order; each entry's URL is hyperlinked to a freely accessible copy of the source (PubMed Central, journal open-access page, or institutional report).

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ONE HEALTH APPROACH TO DDT

A One Health Review of the Persistence, Bioaccumulation, and Multi-System Toxicity of Dichlorodiphenyltrichloroethane (DDT)

TABLE OF CONTENTS

Abstract

As one of the most thoroughly documented persistent organic pollutants, dichlorodiphenyltrichloroethane (DDT) has been instrumental in mitigating vector-borne diseases, notably malaria. Nevertheless, its high lipophilicity, remarkable environmental stability, and resistance to degradation have led to pervasive ecological contamination, with

residues accumulating and biomagnifying through food webs long after initial deployment. Even though globally its agricultural applications have decreased , DDT's ongoing use in disease vector control—compounded by historical soil reserves and climate-induced liberation of legacy deposits—presents a persistent public health and environmental threat. Utilizing a One Health lens, this review synthesizes existing research on the environmental behavior of DDT, mapping its durability, atmospheric transport, and accumulation across trophic levels while exploring its impacts on public health, flora, fauna, and broader ecosystem integrity. Current findings reveal that long-term human exposure is linked to neurological damage, hormonal disruption, reproductive disorders, liver toxicity, and probable carcinogenic effects.

Furthermore, the pollutant suppresses plant nutrient absorption, growth, and photosynthesis and induces reproductive failures, developmental issues, immune compromise, and population contraction in wildlife, particularly in avian and aquatic species.

Ultimately, these findings demonstrate that the ecological and biological ramifications of DDT reach far past its primary targets. This underscores the urgency of deploying a holistic One Health framework that merges green vector management, habitat remediation, and ecological preservation to mitigate enduring contamination risks while protecting global community health.

1. Introduction

Over the past several decades, industrial, agricultural, and public health practices have released massive quantities of xenobiotics, particularly chlorinated aromatic compounds, into global ecosystems. Many of these chemical compounds are highly persistent and ecotoxic. Prominent among these is DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl) ethane] and its primary degradation products, DDE [1,1-dichloro-2,2-bis(p-chlorophenyl) ethylene] and DDD [1,1-dichloro-2,2-bis(p-chlorophenyl) ethane] (1).

Dichlorodiphenyltrichloroethane (DDT) is one of the most widely recognized and extensively used organochlorine insecticides in history. While celebrated for its critical role in controlling vector-borne diseases, it is simultaneously criticized for its severe, long-term environmental impacts. Due to its exceptional persistence, with an environmental half-life of up to 30 years, and its capacity for long-range atmospheric transport, DDT contamination has become ubiquitous, leading to the assertion that there is no longer a single living organism on Earth that does not contain trace amounts of DDT.

DDT is credited with helping approximately one billion people live free from malaria. In 1973, the World Health Organization (WHO) determined that its public health benefits far outweighed its ecological risks. Consequently, it was used globally on a massive scale, with annual applications peaking at approximately 400,000 tons by the 1960s, primarily for agricultural pest control and malaria eradication campaigns (1).

However, due to mounting ecological concerns, DDT was eventually banned in most nations following extensive debates at the Stockholm Convention. Despite its hazards, DDT remains the only highly successful strategy for controlling vector-borne diseases in several countries where malaria is endemic, particularly in regions of Africa, Asia, and Latin America. Acknowledging this, the WHO authorized its targeted reintroduction in 2006 solely for vector control in specific tropical nations.

This reintroduction led to a dramatic decrease in malaria cases across many developing countries, where DDT continues to be used for this purpose, representing a classic example of a "useful pollutant." Consequently, this review evaluates the persistence of DDT across global environments and analyzes its adverse effects on humans, animals, birds, aquatic organisms, plants, and the wider ecosystem.

2. Methodology

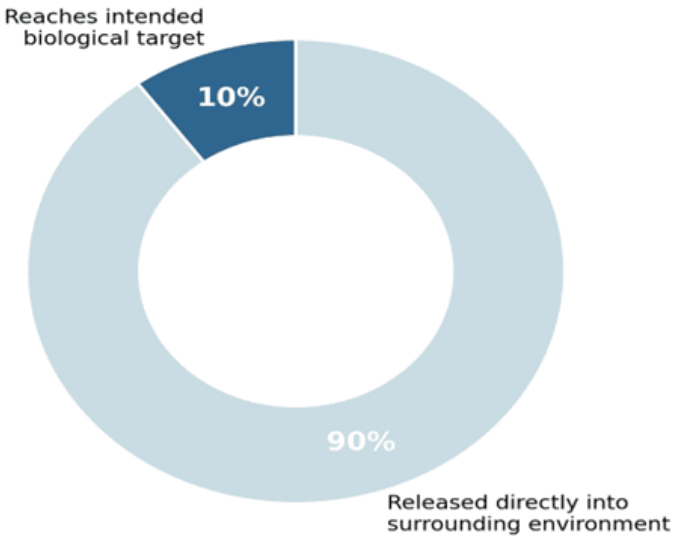
A structured literature search was conducted using PubMed, Scopus, Web of Science, Google Scholar, and WHO publications to identify studies on DDT and its environmental and health impacts. Keywords included "DDT," "persistent organic pollutants," "bioaccumulation," "human health," "plant health," "wildlife," "ecosystem," "One Health," and related terms. Peer-reviewed research articles, systematic reviews, and reports published in English were included, while duplicate and irrelevant studies were excluded. The selected literature was narratively synthesized under themes including environmental behavior, human health, plant health, wildlife, ecosystem disruption, legacy contamination, climate change, and regulatory strategies.

3. Global Distribution and Environmental Behavior of DDT

Despite widespread international restrictions, the global production and release of dichlorodiphenyltrichloroethane (DDT) remain ongoing concerns. Production is currently concentrated in three nations: India, China, and the Democratic People's Republic of Korea (DPRK; North Korea). India represents by far the largest global manufacturer, where the chemical is synthesized primarily for disease vector control, such as combating malaria-carrying mosquitoes (3).

However, the application efficiency of the pesticide is notably poor; it is estimated that only 10% of applied DDT actually reaches its intended biological targets. The remaining 90% is released directly into the surrounding environment, initiating a persistent cycle of

Figure A. Fate of applied DDT: target efficiency vs. environmental release



Source: van den Berg (3).

cntamination (3).

Figure A. Fate of applied DDT — only about 10% reaches its intended biological target; ~90% disperses into the environment (3).

Degradation Kinetics

Once introduced via agricultural or vector-control applications, DDT undergoes environmental and metabolic transformation into its primary metabolites: dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenyldichloroethane (DDD). Because analytical assessments frequently measure these compounds collectively, environmental data is commonly reported as ΣDDT (4).

The environmental persistence of ΣDDT fluctuates significantly depending on the specific environmental matrix and prevailing climate conditions:

- ATMOSPHERIC PERSISTENCE: 4 to 12 days
- TEMPERATE AND TROPICAL SOILS: 1 to 3 years
- POLAR AND ARCTIC SOILS: 20 to 30 years (as reduced temperatures markedly suppress microbial degradation).

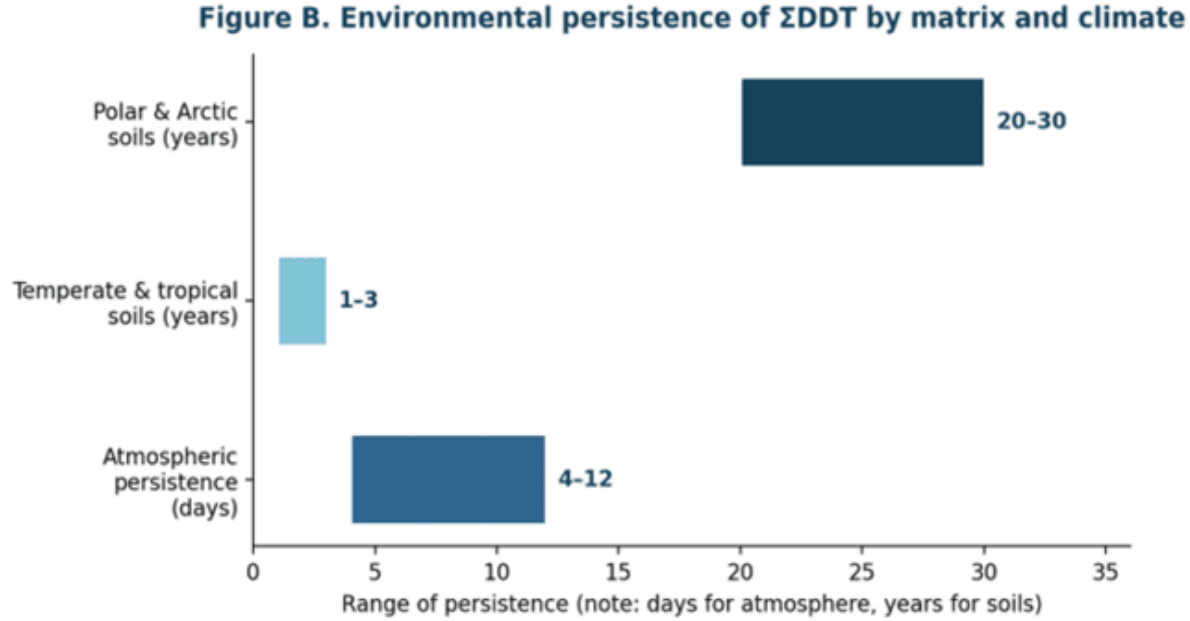


Figure B. Environmental persistence of ΣDDT by matrix and climate (4).

Soil Sedimentation & Absorption

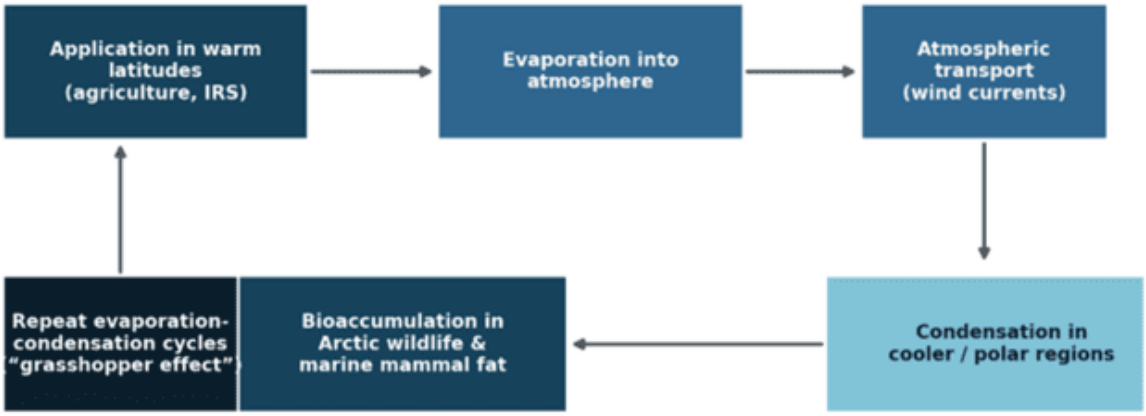
Characterized by high hydrophobicity and minimal aqueous solubility, DDT exhibits a profound affinity for organic matter and suspended particulates. These materials eventually deposit into aquatic beds, where sediments serve as dual-purpose entities: acting as stable sinks that sequester the compound from biodegradation, and functioning as perpetual secondary sources of chronic exposure. In severely contaminated regions, sediment concentrations may reach peaks of 1,600 mg/kg, precipitating extensive contamination of groundwater and benthic ecosystems (5).

Long Range Transport Mechanisms

Despite its limited volatility, DDT achieves global distribution through ocean currents, faunal migrations, and atmospheric circulation patterns. Through the process of global distillation, frequently termed the "grasshopper effect," the chemical undergoes repetitive cycles of

evaporation in warmer latitudes followed by condensation in cooler regions. This mechanism facilitates the transport of the contaminant thousands of miles from its point of origin, culminating in substantial bioaccumulation within pristine Arctic habitats and the adipose tissues of marine mammals(2).

Figure C. Long-range atmospheric transport of DDT: the "grasshopper effect"



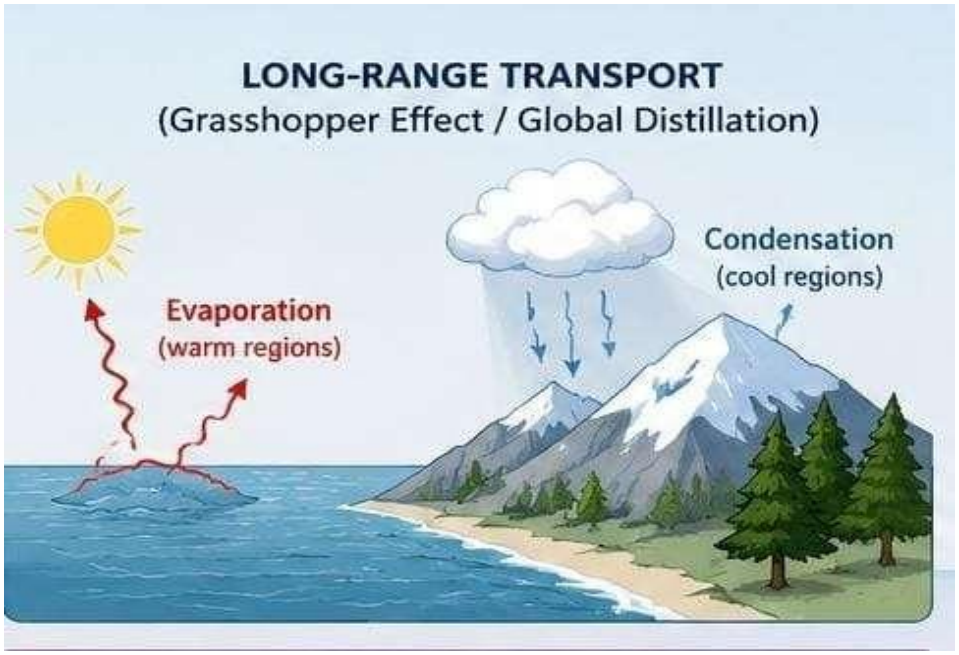


Figure C. Long-range atmospheric transport of DDT via repeated evaporation–condensation cycles, the "grasshopper effect" (2).

Bioaccumulation and Biomagnification

The lipophilic nature of DDT facilitates its rapid absorption and sequestration within the adipose tissues of living organisms. This physiological storage mechanism results in specific ecological vulnerabilities (5):

Process	Description
Assimilation	While aquatic species primarily ingest the chemical from their surrounding water, terrestrial wildlife accumulate significant concentrations through the consumption of contaminated food sources.
Metabolic release	During periods of intense physiological demand — such as migration, breeding, or dormancy — stored lipids are mobilized, causing a sudden influx of DDT back into the systemic circulation and potentially inducing delayed toxicological responses.
Trophic magnification	Due to its extreme resistance to biodegradation, the compound undergoes substantial amplification as it ascends the food chain, ultimately leaving top-tier predators, including human populations, with the most significant toxicological burdens.

Table 1. Ecological vulnerabilities arising from the lipophilic storage of DDT (5).

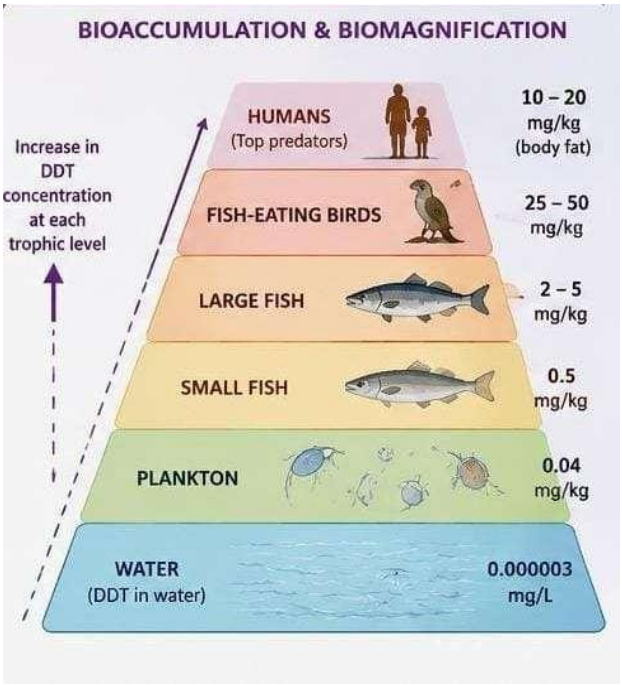


Figure D. Trophic biomagnification of ΣDDT up the food chain (2,5).

4. Impact on Human Health

Many of the recent scientific reviews suggest a long-lasting, multisystemic, multigenerational impact of these substances in the human body. It poses a range of acute and chronic health risks to humans, from neurotoxicity to endocrine disruption and potential carcinogenicity.

Routes of Contamination

Human DDT contamination varies by region and exposure pattern, occurring via diverse environmental and dietary pathways.

Dietary intake of fatty animal products (meat, fish, poultry, and dairy) is the primary route globally. However, in regions utilizing indoor residual spraying (IRS) for malaria control, non-dietary exposure through indoor air, dust, and soil is substantial. A study in South Africa

confirmed widespread contamination, finding DDT across all indoor media: air (mean 3900 ng m^{-3}), floor dust (mean 1200 µg m^{-2}), outdoor soil (mean 25 µg kg^{-1}), and drinking water (mean 2 µg L^{-1}) (6).

In the Arctic, indigenous groups face high exposure by consuming marine mammals despite no local DDT use. In the U.S., DDT contaminates 375 of 1,867 National Priorities List waste sites, with risk depending on proximity. Breastfeeding is another major pathway, transferring accumulated DDT and DDE in breast milk to infants. Washing produce from DDT-using areas provides only limited population-level reduction due to the prevalence of animal-derived dietary sources (7).

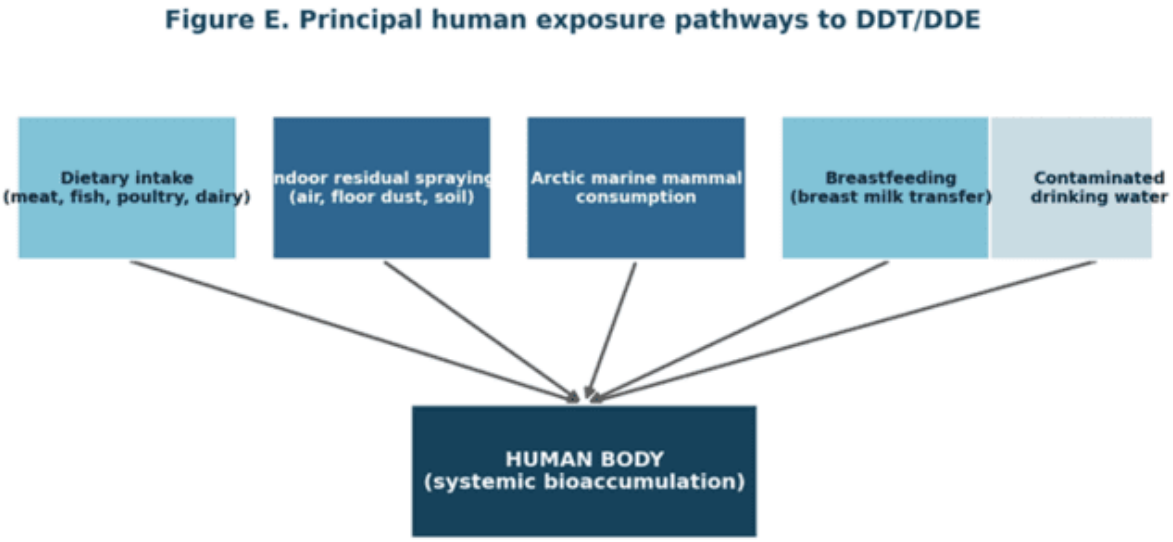


Figure E. Principal human exposure pathways to DDT/DDE (6,7).

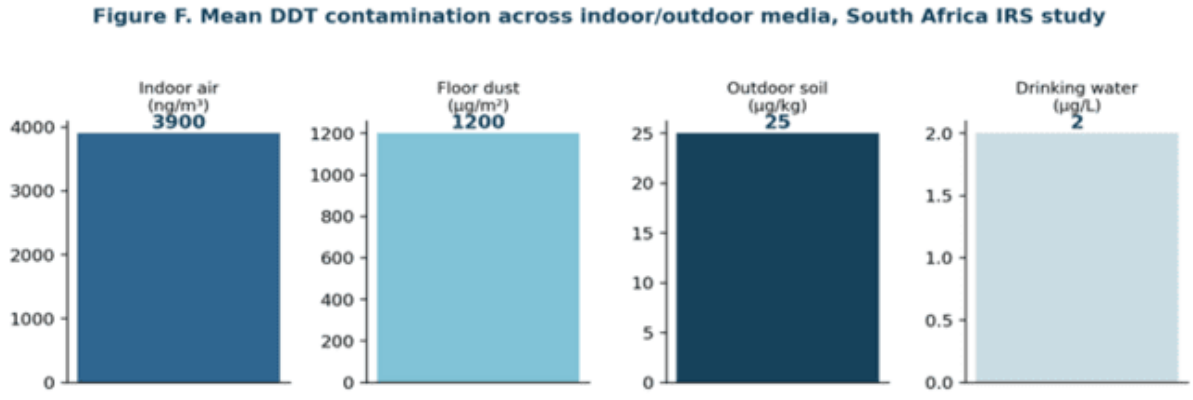


Figure F. Mean DDT contamination across indoor and outdoor media reported in a South African indoor-residual-spraying (IRS) study (6).

Mechanisms of Action

Molecular research shows that DDT and its metabolites disrupt ionic balance and endocrine signaling. In nervous tissues, DDT impairs the membrane sodium-potassium pump, destabilizing calcium regulation and triggering cell death via apoptosis or necrosis (marked

by cellular edema, lost membrane integrity, and nuclear disintegration). This neurotoxicity causes acute tremors and convulsions. Additionally, these endocrine disruptors bind estrogen receptors to alter gene expression, impairing reproduction and development. Highly lipophilic, DDT accumulates in fat reserves, where its extreme persistence causes chronic, systemic toxicity (6,16).

Neurotoxic Effects

The neurotoxic properties of DDT produce both acute physical manifestations and chronic, long-term neurodegenerative pathologies. In clinical settings, acute high-dose exposure triggers classical central nervous system hyperexcitation, clinically characterized by full-body tremors, hyper-irritability, and generalized convulsions [9]. Epidemiological evaluations of aging populations, such as data compiled from the National Health and Nutrition Examination Survey (NHANES), have firmly linked elevated baseline serum levels of DDE with measurable cognitive deficits and accelerated declines in psychomotor performance [12].

Furthermore, post-mortem human brain autopsies have revealed significantly elevated concentrations of DDT residues within the cerebral tissue of Alzheimer's disease patients compared to healthy controls [12]. Animal trials corroborate these findings by demonstrating that chronic exposure to sub-toxic doses alters vital dopaminergic pathway dynamics. In rodent models, DDT metabolites drastically decrease the presence of membrane dopamine transporters (DAT) and downregulate vesicular monoamine transporters (VMAT2), leading to elevated oxidative stress and cellular apoptosis within dopaminergic pathways, mirroring the structural degradation seen in Parkinson's disease [11].

Hormonal Interference

Recognized as a potent endocrine-disrupting chemical (EDC), DDT significantly compromises the structural maturation and physiological homeostasis of hormonal systems [9,10,11]. Longitudinal evaluations of human cohorts associate chronic residues with profound reproductive impairments, including precocious pubertal development, reduced seminal quality, and lower fecundity rates [10]. Experimental data involving human mesenchymal stem cells (hMSCs) indicate that such exposure triggers aberrant, estrogen receptor-mediated differentiation, which subverts standard tissue regeneration by stimulating transcription pathways like AP-1 and ERK1/2 [10].

Furthermore, the compound exerts direct toxicological pressure on the adrenal glands; studies in both mammalian and avian models reveal that technical-grade DDT induces significant adrenal hypertrophy and the breakdown of cortical cells [11]. The metabolites specifically target functional cortical zones, selectively inhibiting the production of androstenedione and aldosterone while simultaneously interfering with catecholamine secretion from chromaffin tissues [11]. These endocrine disruptions collectively destabilize systemic metabolic regulation, homeostatic equilibrium, and essential stress-response mechanisms [10,11].

Oncogenic Potential

The International Agency for Research on Cancer (IARC) classifies DDT as a Group 2B agent, identifying it as a probable human carcinogen [9]. Longitudinal human health studies, such as the Child Health and Development Studies (CHDS), provide crucial clinical evidence showing that women exposed to high levels of p,p'-DDT in utero or during early childhood

susceptibility windows experience up to a five-fold increase in their long-term risk of developing breast cancer [13].

Molecular analyses indicate this oncogenic pathway is driven by stable epigenetic modifications, with prenatal exposure causing altered DNA methylation patterns across specific genes (CYP1A1, CCDC85A) that govern pubertal breast development and tumor suppression [13]. In vivo animal models confirm this clear oncogenic potential, particularly within hepatic tissues. Long-term feeding trials in F344 rats demonstrate that chronic p,p'-DDT exposure induces the formation of pre-neoplastic, hepatocellular eosinophilic foci [9]. Because DDT behaves as a non-genotoxic hepatocarcinogen, its continuous promotion of oxidative DNA damage, paired with its capacity to do a complete block, allows initiated cells to bypass normal growth controls and progress into hepatic neoplasia [9].

Other Systemic Complications

Beyond clear neurological and reproductive damage, chronic accumulation of DDT drives severe, multi-systemic medical complications across various internal organ systems. In the liver, chronic exposure induces marked hepatotoxicity, characterized pathologically by significant increases in total liver weight and widespread hepatocellular hypertrophy [9]. Renal evaluations in animal models demonstrate that the kidneys also experience severe chronic burden, showing clear signs of tubular degeneration and impaired filtration metrics when exposed to persistent metabolic loads of the chemical [9,11].

Furthermore, systemic lipid mobilization dynamics introduce a unique danger. Because DDT and its metabolites are stored securely within adipose tissue reserves, any physiological event that triggers rapid fat metabolism — such as prolonged illness, intensive fasting, or natural migration — suddenly floods the active circulatory system with high concentrations of the trapped toxin [11]. This rapid mobilization induces acute, delayed toxic shocks, triggering sudden metabolic destabilization and extensive secondary organ damage long after the initial exposure occurred [11].

Organ / system	Documented effects	Ref(s)
Nervous system	Membrane Na^+/K^+ pump impairment, tremors & convulsions, cognitive decline, Parkinson's-like dopaminergic degeneration, Alzheimer's-linked cerebral residues	[9,11,12]
Endocrine / reproductive	Estrogen-receptor gene alteration, precocious puberty, reduced sperm quality, adrenal hypertrophy & cortical breakdown	[9,10,11]
Oncogenic	IARC Group 2B probable carcinogen; up to 5-fold breast cancer risk after early-life exposure; hepatocellular pre-neoplastic foci	[9,13]
Hepatic	Increased liver weight, hepatocellular hypertrophy, oxidative DNA damage	[9]
Renal	Tubular degeneration, impaired filtration	[9,11]

Systemic / lipid mobilization	Delayed toxic shock upon rapid fat metabolism (illness, fasting, migration)	[11]

Table 2. Summary of documented DDT effects on human organ systems, as described in this review.

5. Impact on Plant Health & Nutrition

Dichlorodiphenyltrichloroethane (DDT) and its degradation products, primarily dichlorodiphenyldichloroethylene (DDE), create a chronic phytotoxic environment that impairs plant fitness, metabolism, and nutrition (1,5). High soil concentrations of DDT disrupt seed germination and seedling development, leading to stunted growth and reduced crop biomass. DDT alters cellular homeostasis and causes severe chlorosis by degrading chlorophyll a and b (1).

Additionally, it hinders the absorption of key micronutrients and macronutrients, including nitrogen, phosphorus, and potassium, from the soil. This nutritional deficiency undermines global crop quality and yield while increasing plant susceptibility to secondary stressors like drought and pathogens (1,6).

Plant Uptake

Due to its extreme hydrophobicity and low water solubility, DDT binds strongly to soil organic carbon, creating long-term repositories that become secondary exposure sources. Plants actively absorb DDT, DDE, and DDD through root and foliar pathways. In root absorption, lipophilic compounds cross the epidermis into cortical tissues via passive diffusion. Cucurbitaceae (including pumpkins and zucchini) translocate these residues exceptionally well into edible shoots and fruits using specialized low-molecular-weight proteins. For foliar absorption, plant canopies trap volatilized gaseous DDT from the soil or capture windblown contaminated dust directly through open stomata [1,5].

Physiological Effects

At the cellular level, DDT acts as a potent phytotoxic agent that disrupts plant metabolic cascades and structural integrity. Once inside plant cells, DDT induces severe oxidative stress by stimulating the overproduction of reactive oxygen species (ROS) like superoxide radicals and hydrogen peroxide. This ROS surge overwhelms enzymatic defenses (such as superoxide dismutase and catalase), causing membrane lipid peroxidation and irreversible genomic DNA damage.

Additionally, it alters mitochondrial and chloroplast functions by blocking electron transport chains and inhibiting ATP synthase, compromising energy production and photosynthetic efficiency. Finally, DDT disrupts hormonal signaling, altering indole-3-acetic acid (IAA) and abscisic acid balances to trigger premature leaf senescence, impaired root development, and vascular bundle apoptosis [1,6].

Physiological target	Effect of DDT/DDE exposure
Germination & growth	Disrupted seed germination, stunted seedling development, reduced biomass

Photosynthetic apparatus	Chlorosis via chlorophyll a & b degradation; blocked electron transport chains; inhibited ATP synthase
Nutrient uptake	Hindered absorption of nitrogen, phosphorus, and potassium
Oxidative status	Overproduction of ROS (superoxide, H_2O_2); overwhelmed SOD/catalase defences; lipid peroxidation; DNA damage
Hormonal signalling	Altered IAA and abscisic acid balance; premature leaf senescence; impaired root development; vascular bundle apoptosis

Table 3. Physiological effects of DDT/DDE on plants (1,6).

Transfer to Food Chains

The high bioconcentration potential and metabolic stability of DDT drives its hazardous migration from contaminated vegetative matrices into higher trophic levels, generating a severe biomagnification cascade across global food webs [1,2,5]. As primary producers, plants serve as the foundational entry vector for these lipophilic toxins into terrestrial and aquatic ecosystems [1,2]. When herbivorous pests, livestock, or foraging wildlife graze on contaminated vegetation, they absorb the stored DDT residues and sequester them inside their lipid-rich tissues [1,2,4,8].

Because organisms at higher trophic levels lack the specialized metabolic enzymes required to rapidly degrade or eliminate DDE, the chemical amplifies in concentration at each successive step of the food chain [2,5]. Consequently, apex predators — such as birds of prey, large marine mammals, and humans consuming meat, dairy, and fish — bear the highest toxic burden [2,4,5]. This persistent biotransport introduces a severe ecological feedback loop, where historic, soil-bound agricultural inputs continue to biomagnify up the global food web, triggering reproductive failure, systemic pathologies, and chronic health hazards far from the point of origin [2,5,6].

6. Impact on Wildlife

The global contamination of ecosystems by DDT and its highly stable environmental metabolite, DDE, has caused widespread disruptions across diverse wildlife populations [1,2,5]. Because ΣDDT is highly lipophilic and strongly resists metabolic degradation, it rapidly enters local trophic webs through primary producers and lower-order consumers [1,5]. Wild animals face dual exposure vectors: direct bioconcentration from contaminated physical environments (such as water or sediment) and indirect biomagnification via the consumption of contaminated prey [2,5,13].

Because apex predators consume organisms that have already concentrated the compound, the internal tissue burden multiplies by several orders of magnitude as it ascends the food chain [2,5]. This results in massive bioaccumulation within vulnerable wildlife populations globally, even in remote regions like the Arctic where no industrial application has occurred [5,13]. The persistent internal exposure triggers severe systemic complications, including widespread reproductive failure, population declines, altered immune defenses, and acute neurotoxicity during seasonal periods of high energy stress [1,2,8].

Aquatic Organisms

Aquatic ecosystems serve as the ultimate sinks for global DDT residues due to heavy agricultural runoff and wastewater discharge [1,7]. Aquatic organisms, particularly fish and invertebrates, are highly susceptible to direct bioconcentration, absorbing the dissolved chemical rapidly across their gills and skin [2,6]. In fish species, chronic exposure to sub-lethal concentrations of ΣDDT alters vital biochemical and physiological pathways, inducing severe oxidative stress, cellular damage in hepatic tissues, and significant metabolic exhaustion [2,6].

Toxicological trials demonstrate that DDT alters critical iono-regulation by inhibiting adenosine triphosphatases Na^+/K^+-ATPase within the gill epithelium, which severely disrupts the internal osmotic balance [2,6]. Furthermore, developmental toxicity trials highlight high mortality rates during early life stages [2]. When adult female fish mobilize internal lipid stores to develop eggs, high concentrations of accumulated DDT transfer directly into the oocytes, triggering extensive skeletal deformities, neural tube defects, and massive mortality in the developing larvae long before they reach maturity [2,6,14].

Birds

Avian populations, particularly predatory birds and scavengers sitting at high trophic positions (such as eagles, falcons, and pelicans), have historically borne the most severe, visible damage from environmental DDT contamination [2,5]. The primary driver of this crisis is the distinct physiological mechanism of DDE-induced eggshell thinning [2,5,15]. Once ingested via contaminated fish or small mammals, DDE targets the shell gland (uterus) of female birds, where it strongly inhibits the enzyme prostaglandin synthetase and impairs active Ca^{2+}-ATPase transport mechanisms [2,15]. This molecular disruption limits the transport of calcium carbonate to the eggshell gland during formation, leading to structurally compromised, thin-shelled eggs that easily crack or crush under the weight of incubating parents [2,15].

Furthermore, embryonic toxicity trials confirm that DDT residues crossing the egg yolk act as potent endocrine disruptors within the developing egg, causing significant embryo mortality, structural skeletal malformations, and severe gonadal feminization in male hatchlings, which deeply undermines long-term avian reproductive success and population stability [2,5].

Figure G. Mechanism of DDE-induced eggshell thinning in birds

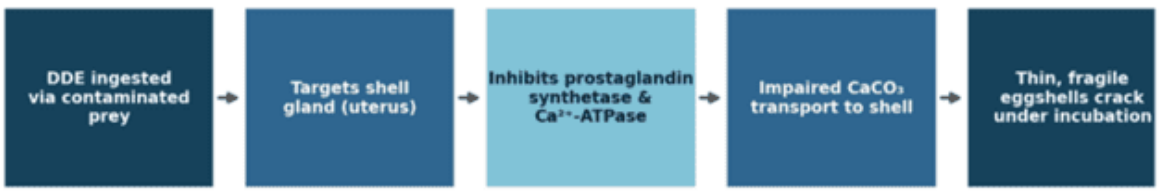


Figure G. Mechanism of DDE-induced eggshell thinning in birds (2,15).

Terrestrial Mammals

In terrestrial mammals, chronic exposure to DDT triggers multi-systemic physiological dysfunction, targeting the reproductive, endocrine, and central nervous systems [2,4,8,10]. Because the chemical exhibits structural similarities to natural hormones, it acts as a classic endocrine-disrupting chemical (EDC) in wild mammals [3,10]. The environmental metabolite p,p'-DDE acts as a powerful androgen receptor antagonist, which severely impairs reproductive health in male mammals, leading to testicular atrophy, compromised spermatogenesis, cryptorchidism, and reduced fertility metrics [2,8].

Animal models and trials in rodents and large marine mammals demonstrate that the adrenal cortex is a primary target of structural toxicity [4,10]. Chronic accumulation drives extensive cell degeneration within the adrenal gland, altering the metabolic synthesis of essential corticosteroids and heavily disrupting the animal's natural stress response, metabolic control, and immune functions [4,10]. Additionally, because these lipophilic toxins are sequestered securely inside adipose tissues, any period of high lipid mobilization — such as seasonal migration, winter hibernation, or lactation — suddenly releases massive, toxic concentrations back into the active bloodstream, inducing severe secondary organ failure [4,8].

Taxon	Key documented effects	Ref(s)
Aquatic organisms (fish, invertebrates)	Gill/skin bioconcentration, Na^+/K^+-ATPase inhibition, oxidative stress, hepatic damage, larval skeletal/neural-tube defects, high early-life mortality	[2,6,14]
Birds	DDE-induced eggshell thinning (inhibited prostaglandin synthetase & Ca^{2+}-ATPase), embryo mortality, skeletal malformation, gonadal feminization in male hatchlings	[2,5,15]
Terrestrial mammals	Androgen-receptor antagonism, testicular atrophy, impaired spermatogenesis, adrenal cortical degeneration, immune/metabolic disruption, delayed toxic shock on fat mobilization	[2,3,4,8,10]

Table 4. Summary of documented DDT/DDE effects across wildlife taxa.

7. Ecosystem & Biodiversity Disruption

The widespread environmental infiltration of dichlorodiphenyltrichloroethane (DDT) and its persistent degradation products accelerates the structural decline of global ecosystems and threatens biological diversity [1,5]. Because total DDT behaves as a highly stable, non-target toxin, its ecological damage cascades well beyond designated vector control areas [1,7]. By eliminating foundational aquatic and terrestrial invertebrates, DDT disrupts critical predator-prey dynamics and triggers structural top-down trophic cascades [2,6].

For example, the toxic decline of sensitive keystone insect and crustacean populations alters resource availability, reducing species richness throughout connected food webs [2,5,6]. Furthermore, its high biomagnification potential leaves apex predators — such as raptors, marine mammals, and large predatory fish — vulnerable to severe chronic exposure [2,5]. This persistent toxic pressure causes widespread reproductive failures, suppresses immune

health, and increases mortality rates, leading to sharp population drops among highly vulnerable and endangered species [2,8]. By selectively eliminating sensitive organisms while favoring more resilient, toxin-tolerant species, DDT contamination fundamentally alters natural community composition, shifts ecosystem dynamics, and permanently lowers overall ecosystem stability and resilience [1,5,7].

8. Legacy Contamination

Despite stringent international production bans enacted decades ago, DDT continues to pose a persistent threat as a premier example of global legacy contamination [1,5]. Due to its chemical stability, low water solubility, and high octanol-water partition coefficient, the parent compound and its primary metabolite, DDE, resist natural chemical and microbial breakdown [1,2]. Over years of heavy application, massive quantities of ΣDDT bound tightly to organic fractions within topsoils and aquatic sediments, transforming these geographic sinks into long-term secondary sources of pollution [1,5].

Natural processes — such as agricultural soil erosion, seasonal floods, and benthic disturbance — continually mobilize these buried historical deposits back into active surface waters and biotic systems [1,7]. This continuous cycling subjects modern organisms to chronic, low-dose exposure loops [1,6]. Consequently, historical agricultural inputs from the mid-20th century continue to contaminate modern food webs, showing no significant signs of environmental clearance and causing ongoing toxic stress in areas where the pesticide has not been used for generations [1,2,5].

9. Climate Change & DDT

The environmental behavior, global transport, and toxicity of legacy DDT deposits are increasingly influenced by ongoing global climate change [5,7]. Through the planetary phenomenon known as global distillation, historic DDT residues evaporated from warmer application zones and migrated via atmospheric currents toward cooler, higher-latitude regions like the Arctic [5]. For decades, these shifted toxins were safely trapped and immobilized within polar ice sheets, sub-polar glaciers, and deep permafrost layers [5,13]. However, accelerating global temperatures and rapid polar warming are inducing widespread melting of these frozen sinks, remobilizing large stores of legacy DDT back into active oceans, coastal zones, and terrestrial environments [5,13]. This climate-driven release triggers a sudden surge of contamination within pristine Arctic food webs [13]. Furthermore, rising ambient temperatures elevate the base metabolic rates of aquatic and terrestrial organisms, increasing their food consumption and direct bioconcentration rates [6]. This thermal stress works in tandem with chemical exposure, amplifying the overall biomagnification of ΣDDT up the food chain and intensifying its chronic toxic effects on vulnerable wildlife [5,6,13].

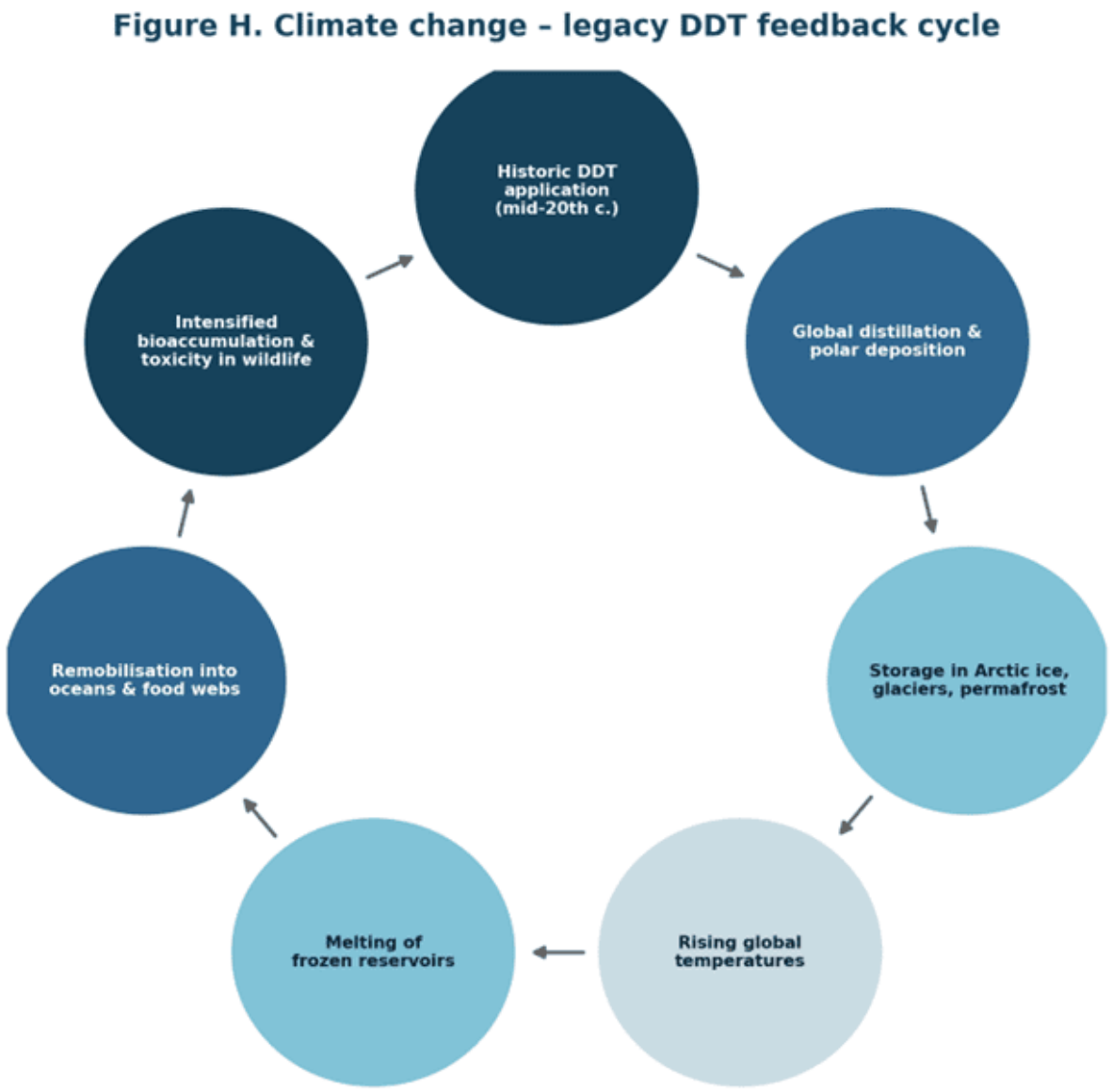


Figure H. Climate change – legacy DDT feedback cycle: warming remobilises historic polar deposits, intensifying bioaccumulation (5,6,13).

10. Regulations & Sustainable Solutions

Managing the ongoing global hazards of DDT requires strict, legally binding international frameworks paired with modern, eco-friendly technological innovations [1,3,4]. Under the global Stockholm Convention on Persistent Organic Pollutants, the manufacturing and open use of DDT are highly restricted, allowing exceptions only for crucial public health interventions, such as indoor residual spraying (IRS) for malaria vector control in specific regions [3,4].

To completely eliminate reliance on this toxic compound, current sustainable strategies focus on implementing Integrated Vector Management (IVM) systems [3]. These programs combine non-chemical control methods — such as distributing long-lasting insecticide-treated bed nets (LLINs), modifying local habitats to eliminate mosquito breeding sites, and deploying biopesticides — with safer, highly biodegradable chemical alternatives [3,4]. Simultaneously, restoring historical legacy hotspots relies heavily on advanced green

technologies [1]. Innovations in microbial bioremediation and targeted phytoremediation leverage specialized bacterial strains and deep-rooted plants to slowly extract, isolate, and break down persistent soil-bound ΣDDT residues into harmless byproducts, offering an effective pathway toward global environmental recovery [1].

Strategy	Approach	Ref(s)
Legal / policy control	Stockholm Convention restrictions on manufacture and open use; exceptions limited to public-health IRS	[3,4]
Integrated Vector Management (IVM)	Long-lasting insecticide-treated bed nets (LLINs), breeding-site habitat modification, biopesticides, safer biodegradable alternatives	[3,4]
Microbial bioremediation	Specialized bacterial strains break down soil-bound ΣDDT residues into harmless byproducts	[1]
Phytoremediation	Deep-rooted plants extract and isolate persistent DDT residues from contaminated soils	[1]

Table 5. Regulatory and sustainable mitigation strategies for DDT (1,3,4).

11. Conclusion

The legacy of dichlorodiphenyltrichloroethane (DDT) presents a central dilemma in modern environmental toxicology. Although it historically offered clear advantages by curbing severe vector-borne illnesses and enhancing global agricultural productivity, its high lipophilicity, deep environmental persistence, and extreme toxicity have severely compromised the global biosphere. Ample scientific data spanning humans, plants, and wildlife demonstrates that DDT and its breakdown products interfere with basic cellular, endocrine, and ecological functions.

These disruptions across all the trophic levels manifest as compromised photosynthetic pathways in vegetation, severe eggshell thinning in avian species, and chronic systemic illnesses, cognitive deterioration, and carcinogenic developments in humans.

Additionally, this pollutant's transboundary threat is highlighted by its atmospheric transport across immense distances, a challenge now compounded as warming climates release historical deposits formerly trapped in polar ice. The persistent detection of DDT in untouched habitats and modern food webs highlights the enduring shadow cast by past chemical applications. Moving forward requires a equilibrium between: safeguarding populations in malaria-prone areas while accelerating the adoption of eco-friendly Integrated Vector Management and advanced green bioremediation methods.

In the end, systematically eliminating legacy organochlorines and restoring degraded habitats remains critical to preserving worldwide biodiversity, defending public health, and maintaining ecological boundaries.

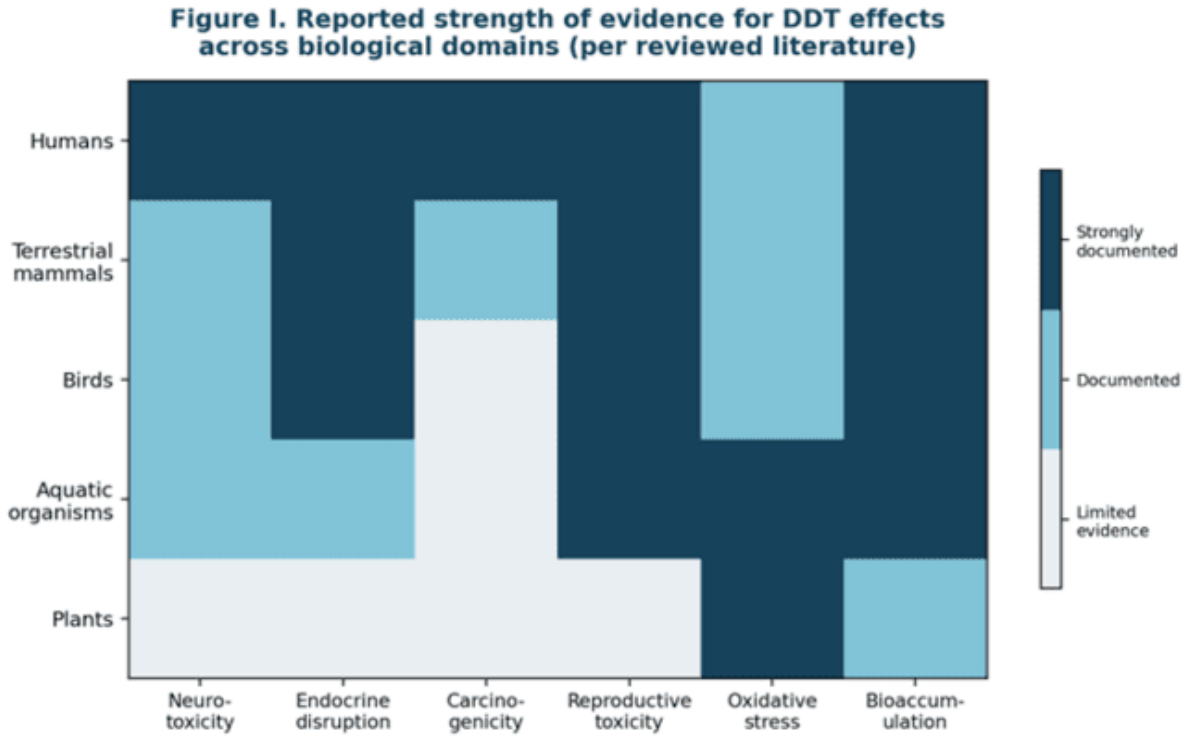
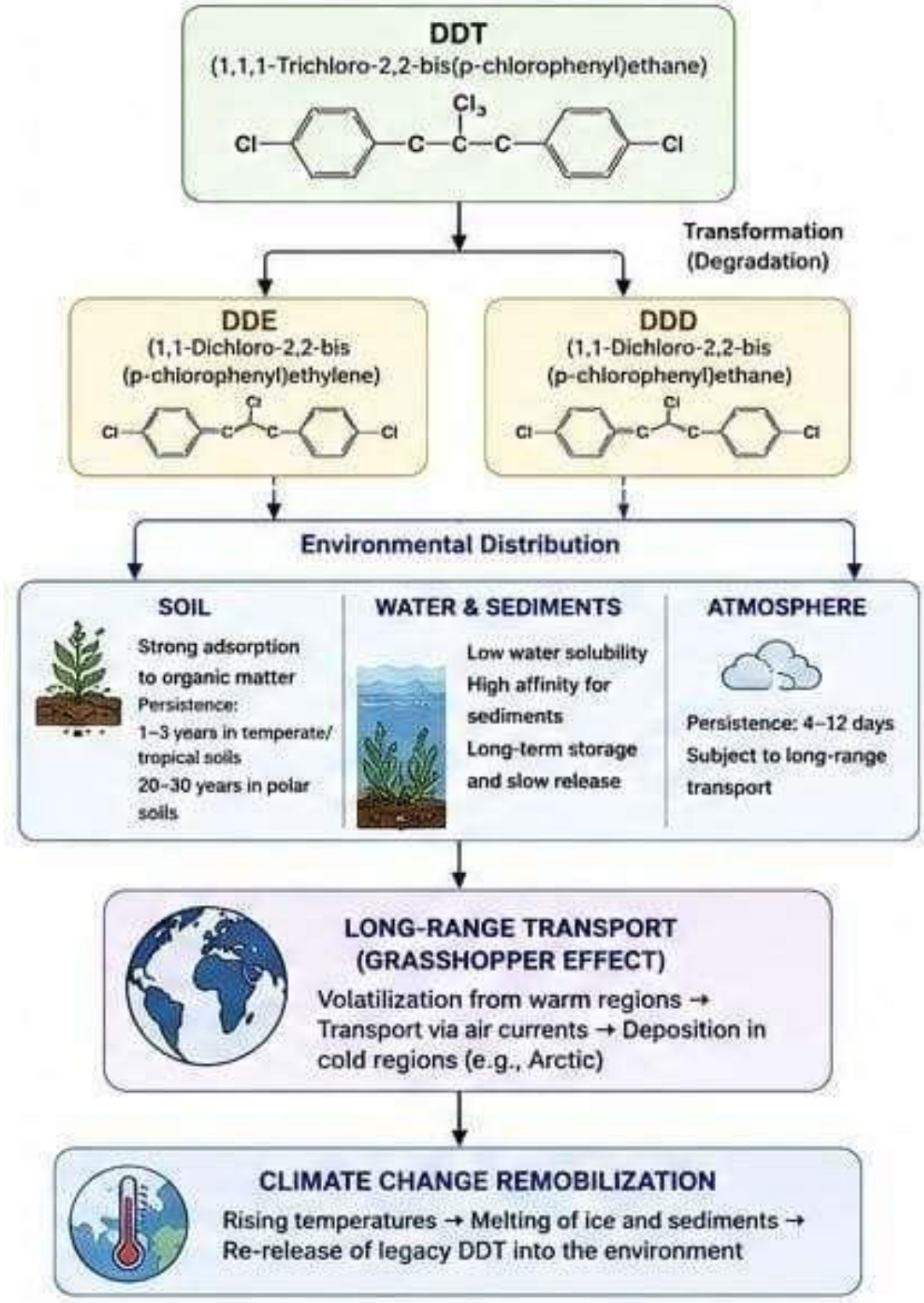


Figure I. Synthesis: reported strength of evidence for DDT effects across biological domains, based on the studies cited throughout this review.

Figure 2. Environmental Fate of DDT



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ONE HEALTH APPROACH TO MICROPLASTICS & NANOPLASTICS

A Focused Systematic Review of Sources, Exposure Pathways, and 360° Impacts on Environment, Animal, Plant, Aquatic, and Human Health

TABLE OF CONTENTS

ABSTRACT

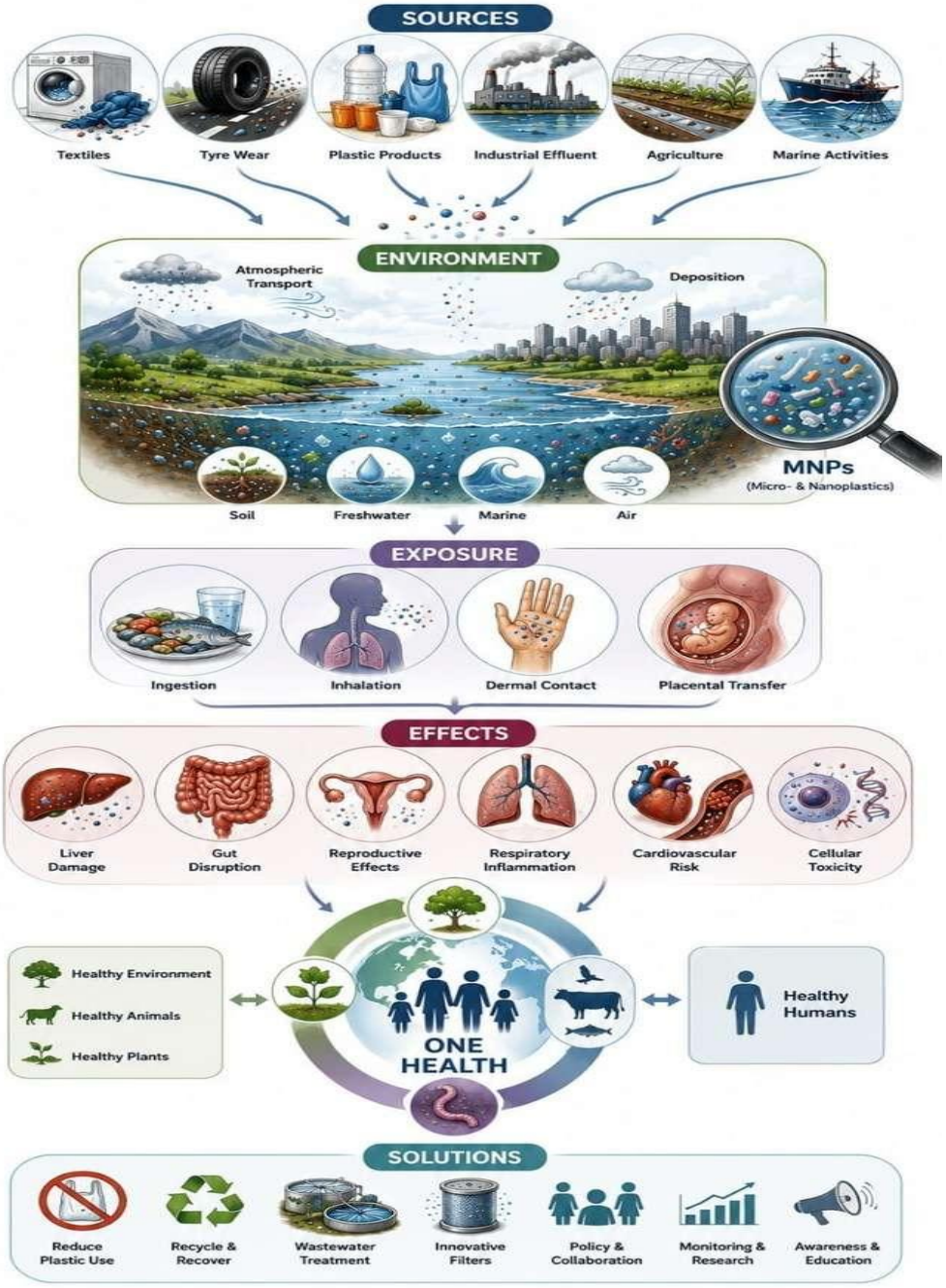
The rapid growth of global plastic production has resulted in the widespread accumulation of microplastics (MPs) and nanoplastics (NPs) across the globe, thus establishing them as emerging contaminants of critical global concern. Their extreme environmental persistence, high mobility, and property to adsorb toxic chemicals and pathogenic microorganisms have transformed them into a profound One Health challenge, while simultaneously undermining environmental integrity, threatening biodiversity, compromising animal health, disrupting food security, and imperiling human well-being.

Current scientific evidences map a cascade of environmental distribution and transport pathways that drive the biological uptake of these particles, allowing them to move dynamically across ecosystems and complex food webs. The resulting impacts on plants, soil organisms, aquatic species, birds, and terrestrial mammals are extensive, systematically disrupting fundamental ecosystem processes, altering nutrient cycling, undermining community biodiversity, and interfering with climate-related biogeochemical functions.

Human risk is driven by ingestion, inhalation, and dermal exposure, with particle physicochemical properties governing the translocation of these particles across biological barriers. This internalization triggers systemic oxidative stress, mitochondrial dysfunction, genotoxicity, chronic inflammation, and

multi-organ toxicity. Growing biomedical evidence links chronic MNP exposure directly to hepatic, gastrointestinal, reproductive, cardiovascular, respiratory, and developmental disorders.
Evidences demonstrate that MNP pollution is a complex issue requiring coordinated multidisciplinary action. Mitigating this global threat requires international priorities focused on source reduction, improved municipal and industrial waste management, standardized monitoring methods, and integrating MNP surveillance into environmental and public health policy frameworks.

GRAPHICAL ABSTRACT



1.INTRODUCTION:

Plastics have revolutionised industry through their durability, adaptability, and cost-efficiency, finding application in sectors from packaging till construction to healthcare & technology. This widespread reliance, however, has come at a steep environmental cost, driving persistent pollution, ecosystem degradation, and mounting threats to wildlife. Global plastic manufacturing now exceeds 380 million tonnes annually and is projected to approach 1,000 million tonnes by 2050 (Gautam et al.

2024; Rellán et al. 2023), with an estimated 1–1.7 million tonnes entering the oceans each year as of 2023. A significant share of this material bypasses waste management systems entirely, leaking into the natural environment, where mechanical wear, photodegradation, and biodegradation progressively fragment it into microplastics (MPs, 1 μm–5 mm) and, ultimately, nanoplastics (NPs, <1 μm). Microplastics are broadly classified into two types: primary microplastics, intentionally manufactured at microscopic scale (such as pre-production pellets and cosmetic microbeads), and secondary microplastics, generated by the breakdown of larger consumer goods including textiles, packaging, and clothing. Together, MPs and NPs (collectively, MNPs) now represent a pervasive and poorly reversible global anthropogenic threat, accumulating across all major environmental compartments, food chains, and human tissues.

Their behaviour in biological and environmental systems is shaped by distinctive physicochemical properties. A high surface-area-to-volume ratio allows MNPs to act as dynamic vectors, adsorbing heavy metals, persistent organic pollutants, and pathogenic microbes onto their surfaces. Nanoplastics, by virtue of their sub-micron scale, exhibit enhanced bio-permeability, readily translocating across complex biological membranes and inducing disproportionate cellular toxicity per unit mass relative to larger fragments.

Emerging human-health evidence underscores the urgency of this issue. Someresearchers suggests that microplastic contamination in critical human tissues, including the brain, may be rising rapidly, reinforcing the growing risk that plastic exposure poses to human health and disease development. Separately, microplastics detected in vital biological sites such as the placenta have been linked to adverse developmental outcomes, including premature birth.

2.Sources and their Contribution

Quantifying the relative share of individual sources is central to designing effective mitigation. Recent comprehensive reviews consolidate source-apportionment analyses showing that synthetic textile laundering and tyre/road wear are the two largest identified contributors to primary ocean microplastic loading, followed by urban dust, road markings, marine coatings, personal-care products, and pre-production pellets (1,2). These widely-used estimates are summarised in Figure 1 and Table 1.

Source category	Relative contribution	Notes	Reference(s)
Synthetic textile laundering	Largest single primary source (~1/3 of primary MPs)	Microfibre shedding during washing	(1,2)
Tyre and road-surface wear	Second-largest primary source	Airborne and road-runoff transport	(1,2)
Urban/city dust	Major contributor	Paint, synthetic footwear, domestic abrasion	(1,2)
Road markings & marine coatings	Minor but consistent contributors	Thermoplastic paint and antifouling wear	(1,2)
Personal care products & pellets	Small, declining share	Microbeads (subject to bans); pre-production spillage	(1,2)

Secondary fragmentation of macroplastic litter	Majority of total environmental MP mass	Packaging, fishing gear, single-use plastics degrading in situ	(2,3)
Wastewater treatment plant effluent	Minority of influent load, but continuous	High sludge retention, but large effluent volumes	(2,3)

Table 1. Consolidated source apportionment of micro- and nanoplastic pollution reported

Independent of the primary/secondary distinction, land-based human activity accounts for the substantial majority of marine plastic debris, transported chiefly via rivers, with the remainder originating from at-sea activities such as fishing and shipping (1,2). Wastewater treatment plants (WWTPs) retain a large fraction of influent microplastics in sludge, but because of the very high volumes processed, the residual effluent fraction still represents a continuous, significant point source to receiving waters (2,3). These relationships are shown in Figure 2.

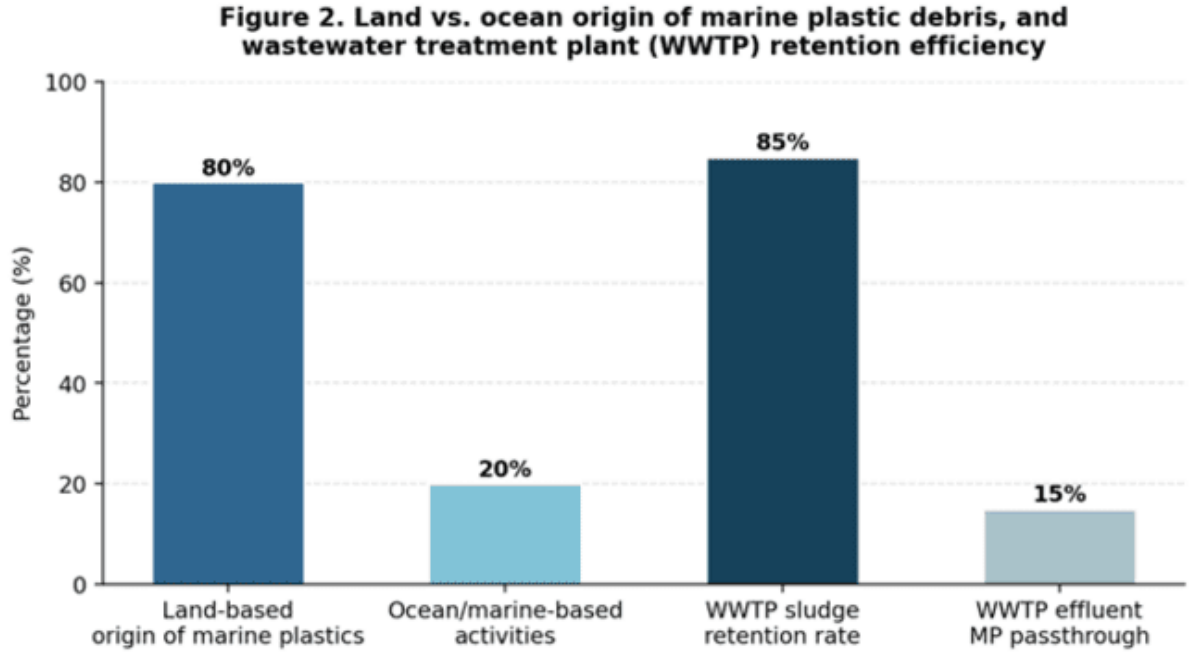
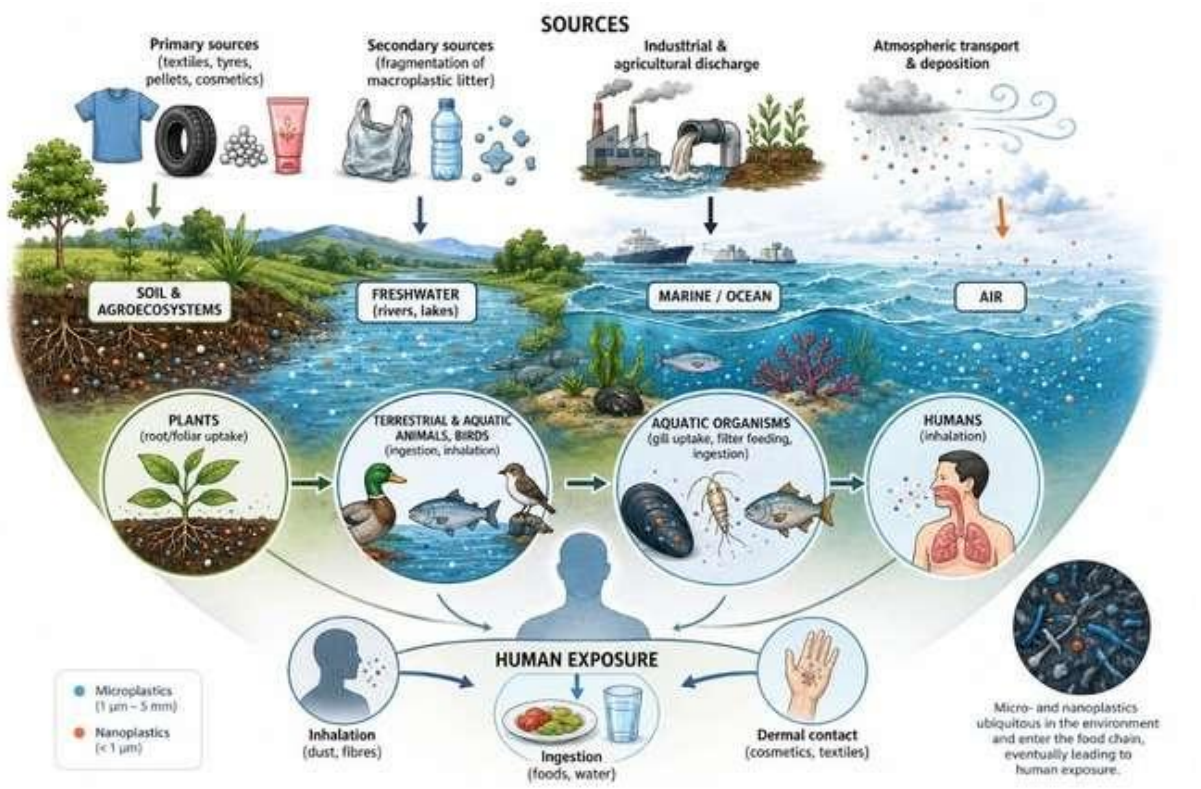


Figure 2. Land- versus ocean-based origin of marine plastic debris, and typical wastewater treatment plant (WWTP) retention efficiency for microplastics (1,2,3).

3.Modes of Entry

MNPs move between environmental compartments (soil, freshwater, marine, atmospheric) via surface runoff, wind erosion and atmospheric deposition, direct WWTP/agricultural discharge, and in-situ fragmentation of litter (1,2). Exposure of biota occurs principally through ingestion, inhalation, and dermal contact, with an additional route of placental/trans-generational transfer documented in mammals (3,10,11). Figure 3 summarises this cascade from source to human exposure.

FIGURE:3



4.Effects on Wildlife & Aquatic organisms

4.1 INVERTEBRATES

According to a literature review evaluating the impacts of MNPs on animal health, ingestion by aquatic invertebrates leads to decreased feeding rates, reduced fertility, delayed larval development, and heightened oxidative stress.

4.2 Birds

Birds are used as bioindicators of environmental MNP contamination because of their broad range and varied feeding ecology. Proteomic analysis of plastic-exposed seabird fledglings has revealed signatures consistent with multiorgan failure and neurodegeneration, including renal toxicity markers and reduced antioxidant capacity that could compromise physiological readiness for long-distance migration (5). Controlled experimental exposure of Japanese quail (Coturnix japonica) to microplastics has demonstrated size-specific effects, including altered cytokine profiles, oxidative stress markers, and hepatotoxicity following oral MP exposure, providing direct causal evidence to complement field observations in wild seabirds (6).

4.3 Terrestrial mammals and soil fauna

Terrestrial mammalian evidence remains comparatively limited but parallels aquatic findings, with documented gut microbiota disruption, oxidative stress, and reproductive effects in rodent models (4). Earthworms, used as model soil macrofauna, show altered survival, growth, and detoxification-enzyme (glutathione S-transferase) activity following exposure to microplastics, with effects varying by species, polymer type, and concentration; because earthworms burrow and redistribute ingested particles through the soil matrix, they may also actively facilitate further MNP dispersal within terrestrial ecosystems (15).

4.4 Aquatic organisms:

Research on environmental toxicology in fish indicates that the ingestion of microplastics (MPs) induces physical blockages within the digestive tract, resulting in decreased feeding, inhibited growth, internal lesions, inflammatory responses, and cellular toxicity.(12) These detrimental effects are further exacerbated when MNPs serve as transport vectors for co-adsorbed chemical contaminants. Within fish systems, exposure translates to structural damage of critical organs—including the intestines, liver, gills, and brain—accompanied by metabolic imbalances, altered behaviors, and impaired reproductive capabilities, with the overall severity of these conditions determined by particle size, exposure concentration, and polymer composition(4).

Additionally, a meta-analysis of experimental research highlights significant toxicological and pathological impacts of MPs in fish. These toxic reactions are predominantly associated with smaller particle fractions (≤5 μm in diameter), whereas larger polystyrene (PS) fragments (100 μm or more) yield no measurable adverse effects. In typical experimental designs, fish and roe are exposed to MP particles suspended in water tanks at concentrations spanning from 1 to 1,000 mg/L.(16)

At a broader scale, MP pollution destabilizes aquatic ecosystems by altering species diversity, impeding biogeochemical nutrient cycling, and compromising community resilience. Specialist and highly vulnerable species are disproportionately affected, causing cascading ecological pressures on dependent predator trophic levels.

TAXON	DOCUMENTED EFFECTS	References
Aquatic invertebrates	↓ feeding & fertility, delayed larval development, ↑ oxidative stress	(4)
Birds(seabirds & experimental avian model)	Proteomic signatures of multiorgan failure & neurodegeneration; hepatotoxicity; oxidative stress; altered cytokine	(5,6)
Terrestrial mammals (rodent models)	Gut microbiota disruption, oxidative stress, reproductive toxicity	(4)
Soil invertebrates (earthworms)	Altered survival, growth, detoxification-enzyme activity	(15)
Aquatic organisms(fishes)	Intestinal/liver/gill/brain damage, altered metabolism & behaviour, impaired fertility	(4,12)

5.Impact on PLANTS and Nutritional Quality

Farmland soils serve as expanding sinks for micro- and nanoplastics, primarily originating from plasticulture mulch, biosolids application, and falling atmospheric particles. These materials penetrate vegetable tissues through radical absorption before migrating into stalks, foliage, and produce, with biological consequences dictated by the specific plant variety and the dimensions of the polymer. Systematic evaluations of cultivated species indicate that exposure often suppresses biomass production, diminishes harvest yields, and triggers physiological aberrations that distort the mineral profile of tissues (7).

Furthermore, meta-analytic data from soil-based investigations confirm that plastic fragments significantly compromise vegetative development and nutrient uptake. For example, microscopic

polyethylene particles are linked to notable productivity losses in essential food crops, alongside disrupted concentrations of nitrogen, phosphorus, and potassium. Such pollution further degrades soil health by altering its pH and structural stability, which indirectly hinders root vitality and nutrient efficiency (8). These findings highlight a critical risk to food security: the ability of microplastics to act as vectors, facilitating the co-transport and accumulation of heavy metals and pesticides into the human diet (7,8).

6.IMPACT ON HUMAN HEALTH:

6.1 Exposure Pathways and Internalization

Humans are exposed to environmental MNPs through three main exposure routes: **ingestion** (via contaminated seafood, drinking water, salt, and packaged foods), **inhalation** (via indoor and outdoor airborne synthetic dust and clothing fibres), and **dermal contact** [1, 6, 10, 11]. Combined dietary ingestion and dust inhalation models calculate that adults internalize between 74,000 and 113,000 plastic particles annually [10].

Once inside the body, particle size dictates internal migration [6, 10, 11]. Larger microplastics >10μm are typically restricted to the gastrointestinal tract, while smaller particles, especially nanoplastics <1μm, readily cross complex biological barriers [6, 10]. These particles cross the protective intestinal epithelium and the blood-brain barrier via passive diffusion, cellular crack-entry, or active endocytosis, granting them direct access to systemic blood circulation and deeper organ tissues [6, 10].

6.2 PATHOPHYSIOLOGY

At the cellular tier, nanoplastics pose a disproportionately high toxicological risk per unit mass compared to larger fragments due to their high surface-area-to-volume ratio [2, 6, 9]. Once internalised within cells, they trigger a cascade of molecular stress reactions:

- **Oxidative Stress & ROS Generation:** MNPs directly stimulate the overproduction of reactive oxygen species (ROS), overwhelming the cell's natural antioxidant defenses [6, 9, 10].
- **Mitochondrial Collapse:** Accumulation within the cytoplasm disrupts mitochondrial membrane potential, impairing cellular energy (ATP) production and causing mitochondrial dysfunction [9, 10].
- **Genotoxicity:** MNP presence has been linked to structural DNA strand damage, which can lead to mutagenic changes or accelerate cellular apoptosis (programmed cell death) [6, 9, 10].

6.3 Systemic Effects

Hepatic Disruption

The liver acts as a primary filtration site for foreign particles entering via the gastrointestinal tract [9, 10]. Systematic reviews of human liver cell lines confirm that MNPs cause severe **lipid metabolism disruption** [9, 10]. The accumulation of sub-micron plastics alters how cells process fats, leading to abnormal intracellular lipid accumulation, chronic hepatic inflammation, and cellular death, with nanoplastics driving the highest dose- and size-dependent toxicity [9].

Gastrointestinal and Digestive Outcome

Within the gut, persistent interaction with MNPs induces continuous low-grade inflammation and **intestinal barrier permeability** (disrupting tight junctions) [6, 10, 11]. This structural disruption is strongly associated with gastrointestinal immunosuppression—weakening the gut's localized immune defense—and severe dysbiosis, which alters the composition and metabolic functionality of the gut microbiome [10, 11].

Reproductive Systems and Development

MNPs serve as physical irritants and dynamic vectors carrying co-adsorbed environmental toxins and endocrine-disrupting chemicals [2, 6, 7]. Systematic evidence maps clear correlations between human exposure and compromised reproductive endpoints:

- **Male Reproductive Decline:** Studies strongly associate higher plastic body burdens with significantly reduced sperm quality and impaired testicular function [11].
- **Placental and Fetal Risks:** Microplastics have been repeatedly detected in human placental tissues [6, 10]. Their presence at this vital biological site triggers local immune dysregulation, which clinical models connect to intrauterine growth restriction (IUGR) and developmental outcomes like premature births [6, 11].

Cardiovascular and Respiratory Functions

Inhaled microfibres lodge deep within the pulmonary alveoli, causing localized mechanical lesions, chronic alveolar inflammation, and conditions like hypersensitivity pneumonitis [6, 10, 11]. From the lungs and gut, particles migrating into the bloodstream interact directly with blood components [6, 10]. This vascular presence alters normal coagulation cascades, encouraging platelet aggregation and accelerating the formation of thrombi (blood clots), which significantly elevates the risk of acute cardiovascular events [6, 10].

Organ / system	Reported effects	Reference(s)
Liver	Oxidative stress, inflammation, apoptosis, mitochondrial dysfunction, disturbed lipid metabolism	(9)
Gastrointestinal tract	Barrier disruption, immunosuppression, inflammation	(10,11)
Reproductive system	Reduced sperm quality (high evidence rating)	(11)
Respiratory system	Lung inflammation (moderate evidence rating)	(11)
Placenta / trans-generational	Documented placental transfer of MNP particles	(10)
Cellular / genetic level	Genotoxicity, cytotoxicity, apoptosis; nanoplastics highest risk (size- and dose-dependent)	(9,10)

Table 4. Summary of documented micro/nanoplastic effects on human organ systems.

7. Effects on Climate Change

A review of the nexus between microplastics and ecosystem/climate processes describes MPs as interacting with carbon and nitrogen biogeochemical cycles.It alters greenhouse gas flux dynamics in both marine and terrestrial systems, and influences the transport, degradation, and toxicity of the pollutant itself under changing climatic conditions (13). A complementary review of MP/NP dynamics on ecosystems and biogeochemical processes further characterises MNPs as bioaccumulating, globally distributed "novel entities" whose interaction with nutrient cycling, microbial community function, and carbon sequestration processes remains only partially quantified, and calls for their integration into climate-relevant regulatory frameworks (14).

8. Effects on Environment and ECOSYSTEMS

MNPs act across trophic levels simultaneously — from soil microbiota and invertebrates to plants, fish, birds, and mammals — their cumulative effect on biodiversity is broader than any single-species toxicology study can capture. Beyond direct biological effects, MNPs alter the physical and chemical properties of the environmental matrices they contaminate. In soil, this includes changes to bulk density, aggregate stability, and microbial community composition, with knock-on consequences for the broader ecosystem services (nutrient cycling, carbon storage, water regulation) that healthy soils provide (8,15). In aquatic sediments and the water column, MNPs alter turbidity, light penetration, and sediment physicochemical properties, with implications for primary productivity and benthic community health (12). Collectively, these findings support treating MNP contamination as a matrix-level environmental change, not solely a direct-toxicity issue for individual organisms (1,2,14).

9. Regulation and Mitigation

The reviewed literature converges on several priorities: source reduction at the point of release (textile microfibre filters, tyre and road-surface material innovation), upgraded wastewater and stormwater treatment, standardised analytical methods for MNP detection to improve cross-study comparability, expansion of direct human epidemiological research (most causal evidence to date derives from animal and in vitro models), and integration of MNP monitoring into climate and biodiversity regulatory frameworks (1,2,11,14).

10.CONCLUSION:

Micro- and nanoplastics (MNPs) constitute an interconnected One Health challenge, threatening ecosystems, animal health, food security, and human well-being. Material leaked from human activities cycles through air, water, soil, and food webs, ultimately returning to human systems. Research establishes that MNPs disrupt biological systems, impairing soil, plants, and biodiversity while causing oxidative stress, inflammation, metabolic imbalances, and organ dysfunction. Although detected in human placenta, blood, and brain tissues, uncertainties remain regarding long-term thresholds and chronic disease links. Resolving these gaps requires standardized analytics, epidemiological studies, and integrated toxicological research.

Addressing this complexity demands multidisciplinary collaboration among scientists, clinicians, engineers, and policymakers. Key mitigation steps include source reduction, advanced waste and wastewater management, sustainable materials development, and unified global regulations. Ultimately, MNPs underscore the links between environmental integrity and human health. A One Health framework offers a comprehensive approach to understanding these impacts and developing sustainable, evidence-based solutions.

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ONE HEALTH APPROACH TO PFAS—
Per- and Poly-Fluoroalkyl substances

A Systematic Review of Sources, Exposure Pathways, and 360° Impacts on Environment, Animal, Plant, Aquatic, and Human Health

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ABSTRACT:

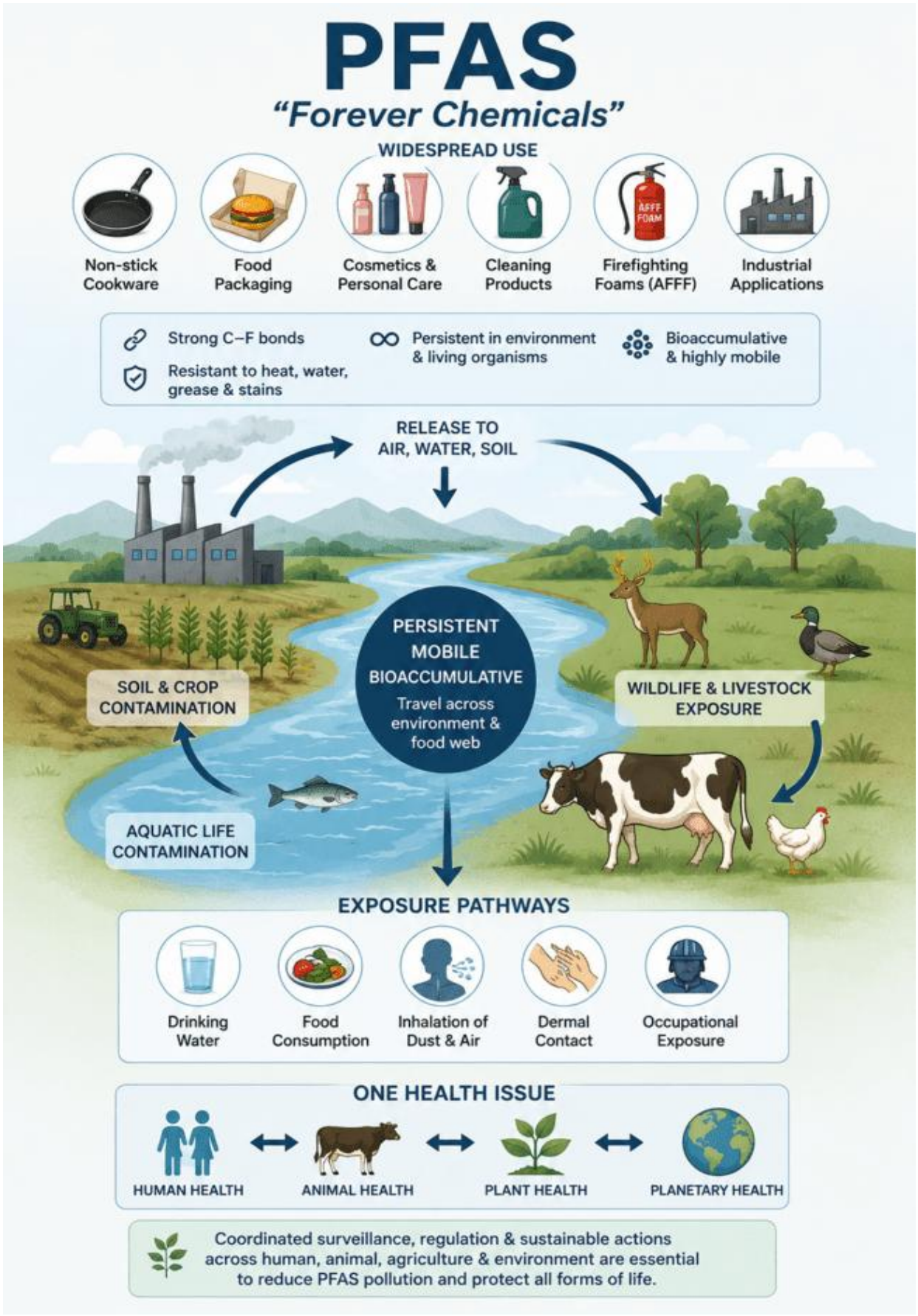
Per- and polyfluoroalkyl substances (PFAS) represent a vast and diverse class of anthropogenic organic compounds that includes perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorononanoic acid (PFNA), perfluorohexane sulfonic acid (PFHxS), and polytetrafluoroethylene (PTFE), among thousands of other synthetic chemicals. Owing to their unique surfactant properties and exceptional resistance to heat, water, grease, and stains, PFAS have been extensively manufactured and used across a wide range of commercial and industrial products over the past 70 years — from everyday consumer items such as non-stick cookware, fast-food wrappers, cosmetics, and cleaning products, to specialised industrial applications such as aqueous film-forming foams (AFFF) used in heavy-duty firefighting.

The strong, stable carbon-fluorine bonds that make PFAS commercially valuable also render them highly resistant to environmental degradation and biological metabolism. Because natural breakdown is negligible on human timescales, these compounds persist indefinitely and bioaccumulate in living organisms, earning the descriptive moniker "forever chemicals" due to their continuous threat to ecological and human health.

Driven by decades of extensive production, heavy usage, and high environmental mobility, PFAS have spread far past their initial release points. They are now routinely detected globally across diverse matrices, including drinking water, agricultural soil, vegetation, crops, wildlife, and human blood.This contamination fluidly cycles across soil, water, plants, animals, and humans through shared exposure pathways and a common food web.

For this reason, PFAS pollution is increasingly recognised as a One Health issue: a problem that cannot be adequately understood, monitored, or managed by focusing on human health, animal health, or environmental health in isolation. Wildlife and livestock act as sentinels for human contamination, while agricultural recycling of biosolids or irrigation water links soil, crop, and livestock impacts directly to the human diet. Additionally, the persistence driving bioaccumulation in fish and birds causes lifelong PFAS accumulation in humans. Effectively addressing this threat requires coordinated surveillance and policy across human medicine, veterinary science, agriculture, and environmental regulation, rather than siloed responses.
To address this widespread contamination, this systematic review synthesizes recent scientific literature to outline multi-systemic PFAS exposure pathways. It provides a comprehensive, One Health–informed overview of aggregate impacts on plant, animal, aquatic, human, and planetary health, while also examining global regulatory and mitigation measures.

GRAPHICAL ABSTRACT :



1. INTRODUCTION:

Per- and polyfluoroalkyl substances (PFAS) are defined as- one or more fully fluorinated carbon atoms, giving rise to the carbon-fluorine bond — the strongest and most stable bond in organic chemistry(1,4).theyareare a diverse group of more than 4,700 synthetic chemicals whose strong C-F bonding makes it commercially valuable, conferring thermal stability, water- and oil-repellency, and surfactant behaviour that has been exploited for over 70 years in non-stick cookware, water-resistant textiles, food packaging, firefighting foams, and countless industrial processes (1,3) It is also environmentally intractable: because natural biodegradation of the carbon-fluorine bond is negligible on human timescales, PFAS persist indefinitely in the environment and bioaccumulate in living tissue, earning them the label "forever chemicals" (1,4).

An influential 2022 analysis proposed that PFAS contamination now constitutes its own transgressed planetary boundary, comparable in concept to climate change or ozone depletion, on the grounds that PFAS levels in rainwater, soil, and surface water are above safety guideline values worldwide, and that this contamination is effectively irreversible at a human timescale (2). Reinforcing this concern, the European Chemicals Agency estimates that a further 4.4 million tonnes of PFAS could be released into the environment by 2053 without urgent restriction of their use (3). Once released, PFAS do not remain localised: they migrate readily through water, soil, and atmospheric pathways, and have been detected in environments with no direct industrial history, including Arctic snow and remote polar wildlife (2,4).

3. Sources of PFAS:

Unlike many other environmental pollutants, PFAS lack a single, globally recognized quantitative source-apportionment model. This absence of an equivalent global percentage breakdown stems from the immense diversity within the PFAS class—comprising thousands of distinct chemical compounds with unique usages. According to the reviewed literature, industrial manufacturing and processing facilities, alongside the deployment and training exercises associated with aqueous film-forming foams (AFFF) in firefighting, constitute the most significant and thoroughly documented major point sources of worldwide contamination. Further down the spectrum of environmental inputs, substantial contributions originate from municipal and commercial activities. These include the land application of biosolids and wastewater sludge, the generation of landfill leachate, and the overall life cycle of treated textiles, leather, and various consumer products. Lastly, direct everyday consumer usage—

encompassing cosmetics, ski wax, and car care items—functions as a minor, highly diffuse contributor to the total environmental burden of PFAS. This hierarchy of documented significance presents PFAS sources as a categorical ranking, which is summarized in Figure 1 and Table 1(1,2,3).

Source category	Relative significance	Notes	Reference(s)
Industrial manufacturing & processing facilities	Major, well-documented point source	Direct waste-stream discharge, air emissions, spills, waste disposal	(1,3)
Aqueous film-forming foam (AFFF) firefighting use	Major, well-documented point source	Airports, military bases, refineries; decades of use with long shelf life	(1,3,4)
Biosolids / wastewater sludge applied to land	Significant contributor	Conventional treatment does not remove PFAS; sludge reused as fertiliser	(1,3)
Landfill leachate	Significant contributor	PFAS-containing consumer products degrade over time in landfills	(1,3)
Textiles, leather & food packaging	Significant, diffuse contributor	Stain- and water-resistant coatings applied during manufacture	(1,3)
Everyday consumer use (cosmetics, ski wax, car care products)	Smaller, diffuse contributor	Individually minor but cumulative and difficult to control at source	(3)

Figure 1. Principal categories of PFAS release to the environment (qualitative ranking based on the reviewed literature; no single quantitative global source-apportionment model exists for PFAS)

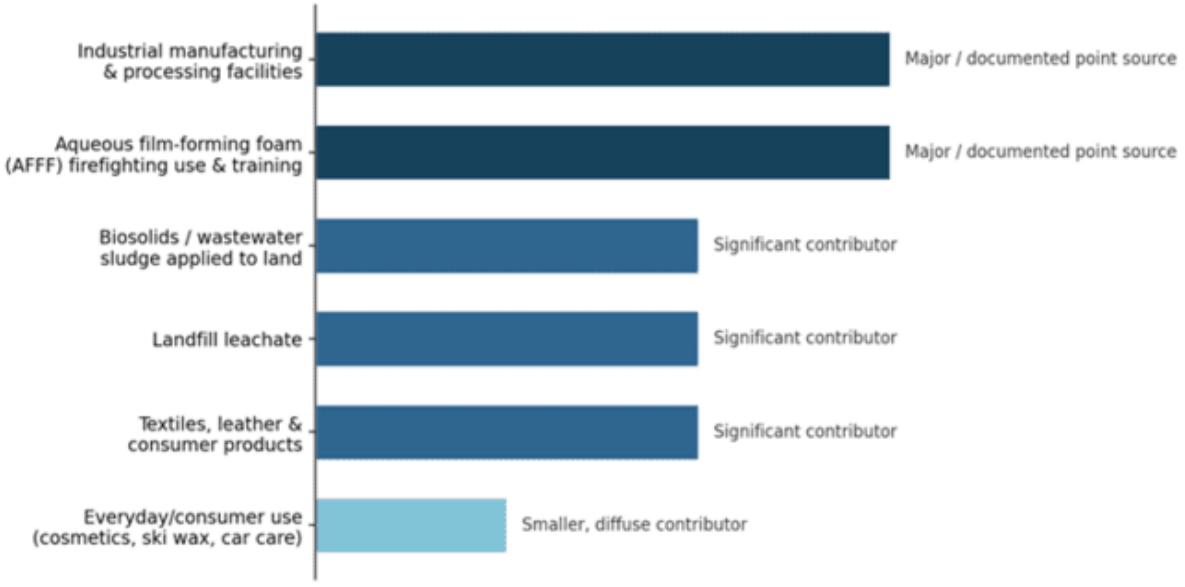
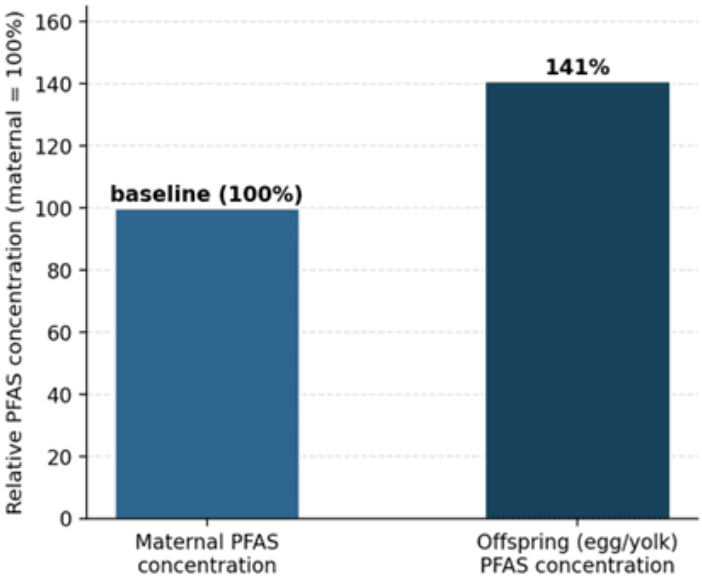


Figure 1. Principal categories of PFAS release to the environment, ranked qualitatively

4. Modes of Entry

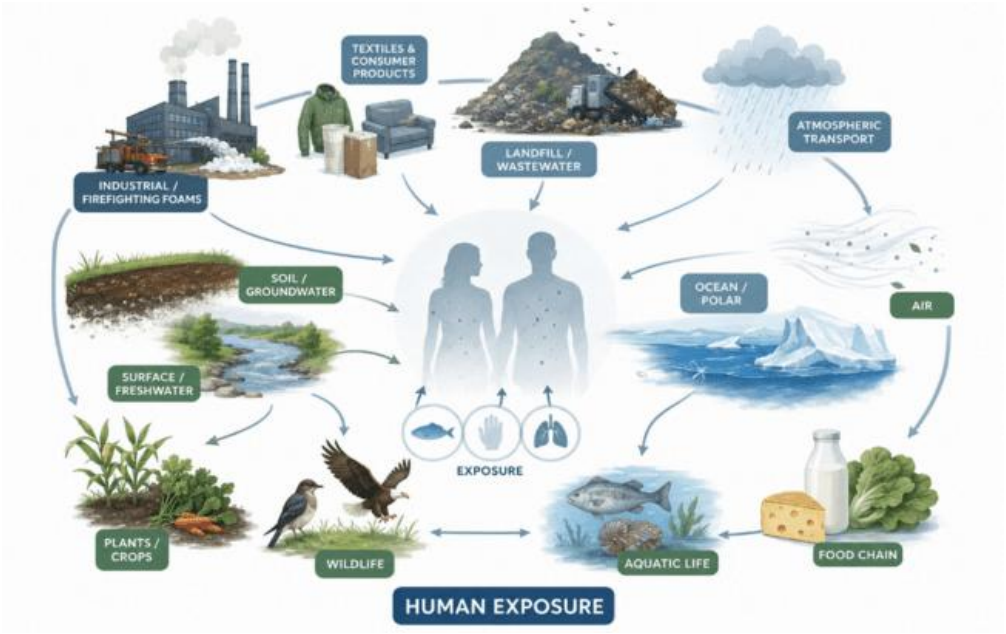
Since most PFAS are highly water-soluble and only weakly sorbed to soil particles, they migrate rapidly through groundwater and surface water once released, in contrast to more hydrophobic legacy pollutants that bind tightly to sediment (1,3,4). Exposure of biota occurs principally through ingestion (contaminated water, food, and prey), inhalation (occupational and airborne routes), dermal contact (treated textiles and consumer products), and — critically for wildlife and humans alike — maternal transfer to eggs, placenta, and offspring (4,5,7). Figure 2 shows the striking magnitude of this maternal-transfer route in birds, where a systematic review and meta-analysis of thirteen studies across sixteen species found PFAS concentrations 41% higher in offspring than in the mothers that transferred them (5).

Figure 2. Maternal-to-offspring PFAS transfer in wild birds (meta-analysis of 13 studies, 16 species, 25 compounds)



Source: systematic review and meta-analysis of maternal PFAS transfer in wild birds (ref. 5).

Figure 3 summarises the broader cascade from release source through environmental compartments to biota and human exposure.



Receptor	*Principal entry routes*	*Reference(s)*
Soil & plants	Root uptake and active transport, translocation to shoots/edible tissue	(8,9,10)
Soil & aquatic invertebrates	Direct ingestion of contaminated soil/sediment; bioaccumulation via exposure medium	(18)
Birds	Dietary ingestion (fish, prey), maternal transfer to eggs, inhalation	(5,6,7)
Aquatic organisms	Direct uptake from water, dietary ingestion, trophic transfer	(15,16,17)
Humans	Drinking water, food chain (fish, crops, dairy), occupational exposure, consumer products, placental transfer	(1,3,4,11–14)

5. Effects on Plant Health and Nutrition

PFAS enter agricultural systems primarily through PFAS-contaminated irrigation water, biosolids applied as fertiliser, and residual soil contamination near industrial or AFFF-impacted sites (8,9). A review of PFAS bioavailability, phytotoxicity, and plant uptake found that active root transport contributes to PFAS uptake, that short-chain PFAS show consistently higher accumulation in plant shoots than long-chain compounds, and that the degree of bioaccumulation depends on PFAS concentration, carbon-chain length, functional groups, plant species, and soil properties (8). A companion review compiling more than 4,500 soil-to-plant bioaccumulation factors across 45 PFAS compounds and 27 crop genera found that grasses (Poaceae) provided the largest evidence base, with the highest recorded bioaccumulation for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) (9).

At the physiological level, PFAS exposure induces over-generation of reactive oxygen species that damages plant cell structure and organelle function, and perturbs photosynthesis, gene expression,

protein synthesis, and carbon and nitrogen metabolism; plants respond by activating enzymatic and non-enzymatic antioxidant defences and metabolic reprogramming, though the concentration required to cause visible phenotypic damage is typically several orders of magnitude higher than environmentally realistic concentrations (9). A study using transcriptomic analysis of Arabidopsis thaliana exposed to next-generation GenX-family replacement compounds found that these substances are absorbed by roots, translocated to aerial tissues, and accumulate preferentially in reproductive organs (flowers and seeds), with bioaccumulation and translocation potential decreasing as chain length increases — a finding with direct relevance to food-chain transfer via edible seeds and grains (10).

Because several plant species — including cereals, fruit plants, and vegetables — accumulate PFAS in their edible parts, crop uptake represents a direct pathway for PFAS to enter the human food chain independent of direct water or industrial exposure, a food-safety concern that is compounded by the persistence of PFAS in soil long after the original contamination event (8,9,10).

6. Effects on WILDLIFE:

PFAS as an emerging animal health threat identifies liver damage, thyroid disease, obesity, fertility problems, and increased cancer risk among the multiple adverse effects documented across species, noting that because PFAS have been in widespread use for decades, their presence and impact are now essentially global in scope (4).

6.1 Birds

Birds — particularly seabirds occupying high trophic positions — are widely used as sentinel species for PFAS biomonitoring because of their long lifespans, well-characterised diets, and wide geographic distributions (6,7). Unlike lipophilic legacy pollutants, PFAS are proteinophilic, accumulating preferentially in protein-rich tissues such as liver and blood rather than fat, which shapes both their toxicokinetics and the tissues typically sampled in monitoring studies (7). A review of global seabird exposure found that maximum liver PFOS concentrations in species such as cormorants and fulmars from Europe and North America exceeded estimated toxicity reference values, with disruptions to the endocrine, immune, and metabolic systems documented across multiple species (6). A study of Arctic black-legged kittiwakes found a positive correlation between long-chain perfluoroalkyl carboxylate concentrations in blood and the percentage of abnormal spermatozoa, along with associations between specific PFAS compounds and the stress hormone corticosterone — evidence of both reproductive and endocrine disruption operating through distinct compound-specific pathways (7). As shown in Figure 2, the meta-analysis of maternal PFAS transfer in wild birds further found that longer, heavier PFAS compounds are preferentially transferred to eggs, that larger clutch size is associated with decreased per-egg transfer, and that species with piscivorous or opportunistic diets show higher transfer rates — variation with direct implications for modelling population-level risk (5).

6.2 Terrestrial mammals and other wildlife:

PFAS contamination and its associated health impacts have been documented across a striking range of wildlife, including hawksbill turtles, American alligators, hooded seals, striped bass, bottlenose dolphins, and freshwater turtles, with concentrations and severity of impact varying substantially by species and trophic position (4). The animal health threat review notes that PFAS toxicity was recognised by manufacturers early in the chemicals' commercial history — internal industry documentation from the 1960s already described liver, kidney, and spleen damage in exposed animals — underscoring that the ecological and health risks are not a recent discovery but a longstanding, still-unresolved concern (4).

- **Birds (seabirds & Arctic species):**Endocrine/immune/metabolic disruption; reduced sperm quality; corticosterone elevation; maternal-to-egg transfer (41% higher in offspring)(5,6,7)

- **Reptiles (turtles, alligators):** PFAS exposure in reptiles disrupts endocrine pathways and reproductive development, causing altered hormone levels, reduced hatching success, and immune dysfunction in vulnerable populations.(4)
- **Marine mammals (seals, dolphins):** Top predators accumulate elevated tissue concentrations of legacy compounds like PFOS, resulting in chronic immune system alteration, hepatotoxicity, and endocrine disruption.(4)
- **Fish (striped bass and others):** Bioaccumulation in fish species induces oxidative stress, organ pathology, metabolic dysfunction, and reduced reproductive fecundity, posing critical risks to aquatic life and secondary consumers.(4)

6.3 Aquatic Health:

A review of PFAS relevance to aquatic ecosystems reports that exposed fish and other aquatic species show reproductive toxicity, oxidative stress, metabolic disruption, immunological toxicity, developmental toxicity, and cellular damage, with in silico modelling showing that PFAS are incorporated into maturing fish oocytes during vitellogenesis by binding to vitellogenin and other yolk proteins, a mechanism directly linking PFAS exposure to reproductive impairment (15). The same review found that PFAS pollution alters extracellular polymeric substance production and chlorophyll content in aquatic biofilms, along with the diversity of biofilm-associated microorganisms, indicating effects that extend beyond individual organisms to the base of aquatic food webs (15).

A review of PFAS pollution, bioaccumulation, biomagnification, and toxicity across aquatic ecosystems found that PFOS and PFOA remain the most frequently detected compounds in most aquatic environments globally, though short-chain PFAS increasingly dominate in specific water bodies due to their higher water solubility, and confirmed that PFAS disrupt photosynthesis, growth, reproduction, oxidative balance, nervous system function, behaviour, digestion, metabolism, and immune function across the aquatic species studied (16). Because PFAS exhibit significant bioaccumulation potential in aquatic organisms, this raises direct food-safety concerns for human populations that rely on fish and seafood consumption (16). Species sensitivity also varies markedly: a comparative study across three amphibian species found that chronic PFAS exposure negatively affected body condition and development at concentrations as low as 10 µg/L, with species that have prolonged larval development (frogs and salamanders) proving more sensitive than more rapidly developing species (toads) — a finding that cautions against relying on single-species toxicity data for broader ecological risk assessment (17).

7. Effects on Human Health

Human exposure to PFAS occurs primarily through contaminated drinking water, food (particularly fish, dairy, and crops grown on contaminated soil), occupational contact at industrial, military, and firefighting sites, and consumer products, with leaching from contaminated sites into waterways making drinking water a substantial exposure route for many populations globally PFAS present in air, especially volatile or semi-volatile precursors, as well as PFAS bound to particulate matter, can enter the body via the respiratory system. Given that humans typically spend about 90% of their time indoors, and that indoor environments contain multiple PFAS emission sources, PFAS concentrations in indoor air and dust may be higher than outdoors, constituting a significant source of inhalation exposure (1,3,14).

7.1 Liver and metabolic effects

The liver is a major target organ for PFAS toxicity because many PFAS compounds accumulate preferentially in hepatic tissue and interact with key metabolic regulatory pathways (14). A systematic review of animal and epidemiological studies evaluating PFAS exposure and liver cancer risk found substantial experimental evidence that PFAS exposure induces liver injury across multiple model systems, including reduced hepatocyte viability, increased reactive oxygen species production, and activation of stress pathways linked to liver injury (11). A related review of PFAS exposure and liver

disease reports that large-scale population studies, including NHANES-based analyses covering more than 13,000 participants, have found significant associations between PFAS exposure and metabolic dysfunction-associated fatty liver disease, although the direction and strength of association vary by specific compound, sex, and body mass index subgroup (12).

7.2 Immune, endocrine, and reproductive effects

A systematic review and meta-analysis of childhood PFAS exposure and immunotoxicity, covering human epidemiological studies through 2023, examined the relationship between prenatal and childhood PFAS exposure and vaccine-induced antibody response, alongside the occurrence of common childhood infectious diseases, and found evidence that PFAS exposure diminishes immunisation efficiency in exposed children (13). A comprehensive review of PFAS toxicological effects and health impacts describes a multisystem, multitarget pattern of harm, with endocrine disruption, immune suppression, liver damage, reproductive toxicity, carcinogenic potential, and cardiovascular disease all documented, and notes that even modest regular fish consumption in contaminated areas can independently drive PFAS exposure levels orders of magnitude above current regulatory safety thresholds (14).

7.3 Carcinogenicity

In 2023, the International Agency for Research on Cancer classified PFOA as carcinogenic to humans (Group 1), reflecting the strength of accumulated epidemiological and mechanistic evidence (3). Consistent with this classification, studies have found associations between PFAS exposure and increased risk of several cancers, including testicular, kidney, pancreatic, breast, and liver cancer, with some sex-specific patterns observed — for example, PFOA associated with renal cell carcinoma among women and PFHxS with leukaemia among men in prospective cohort analysis (1,11)

7.4 Cardiovascular risk:

Growing epidemiological evidence correlates PFAS exposure with proven cardiovascular risk factors, particularly dyslipidemia. Data from numerous cohorts strongly indicate that exposure to different PFAS variants leads to increased levels of low-density lipoprotein cholesterol (LDL-C) and total cholesterol. The biological mechanisms tying these chemicals to cardiovascular diseases are complex and driven by likely synergistic effects. Key among these is metabolic disruption; PFAS contact shows strong correlations with hypertension, glucose metabolism disorders, and dyslipidemia, all of which are established promoters of atherosclerosis.

These metabolic changes can induce lipid accumulation within vascular structures, speeding up atherosclerotic plaque advancement. Additionally, PFAS exposure triggers endothelial dysfunction and persistent systemic inflammation, evidenced by elevated circulating levels of inflammatory biomarkers such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6); these factors combine to intensify vascular inflammation and accelerate the formation of atherosclerotic plaques.

Organ / system	Reported effects	Reference(s)
Liver	Hepatocellular injury, oxidative stress, apoptosis, fatty liver disease (MASLD/NAFLD)	(11,12,14)
Immune system	Reduced vaccine antibody response in children; immune suppression	(13,14)
Endocrine system	Thyroid hormone interference; disrupted steroidogenesis	(4,14)
Reproductive system	Reduced fertility; developmental delays in children	(1,3)

Cardiovascular system	Increased cardiovascular disease risk	(14)
Cancer risk (multiple sites)	Testicular, kidney, pancreatic, breast, and liver cancer; PFOA classified IARC Group 1 carcinogen (2023)	(1,3,11)

8. Effects on Climate Change and Planetary Boundaries

PFAS contamination is now formally proposed as its own planetary boundary, alongside established boundaries such as climate change and biosphere integrity, on the basis that PFAS levels in rainwater, soil, and surface water are ubiquitously above safety guideline values across the globe, and that the associated environmental and biological effects are effectively irreversible once PFAS are released (2). Unlike the climate change boundary, which concerns the release of specific gases with well-quantified atmospheric effects, the PFAS boundary reflects the cumulative, poorly reversible risk associated even with low-level chronic exposure across an entire chemical class comprising thousands of related compounds (2).

The broader planetary boundaries framework classifies PFAS within the "novel entities" boundary — new substances with the potential for unwanted geophysical and/or biological effects on the Earth system — a category that, together with the crossed boundaries for climate change and biosphere integrity, indicates that humanity now operates outside the safe operating space across multiple interacting Earth-system processes simultaneously (2). This interaction matters because environmental conditions altered by climate change — including changing precipitation patterns, temperature, and ocean circulation — can influence the transport, deposition, and bioavailability of persistent pollutants such as PFAS, meaning the two boundary transgressions are not independent of one another (2).

9. Effects on Biodiversity and Ecosystem Services

PFAS contamination has been documented across an unusually broad span of the tree of life — from soil and aquatic invertebrates, through amphibians, fish, and birds, to apex predators including marine mammals and reptiles — with the wildlife health impacts described as severe and occurring "the world over" rather than being confined to heavily industrialised regions (4,17,18). A review of PFAS and their substitute compounds in terrestrial and aquatic invertebrates found that these organisms — which occupy foundational positions in both soil and aquatic food webs — accumulate and are harmed by PFAS exposure, with documented effects on invertebrate neurotoxicity and reproductive and developmental toxicity that have direct implications for the food webs that depend on invertebrate prey (18).

Because bioaccumulation and biomagnification concentrate PFAS at higher trophic levels, species sitting at the top of food chains — raptors, marine mammals, and apex fish predators — face disproportionate exposure and risk, a pattern consistent with the biomagnification dynamics documented for other persistent pollutants (4,16). The cross-species breadth of documented harm, combined with the species-specific sensitivity differences observed even among closely related taxa such as amphibians, underscores that PFAS represents a biodiversity-relevant stressor whose ecological risk cannot be adequately captured by single-species or single-ecosystem assessment alone (17,18).

10. Regulation and Mitigation :

Per- and polyfluoroalkyl substances (PFAS) provide significant commercial utility, yet their exceptional durability in the environment, potential to bioaccumulate, and widespread ecotoxicity have raised serious global concerns. A major obstacle in managing these chemicals is the fragmented

state of international oversight, which is defined by inconsistent regulatory frameworks and varying approaches across different jurisdictions (19).

Prominent regulatory bodies in the United States, the European Union, and Australia have increasingly transitioned toward stricter mandates, such as implementing categorical bans on certain compounds, introducing restrictive drinking water standards, and establishing comprehensive surveillance and reporting frameworks. This worldwide regulatory shift intensified significantly in 2023. By April 2024, the US Environmental Protection Agency established its first legally enforceable national drinking water standards, setting maximum contaminant levels at 4 ng/L for PFOA and PFOS, and 10 ng/L for PFNA, PFHxS, and the GenX-family replacement HFPO-DA, as shown in Figure 4 (1,3). These stringent legal limits reflect a growing scientific consensus on the necessity of integrated mitigation strategies to protect public health and ecological safety. However, the regulatory landscape remains complex and unstable; the absence of aligned safety thresholds and cleanup enforcement across neighboring regions continues to disrupt international trade and hinder coordinated environmental protection initiatives (19).

Existing literature emphasizes several critical priorities moving forward: the ongoing phase-out and replacement of long-chain PFAS in consumer goods and Aqueous Film Forming Foam (AFFF) firefighting agents; the advancement of water treatment technologies capable of removing PFAS, which traditional chemical and biological methods fail to achieve; the remediation of contaminated groundwater and soil at AFFF-impacted and industrial sites; the expansion of biomonitoring programs using wildlife sentinel species, like seabirds, to monitor long-term trends; and a transition toward class-based regulation rather than a compound-by-compound approach, given the immense size and diversity of the PFAS class (1,3,4,6). Furthermore, because diseases related to PFAS exposure result in an estimated annual economic burden of US$5.5–62 billion in the United States alone, the financial argument for preventative regulation strongly reinforces the public health justification (1).

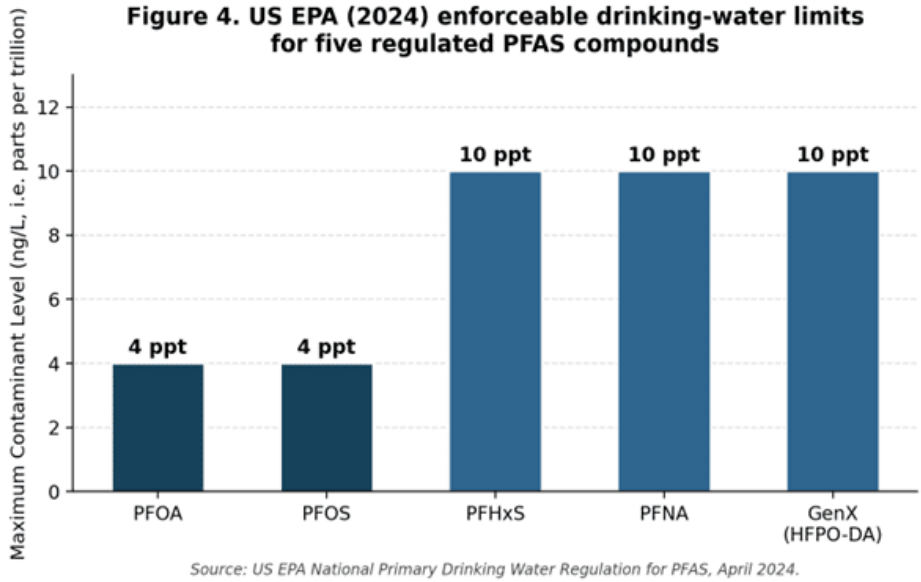


Figure 4. US EPA (2024) enforceable drinking-water limits for five regulated PFAS compounds (1,3).

11. Conclusion:

PFAS emerge as a genuinely cross-cutting environmental pollutant whose documented effects span soil and water chemistry, plant physiology, invertebrate and vertebrate toxicology, avian reproductive biology, aquatic ecosystem function, human organ-system health, and — uniquely among the pollutants commonly reviewed in this way — a proposed transgression of a dedicated planetary boundary. Industrial manufacturing, AFFF firefighting foam, and diffuse consumer-product use are consistently identified as the dominant release categories, even though the field currently lacks the

kind of quantitative global source-apportionment model available for pollutants such as microplastics. The persistence, environmental mobility, and cross-taxa effects of PFAS mean that risk reduction will require coordinated action across manufacturing, firefighting practice, wastewater and biosolids management, agricultural practice, and international regulatory policy, building on the momentum of the 2024 US drinking water standards while extending equivalent protection to the many jurisdictions that have not yet acted.

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ONE HEALTH APPROACH TO ANTIBIOTIC RESIDUES

A Systematic Review of Sources, Exposure Pathways, and 360° Impacts on Environment, Animal, Plant, Aquatic, and Human Health

TABLE OF CONTENTS

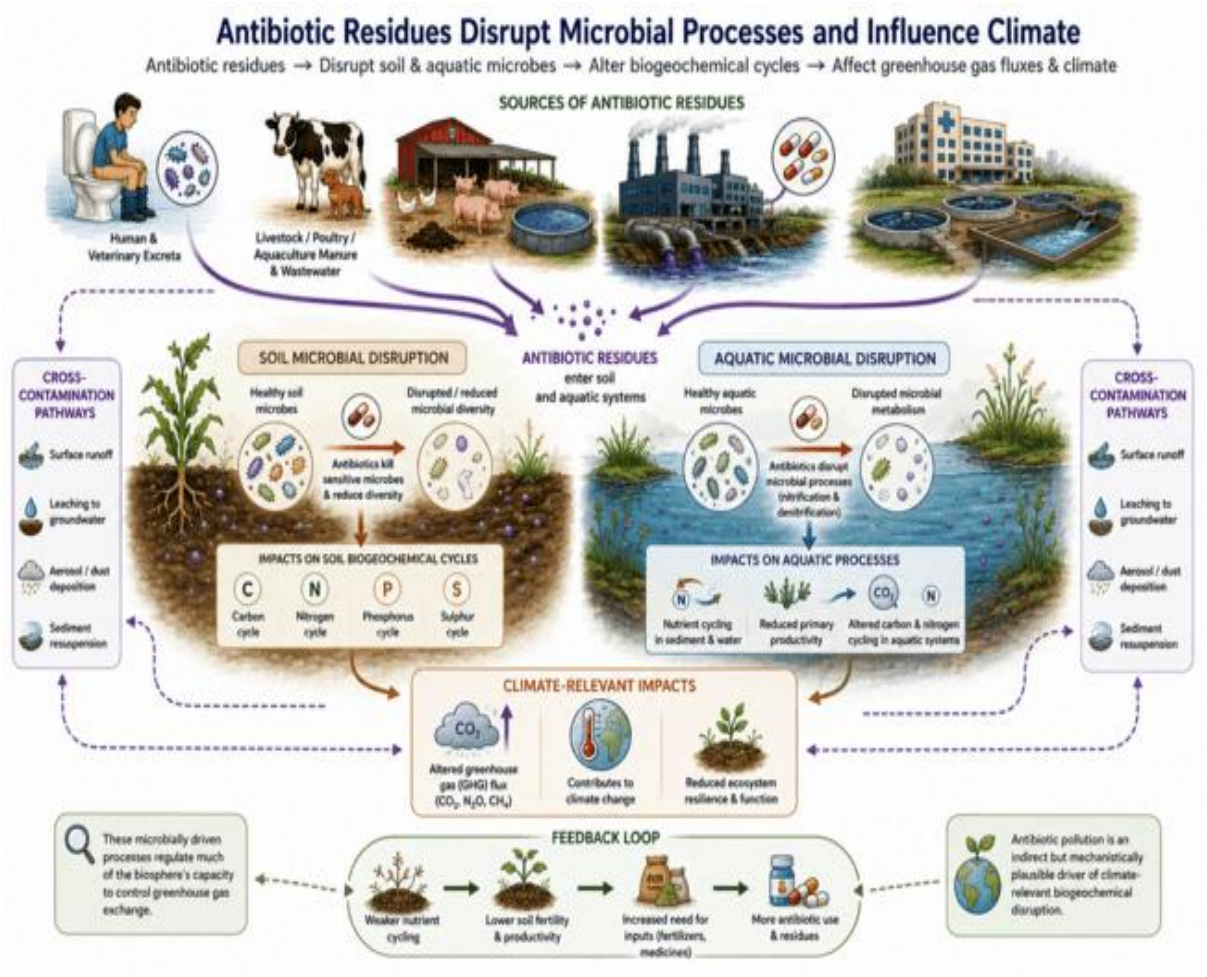
ABSTRACT

Antibiotic residues have emerged as an important environmental contaminant that extends the challenge of antimicrobial resistance (AMR) beyond clinical settings into natural ecosystems. Large proportions of administered antibiotics are excreted unchanged or as active metabolites from humans and animals, while additional environmental inputs arise from pharmaceutical manufacturing, livestock production, aquaculture, hospital effluents, municipal wastewater, and agricultural practices. These residues persist in soil, surface water, sediments, and groundwater, where they exert selective pressure on microbial communities, promote the dissemination of antibiotic-resistant bacteria (ARB) and antibiotic resistance genes (ARGs), and circulate continuously through interconnected environmental compartments.(1,2,3)

This review synthesizes recent evidence (2023–2026) using a One Health perspective to examine the major sources, environmental pathways, and multidimensional impacts of antibiotic residues on humans, animals, plants, wildlife, aquatic ecosystems, biodiversity, and climate-relevant processes. Evidence indicates that antibiotic residues impair soil and aquatic microbial communities responsible

for nutrient cycling, alter plant growth and crop quality through phytotoxicity and root uptake, accumulate throughout food chains, contribute to wildlife dissemination of resistant organisms, disrupt aquatic ecosystem functioning, and indirectly influence greenhouse gas regulation through altered biogeochemical cycling. Human exposure occurs through contaminated food, drinking water, environmental contact, and transfer of resistance determinants, contributing to gut microbiome dysbiosis, toxicological effects, and the escalating global burden of antimicrobial resistance.(6) Collectively, the evidence demonstrates that antibiotic residues should be regarded not only as pharmaceutical pollutants but also as ecosystem stressors with far-reaching One Health consequences. Strengthening wastewater treatment, improving antimicrobial stewardship, enforcing veterinary withdrawal periods, expanding environmental surveillance, and integrating environmental considerations into AMR policies are essential to limit the continued dissemination of antibiotic residues and preserve ecosystem integrity and public health.

GRAPHICAL ABSTRACT:



1. INTRODUCTION:

The World Health Organization (WHO) defines antibiotics as medicines used to prevent and treat bacterial infections. Antibiotic residue refers to the trace amounts of antibiotic drugs, theirdegradation products, or metabolites that remain in animal-derived food products (such as meat, milk, and eggs) after the animal has been treated with medication.

They are among the most consequential pharmaceutical interventions of the twentieth century, yet the same biological activity that makes them effective against pathogenic bacteria makes their

environmental presence uniquely hazardous. A fraction of any administered dose - commonly up to 90% in veterinary use - is excreted from the treated organism unchanged or as active metabolites, entering wastewater, manure, and soil largely intact (1,2). Once released, antibiotic residues persist across environmental media at highly variable concentrations: reported ranges span roughly 1 µg/kg to 100 mg/kg in animal faeces and 1 ng/L to 10 µg/L in wastewater, with tetracyclines typically the most prevalent class detected and macrolides typically the least (1).

Unlike most chemical pollutants, antibiotic residues pose a hazard less through direct toxicity and more by exerting continuous selective pressure on bacterial communities. This drives the emergence and spread of antibiotic-resistant bacteria (ARB) and antibiotic resistance genes (ARGs) via horizontal gene transfer mediated by mobile genetic elements like plasmids, transposons, and integrons. This links chemical pollution directly to a global health crisis: global antibiotic usage rose by 20.9% between 2016 and 2023, and is projected to increase by 52.3% relative to 2016 levels by 2030 without intervention, accelerating future AMR emergence. Consequently, "antibiotic pollution" encompasses both the drug residues and the resulting resistant bacteria and genetic traits.(1,2)

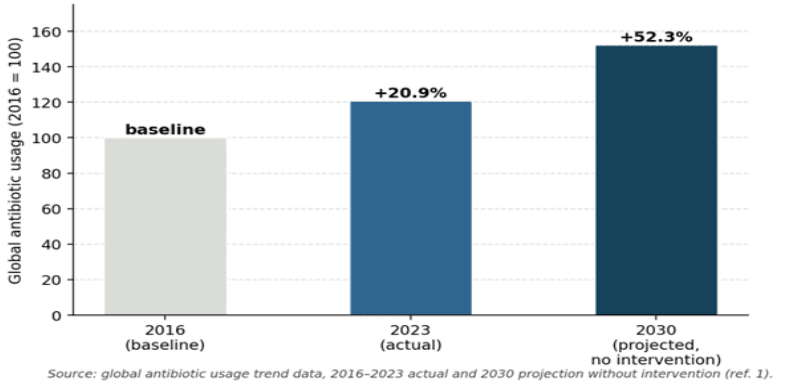
2. METHODOLOGY

Literature was retrieved from PubMed/PMC and Scopus-indexed journals (ScienceDirect, Springer Nature, MDPI, Frontiers, ASCE Library) using combinations of the terms "antibiotic residues," "antimicrobial resistance," "antibiotic resistance genes," "source," "exposure," "birds," "wildlife," "plant uptake," "human health," "aquatic," "biodiversity," and "soil," restricted to free/open-access articles published between 2023 and 2026. From the results, some specific articles were selected to give the broadest possible topical coverage — comprehensive reviews for background and source data, and focused reviews for each specific health or ecological domain.

3. Sources of Antibiotic Residue Contamination

Antibiotic residues enter the environment from four principal categories of source: human healthcare (hospital and municipal wastewater), veterinary and agricultural use (livestock, poultry, and aquaculture manure and wastewater), pharmaceutical manufacturing effluent, and — in many low- and middle-income countries — inadequately treated sewage discharged directly into surface water (1,3,4,5). Figure 1 shows the trajectory that is common to nearly all of these sources: global antibiotic consumption itself, which has risen sharply since 2016 and is projected to accelerate further without intervention.

Figure 1. Global antibiotic usage trend, 2016–2030 (index, 2016 = 100)



Source: global antibiotic usage trend data, 2016–2023 actual and 2030 projection without intervention (ref. 1).

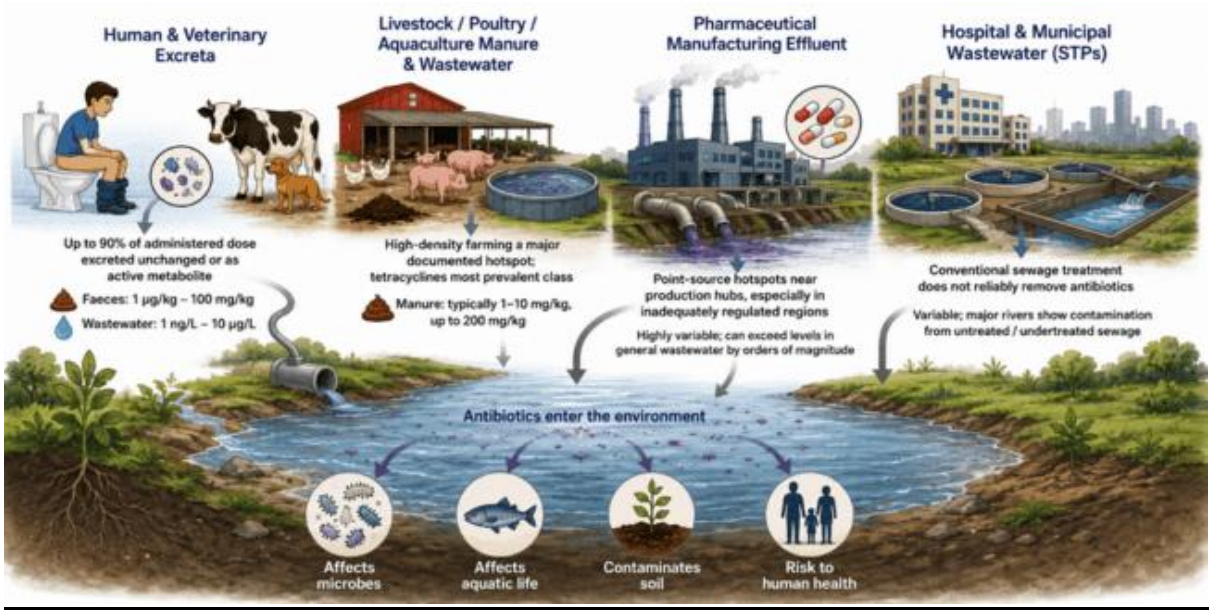
A case study of a North Indian pharmaceutical manufacturing hub illustrates how inadequately treated pharmaceutical, industrial, domestic, and hospital wastes converge into rivers, creating persistent antibiotic hotspots where ciprofloxacin concentrations select for resistance(5).Similarly, high-density

livestock and poultry farming operations represent a distinct source category, with antibiotic concentrations in animal faeces and wastewater varying by orders of magnitude depending on farming intensity, regulatory context, and drug class.(1)

4. Modes of Entry

Antibiotic residues move between soil, water, and sediment compartments primarily via wastewater discharge, agricultural runoff, manure and biosolid application to land, and irrigation with reclaimed wastewater (1,2,9,10). Since most of the conventional wastewater treatment processes are not designed to remove pharmaceutically active compounds, treatment plants act as a persistent point source rather than an effective barrier (1,4). Biological uptake occurs through direct ingestion (via contaminated water, food, and prey), root uptake and translocation occurs in plants, and for the AMR dimension of these antibitc residues, horizontal gene transfer between environmental bacteria and clinically relevant pathogens occurs (2,9). Figure 3 summarises this cascade from release source through environmental compartments

Receptor	Principal entry routes	Reference(s)
Soil & plants	Root uptake and translocation from manure-, biosolid-, or wastewater-irrigated soil	(9,10,11,12)
Soil microbiota	Direct exposure via manure/biosolid application; selective pressure on microbial communities	(11)
Birds & wildlife	Ingestion of contaminated prey/water; environmental exposure to ARB	(6,7)
Aquatic organisms	Direct uptake from contaminated water, dietary ingestion, bioaccumulation in tissue	(16,17,18)
Humans	Food chain (meat, dairy, fish, crops), drinking water, direct healthcare exposure, horizontal gene transfer of resistance to human pathogens	(1,2,8,13,14,15)



Schematic overview of sources, environmental compartments, and principal modes of entry

5. Effects on Wildlife:

Livestock and poultry represent both a major source of antibiotic residues and a directly affected population. A review of antibiotic residues in poultry food products found that the concurrent presence of multiple residues within the same samples reflects a lack of veterinary guidance and poor withdrawal-period management, increasing the risk of resistance transfer through the food chain and raising the potential for cumulative toxicological effects in both the animals themselves and downstream consumers (8).

5.1 Birds

Wild birds have emerged as an important, historically overlooked sentinel population for environmental AMR. A systematic review examining antimicrobial-resistant Escherichia coli in wild birds found substantially higher resistance prevalence in birds from low- and middle-income countries compared with high-income countries, and — most strikingly — found resistance to WHO Critically Important Antimicrobials at levels of 100% for cefotaxime, ceftazidime, nalidixic acid, and gentamicin, and over 90% for ciprofloxacin, among the resistant isolates studied (6). The same review notes that wildlife and the environment are frequently overlooked in AMR health security strategies that focus primarily on humans and domestic animals, despite wild birds' capacity for long-distance migration and their role in disseminating resistant bacteria across regions and continents (6).

Similarly, wildlife rehabilitation centres present a direct clinical dimension of this problem: a study of antimicrobial-resistant bacteria isolated from wounds in wild birds admitted for rehabilitation found resistant isolates complicating treatment of injuries such as osteomyelitis, underscoring that AMR does not remain confined to agricultural or clinical settings but directly compromises wildlife care itself (7).

Taxon	Documented effects	Reference(s)
Wild birds (general)	Reservoir and disseminator of antimicrobial-resistant E. coli; up to 100% resistance to WHO Critically Important Antimicrobials in some populations	(6)
Wild birds (clinical/rehabilitation)	Antimicrobial-resistant wound and fracture infections complicating veterinary treatment	(7)

Poultry & livestock	Residue accumulation in tissue and eggs; multiple concurrent residues from poor withdrawal-period compliance	(8)
Migratory birds	Documented carriers of resistant E. coli and virulence genes across international flyways	(6)

Table 3. Summary of documented antibiotic-residue and AMR-related effects across animal taxa.

5.2 ANIMALS:

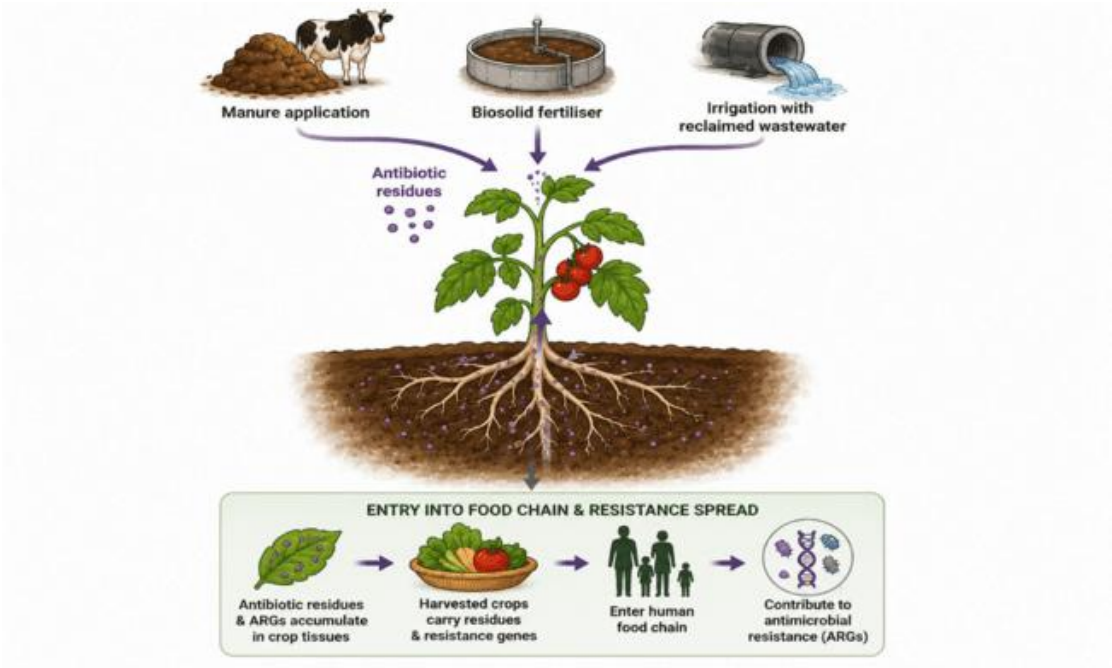
The widespread incorporation of antimicrobials in animal farming frequently leads to the detection of antibiotic residues within global food chains, particularly in products like milk, eggs, and meat. These persistent residues, which regularly surpass established maximum residual limits (MRLs), typically stem from improper drug administration, non-compliance with mandatory withdrawal periods, or off-label applications. The presence of these substances—including tetracyclines, β-lactams, fluoroquinolones, macrolides, sulfonamides, and aminoglycosides—poses significant health risks. These include the transmission of antibiotic-resistant strains to human populations, immunopathological reactions, hypersensitivity, mutagenic risks, nephrotoxicity (such as from gentamicin), liver toxicity, reproductive issues, bone marrow suppression (linked to chloramphenicol), and potential carcinogenic effects associated with compounds like sulphamethazine, oxytetracycline, and furazolidone. Among various livestock sectors, poultry farming—especially broiler chicken production—demonstrates the most pronounced diversity and frequency of contamination. Research conducted in nations such as Egypt and Bangladesh has identified substantial accumulations of chloramphenicol, tylosin, ciprofloxacin, and oxytetracycline in critical tissues like the liver, kidneys, and muscles, highlighting an extensive dependency on fluoroquinolone, macrolide, and tetracycline drug classes in modern husbandry practices.(19,20)

6. Effects on Plant Health and Nutrition

Antibiotic residues reach agricultural soils primarily through manure application, biosolid fertiliser, and irrigation with reclaimed wastewater, and are subsequently taken up by crops via root systems in a process governed by the antibiotic's ionic partitioning and physicochemical properties, soil organic carbon content and pH, and the plant's own transpiration rate (9,10,12). A meta-analysis of antibiotic uptake from soil and translocation in plants found that tetracyclines show higher uptake into plant leaves than sulfonamides, and that the two classes also differ in internal translocation pattern — tetracyclines distribute relatively evenly between root and leaf tissue, whereas sulfonamides tend to accumulate preferentially in roots — differences attributable to tetracyclines' higher water solubility and uncharged speciation across typical agricultural soil pH ranges (12).

At the physiological level, antibiotics can be directly phytotoxic to crops, damaging photosynthetic processes, reducing nutrient uptake, and inducing oxidative stress, with downstream effects including delayed germination, reduced crop biomass, and lower crop quality through reduced nitrogen uptake — though a review of soil microbial and plant responses to antibiotic exposure notes that such effects are more commonly observed at concentrations well above those typically found in natural soil environments (7,9). Antibiotics also reshape the soil microbial community in ways that indirectly affect plant health: exposure can suppress plant growth-promoting microorganisms such as Streptomyces species that stimulate crop growth, while simultaneously increasing the relative abundance of microbial genera associated with poorer soil quality (11). A comprehensive review found that residue accumulation in manure and soil varies seasonally — higher in winter than summer - reflecting reduced microbial degradation activity and drug migration capacity under colder conditions (10).

Because antibiotic-resistant bacteria and resistance genes can also accumulate directly within crop tissue at harvest under continued selective pressure from soil-residue exposure, plant uptake represents not only a phytotoxicity concern but also a direct route by which resistance determinants along with the parent antibiotic - enter the human food chain (9).



7. Effects on Human Health

Human exposure to antibiotic residues occurs through the food chain (meat, dairy, fish, and crops grown on contaminated soil or irrigated with contaminated water), drinking water, and direct environmental contact, while a parallel and arguably more consequential exposure pathway operates through horizontal transfer of resistance genes from environmental bacteria to human pathogens (1,2,13).

7.1 Gut microbiome disruption

A review of the potential impact of antibiotic exposure on the microbiome and human health describes two major, interlinked challenges: antibiotic-induced changes to the microbiome can disrupt immune function and increase susceptibility to a range of diseases, while prolonged or repeated exposure independently fosters the proliferation of antibiotic resistance genes within the gut resistome, favouring the emergence of resistant strains (13). Because antibiotic molecular targets — cell walls, ribosomes, and RNA polymerases — are not unique to pathogenic bacteria, even therapeutically appropriate antibiotic use indiscriminately affects beneficial commensal bacteria alongside disease-causing organisms, disrupting the balance of numerous host metabolic processes that depend on a stable microbiome (13). A review of the complexity of antibiotic resistance and its impact on gut microbiota dynamics further highlights that microbial diversity, gut metabolism, and inflammatory responses collectively shape how resistance mechanisms develop and persist following antibiotic-induced dysbiosis, and identifies secondary bacterial metabolites as potential early biomarkers for detecting emerging resistance (14).

7.2 AMR mortality

The clearest evidence of the human health stakes involved comes from global mortality data. According to the Global Research on Antimicrobial Resistance (GRAM) project, bacterial AMR was directly responsible for approximately 1.27 million deaths and was associated with 4.95 million deaths globally in 2019; updated Global Burden of Disease estimates for 2021 found 1.14 million deaths directly attributable to AMR and 4.71 million deaths associated with resistant infections that

year, figures summarised in Figure 2 (15). A review of the wider environmental drivers of this burden notes that in the United States alone, antibiotic-resistant bacteria are estimated to cause between 23,000 and 35,000 deaths annually, underscoring that this is not solely a problem of low-resource settings but a truly global health security threat (3).

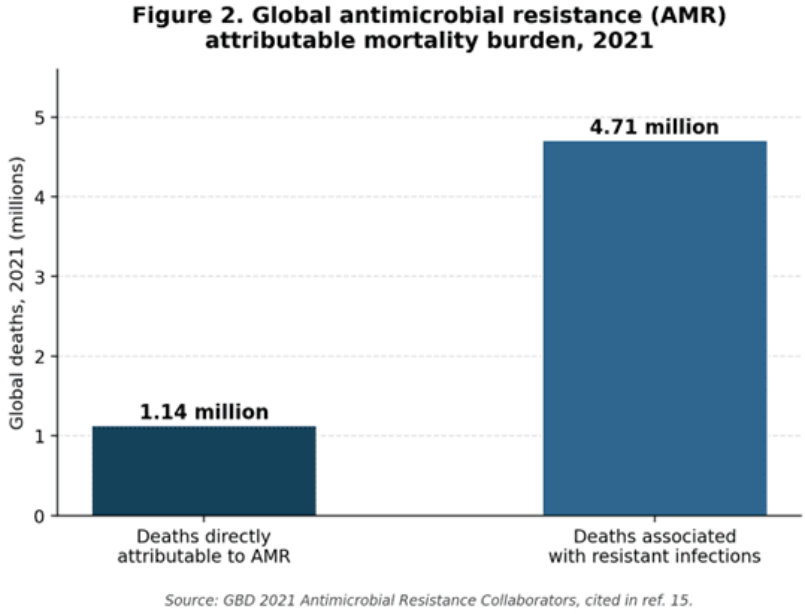


Figure 2. Global antimicrobial resistance (AMR)-attributable mortality burden, 2021

7.3 Direct toxicological effects of residue exposure

Beyond the AMR pathway, direct toxicological effects of antibiotic residue exposure have also been documented experimentally. A study of long-term amoxicillin and ciprofloxacin residue exposure at levels above maximum residue limits found significantly increased body weight in exposed mice, with the most pronounced effects observed after three months of continuous exposure, alongside documented effects on growth, immunity, and organ function — findings the authors argue highlight the need for more tightly controlled antibiotic use across both clinical and animal-health sectors to prevent potential downstream hazards to human consumers (2).

Organ / system	Reported effects	Reference(s)
Gut microbiome	Reduced microbial diversity, dysbiosis, disrupted immune function, resistome enrichment	(13,14)
Immune system	Increased disease susceptibility associated with microbiome disruption	(13)
Metabolic system	Increased body weight and altered metabolic processes in chronic low-dose exposure models	(2)
Organ function (liver, other organs)	Documented effects on organ function in long-term residue exposure studies	(2)
Population-level mortality (via AMR)	1.14 million deaths directly attributable to AMR; 4.71 million associated deaths globally (2021)	(15)

Table 4. Summary of documented antibiotic-residue and AMR-related effects on human health.

8. Effects on Aquatic Health

Aquatic ecosystems receive antibiotic residues from aquaculture, agricultural runoff, and wastewater discharge, and a review of antibiotic pollution in aquatic ecosystems found toxic effects across multiple trophic levels — vertebrates, invertebrates, algae, and diatoms — with accumulation, persistence, and disruption of biological processes documented across the species studied (16). Species sensitivity varies substantially by compound and life stage: controlled toxicity testing across Daphnia magna, Artemia salina, a marine diatom, and a freshwater duckweed species found that ciprofloxacin and sulfamethoxazole produced markedly different effect concentrations depending on species and developmental stage — for example, Daphnia magna embryos were more sensitive than juveniles to sulfamethoxazole, while the reverse pattern held for ciprofloxacin — underscoring that single-species toxicity data cannot be safely extrapolated across aquatic communities (17).

The consequences of antibiotic residues at sub-lethal, environmentally realistic concentrations extend significantly beyond acute toxicity. A review focusing on aquatic ecosystems highlights that chronic exposure triggers genotoxic and cellular stress in algae, fish, and invertebrates, while also disrupting vital microbial metabolic processes—such as nitrification and denitrification—that drive nutrient cycling in water and sediment. Additionally, these residues can interact synergistically with other environmental pollutants, including heavy metals, to compound total ecosystem toxicity. The population-level implications of such trace exposures are clearly illustrated in a behavioral study of guppies, where exposed males demonstrated reduced body condition, decreased sperm velocity, and altered secondary sexual characteristics. The accompanying reduction in behavioral variability could critically compromise their mating success and natural adaptive capacity. Reflecting the massive scale of this ecological and economic dilemma, a risk assessment highlights a World Bank estimate that antibiotic residues and resistance collectively cause a 1.1–3.8% drop in global gross domestic product—a profound figure that reframes this class of pollutants from a strictly toxicological issue to a major macroeconomic concern.(16,18)

9. Effects on Air quality

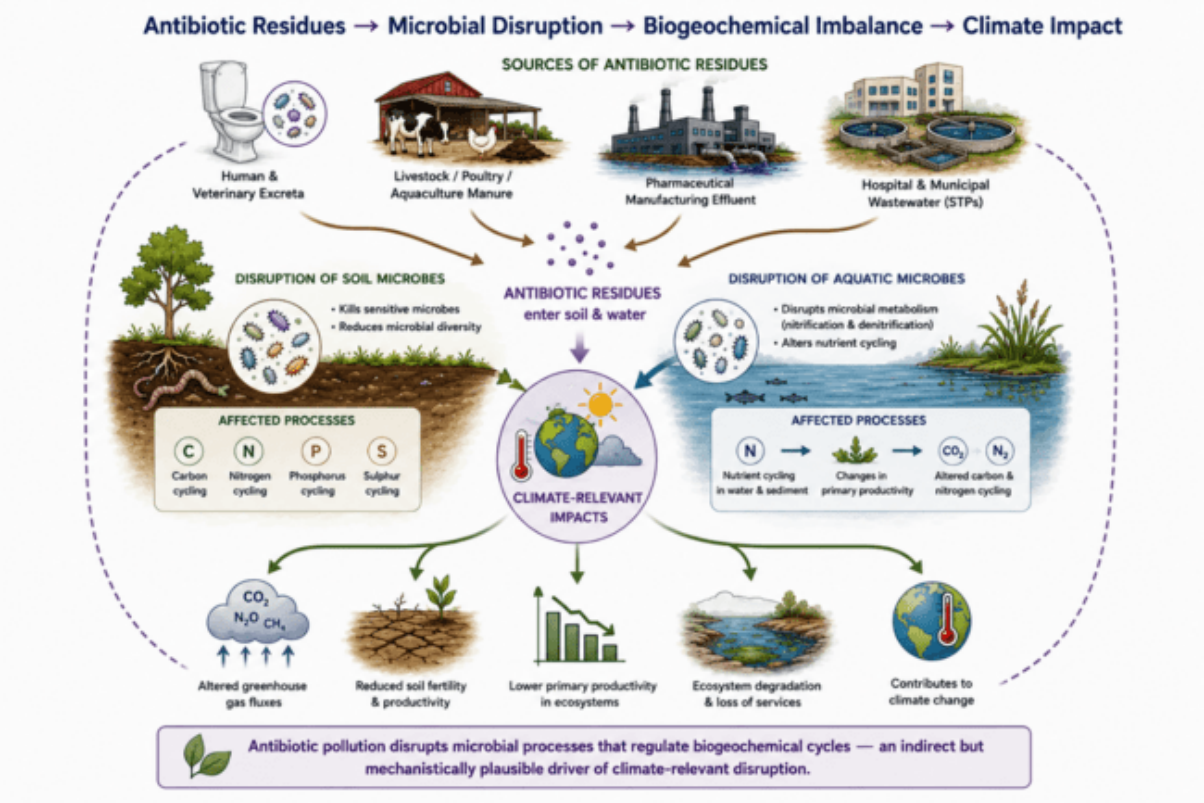
Although environmental antibiotic research has traditionally centered on soil and water, the atmosphere serves as a critical but neglected vector for the transport of antibiotic residues and antibiotic resistance genes (ARGs) via total suspended particles and fine particulate matter (PM2.5). These active residues and resistant bacteria are directly aerosolized into the air from industrial agricultural operations, such as poultry farms and cattle feedyards, as well as wastewater treatment plants and hospital emissions.

When inhaled, PM2.5 bound with these active residues and ARGs penetrates deep into the alveolar zones of human and animal lungs before crossing directly into the bloodstream. This process drives localized microbial resistance, disrupts nasal and intestinal microbiomes, and hastens the global spread of superbugs. Furthermore, atmospheric currents can carry these bioaerosols over long distances stretching up to several kilometers downwind, effectively connecting rural agricultural hotspots with urban centers.(21)

10.Effects on Climate Change

Antibiotic residues affect climate-relevant processes primarily through their disruption of soil and aquatic microbial communities responsible for biogeochemical cycling. Soil microorganisms are essential to the decomposition of organic matter and the transformation of nitrogen, phosphorus, sulphur, and carbon; because antibiotic residues can kill sensitive microbial populations and reduce microbial diversity, their presence in soil has direct consequences for these nutrient cycling processes, with corresponding implications for greenhouse gas flux from agricultural soils (3). In aquatic systems, antibiotic-driven disruption of microbial metabolism — including nitrification and denitrification — alters nutrient cycling in sediment and the water column, with downstream effects on primary productivity and, potentially, on the aquatic system's role in carbon and nitrogen cycling more broadly (16). Because these microbial processes underpin much of the biosphere's capacity to

regulate greenhouse gas exchange, antibiotic pollution represents an indirect but mechanistically plausible contributor to climate-relevant biogeochemical disruption, an area that — unlike the AMR mortality burden — remains comparatively underquantified in the current literature (3,16).



11.Effects on Biodiversity and Ecosystem

Wild birds illustrate how AMR itself has become a biodiversity-relevant phenomenon rather than a purely clinical or agricultural one: because these species range across continents via migration, they now function as both indicators and vectors of global AMR spread, carrying resistant bacteria into remote and otherwise unexposed ecosystems far from the original source of antibiotic contamination (6). Combined with the demonstrated capacity of antibiotic residues to suppress plant growth-promoting soil microorganisms and beneficial bacterial genera, this indicates that antibiotic pollution's biodiversity footprint extends from soil microbiota through plants, invertebrates, fish, and birds thus impacting all the ecosystems.

12.Regulation and Mitigation

Regulations and policies responding to antibiotic pollution has strengthened in recent years but remains fragmented relative to the scale of the problem. In September 2024, the World Health Organization released its first-ever guidance on wastewater and solid waste management specifically for antibiotic manufacturers, targeting the pharmaceutical-manufacturing source category directly (4). At a broader policy level, the European Council adopted a recommendation in June 2023 intensifying European Union action against AMR, emphasising infection prevention and control, monitoring and surveillance, and responsible access to antimicrobials across human, animal, and environmental sectors simultaneously, while the WHO Quadripartite has recommended a five-part strategy spanning governance, innovation, global collaboration, investment, and accountability (6,13).

A consensus across the literature highlights several critical priorities moving forward: upgrading wastewater treatment infrastructure to target antibiotic residues that conventional biological systems fail to reliably remove; strictly enforcing veterinary withdrawal protocols to minimize residue carryover into food products; broadening the biomonitoring of wildlife sentinel species to monitor cross-border AMR dissemination; expanding field-scale investigations into antibiotic dynamics within

soil and crops; and sustaining investment in the antimicrobial development pipeline, which the WHO's 2024 assessment deemed insufficient to counteract escalating drug-resistant threats. Furthermore, the economic imperative for robust, synchronized mitigation is underscored by the World Bank's estimate of a 1.1–3.8% contraction in global GDP due to antibiotic residues and resistance, which powerfully reinforces the core public health rationale.

13.CONCLUSION

Antibiotic residues have evolved from being a localized pharmaceutical contaminant into a global environmental health concern that transcends traditional boundaries between human, animal, and ecosystem health. This review demonstrates that these residues persist across interconnected environmental compartments, continuously cycling through soil, water, plants, wildlife, livestock, aquatic organisms, and ultimately the human food chain.

Beyond selecting for resistant bacteria and genes, antibiotic residues disrupt nutrient-cycling microbial communities in soil and water, impair crop growth and quality, alter aquatic ecosystems, and reduce biodiversity. These widespread impacts threaten ecosystem resilience, food security, environmental sustainability, and public health, while indirectly contributing to climate-relevant biogeochemical disturbances.

The literature consistently supports the One Health framework, for addressing antibiotic residue pollution because the sources, transmission pathways, and consequences are inseparably linked across human, veterinary, agricultural, and environmental sectors. Future priorities should include advanced wastewater treatment technologies, stricter regulation of pharmaceutical effluents and veterinary antibiotic use, improved compliance with withdrawal periods, routine environmental surveillance of residues and resistance genes, wildlife biomonitoring, and continued research into environmental thresholds and long-term ecological consequences.

Recognizing antibiotic residues as both an environmental pollutant and a driver of antimicrobial resistance shifts the focus from treating resistant infections alone to preventing their environmental emergence. Such an integrated One Health strategy is essential for safeguarding ecosystem function, preserving antibiotic effectiveness, protecting biodiversity, and ensuring sustainable health for future generations

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ONE HEALTH APPROACH TO VOLATILE ORGANIC COMPOUNDS

A Systematic Review of Sources, Exposure Pathways, and 360° Impacts on Environment, Animal, Plant, Aquatic, and Human Health

TABLE OF CONTENTS:

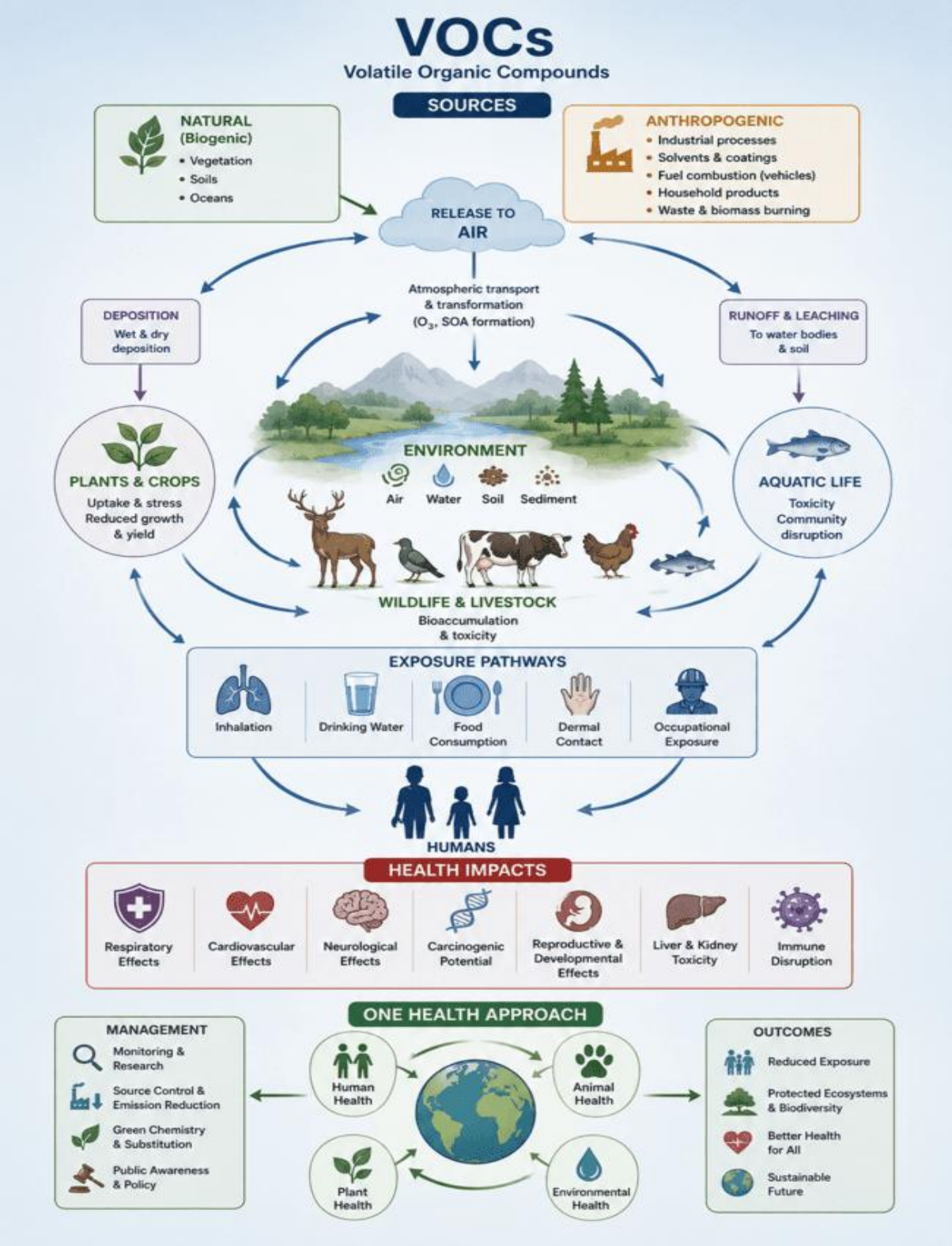
ABSTRACT

Volatile organic compounds (VOCs) represent a significant, dual-natured pollutant class, functioning both as essential atmospheric constituents and potent environmental toxics. Volatile organic compounds (VOCs) represent a significant, dual-natured pollutant class, functioning both as essential atmospheric constituents and potent environmental toxics. This systematic review explores the "One Health" implications of VOCs, examining their interconnected impacts across human, animal, plant, and aquatic systems. VOCs exhibit a unique toxicological profile, exerting direct harm through inhalation, ingestion, and dermal contact, while simultaneously acting as precursors to hazardous secondary pollutants like ground-level ozone and secondary organic aerosols (SOA) via photochemical reactions with nitrogen oxides in sunlight.

Despite the absolute dominance of natural (biogenic) sources globally—with vegetation, soils, and oceans contributing an estimated 65–90% of global emissions—anthropogenic VOC emissions are heavily concentrated in urban and industrial zones where human exposure is maximized. These human activities, particularly industrial solvent usage, industrial process emissions, domestic combustion, vehicle exhaust, and volatile chemical products (VCPs), drive the most critical health and ecological risks because they are enriched with highly toxic air toxics such as benzene and formaldehyde.

This review synthesizes current evidence on the cascading effects of VOCs across ecosystems. In humans, exposure is linked to mutagenic cancer risks, acute respiratory illnesses, nervous system harm, and cardiovascular disorders. In domestic companion animals and wild fauna, VOC exposure induces significant respiratory complications, systemic organ toxicity affecting hepatic and renal functions, and progressive neurological damage. Furthermore, VOC-derived ground-level ozone inhibits plant physiology, altering biogenic emissions and natural chemical defenses, which persistently impacts global crop yields and threatens food security. In aquatic ecosystems, contamination deteriorates physical and chemical water quality indices, affecting biological community dynamics. Concluding with a framework for integrated management, this article highlights the urgent need for precise source apportionment and cross-sectoral regulation to mitigate the multifaceted health, ecological, and indirect climate threats posed by VOC pollution.

GRAPHICALABSTRACT



1.INTRODUCTION:

Volatile organic compounds (VOCs) are a broad chemical class of carbon-based compounds, including hydrocarbons, aldehydes, alcohols, and halogenated organics .The are well known for their high vapour pressure and consequent tendency to evaporate readily into the atmosphere at ambient temperature (1,4). Unlike most pollutants, VOCs are not exclusively anthropogenic in origin: plants, soils, and oceans emit substantial quantities of biogenic VOCs (BVOCs) such as isoprene and

monoterpenes as part of normal physiological function, communication, and defence, while human activity contributes a separate and chemically distinct anthropogenic VOC (AVOC) load (3,18). This dual origin is central to understanding VOCs as a pollutant class: the same broad chemical category includes both a beneficial, naturally-occurring atmospheric constituent and some of the most hazardous industrial and combustion-derived air toxics known, including benzene and formaldehyde (1,13,14).

What further distinguishes VOCs from most pollutant classes is their dual toxicological identity: many individual VOCs are directly toxic on inhalation, ingestion, or dermal contact, while as a class they also function as chemical precursors that react photochemically with nitrogen oxides in sunlight to generate ground-level ozone and secondary organic aerosol (SOA) — two additional, highly consequential air pollutants in their own right (1,18). This means VOC-related harm operates through two parallel mechanisms: the direct toxicity of the parent compounds themselves, and the indirect toxicity of the secondary pollutants their atmospheric chemistry generates

2.Methodology:

Literature was retrieved from PubMed/PMC and Scopus-indexed journals (ScienceDirect, MDPI, Nature Portfolio, Wiley, RSC, IWA Publishing) using combinations of the terms "volatile organic compounds," "VOC," "source apportionment," "biogenic," "anthropogenic," "birds," "wildlife," "plant," "ozone," "human health," "aquatic," "climate," and "biodiversity," restricted to free/open-access articles published between 2022 and 2026, with a small number of frequently-cited foundational primary studies retained where no more recent equivalent exists. From the results, articles were purposively selected to give the broadest possible topical coverage — comprehensive reviews for background and source data, and focused primary/systematic reviews for each specific health or ecological domain consistent with a focused rather than exhaustive systematic review design.

3. Sources of VOC Contamination:

Global VOC emissions are dominated, in mass terms, by natural (biogenic) sources rather than human activity — an unusual feature among the pollutant classes reviewed in this series. Vegetation, soils, and oceans are estimated to contribute approximately 65–90% of global VOC emissions, with anthropogenic activity contributing the remaining 10–35%, a range summarised in Figure 1 (3). This does not mean anthropogenic VOCs are of secondary concern, however: anthropogenic sources are disproportionately enriched in the specific compounds — benzene, toluene, formaldehyde, and related aromatics — most strongly associated with human toxicity, and are heavily concentrated in urban and industrial areas where human exposure is highest (1,4,5).VOC (Volatile Organic Compound) is used in a vast array of household, commercial, and industrial products that easily evaporate at room temperature.

Everyday Consumer & Household Products

- **Building Materials:** Paints, varnishes, lacquers, wood preservatives, and new flooring.
- **Cleaning & Personal Care:** Disinfectants, air fresheners, makeup, and aerosol sprays.
- **Office & Craft Supplies:** Printer ink, permanent markers, glues, and correction fluid.
- **Automotive & Hobby:** Petroleum fuels, paint thinners, and pesticides.

Industrial & Commercial Uses:

- **Manufacturing:** Solvents used for cleaning and etching in the electronics, semiconductor, and circuit board industries.
- **Automotive & Machinery:** Industrial degreasers, hydraulic fluids, and engine repair supplies.
- **Textiles & Printing:** Dry cleaning agents and printing press inks

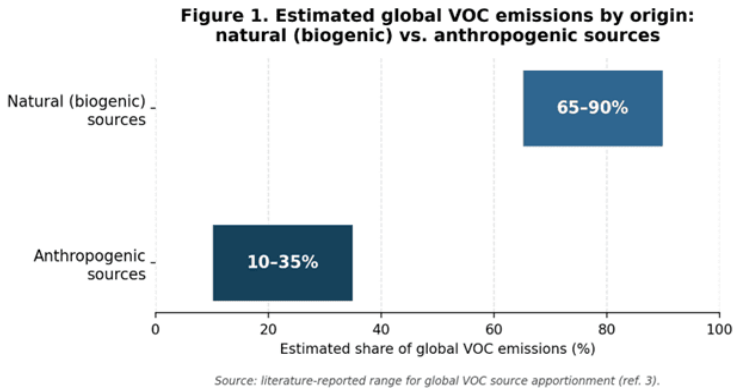


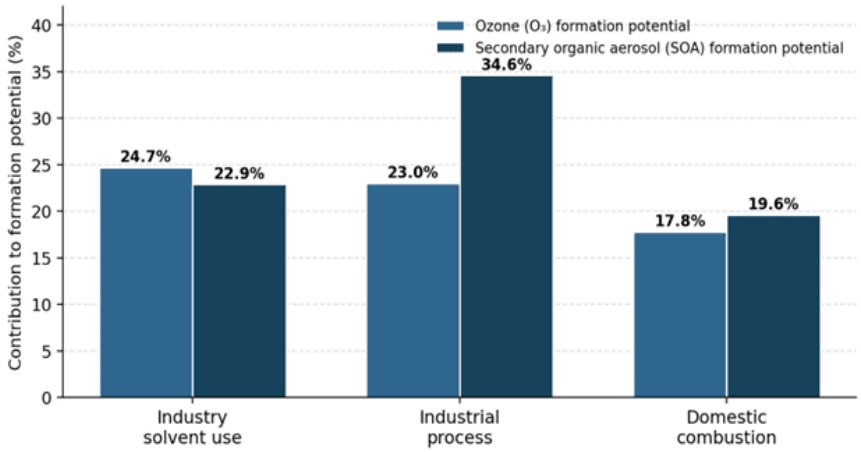
Figure 1. Estimated global VOC emissions by origin: natural (biogenic) versus anthropogenic sources .

Among anthropogenic sources, industrial solvent use, industrial process emissions, and domestic combustion are consistently identified as leading contributors to the downstream formation of ozone and secondary organic aerosol, the two principal secondary pollutants VOCs generate. A source-apportionment study of anthropogenic VOC emissions found that alkylbenzenes were the largest chemical contributor to both ozone (36.0%) and SOA (51.6%) formation potential, with industry solvent use, industrial process emissions, and domestic combustion the three leading sectoral sources, as shown in Figure 2 (18).

A city-scale case study applying environment- and health-risk-oriented source apportionment in Harbin, a megacity in northeastern China, similarly found alkanes and aromatics to be major contributors to total measured VOCs, with aromatics responsible for the highest hydroxyl-radical loss rate, ozone formation potential, and secondary organic aerosol formation potential among the chemical groups analysed, despite the study site having no major industrial point sources nearby — illustrating that even a mixed transportation, residential, and commercial urban area can generate substantial VOC-driven secondary pollution risk (6).

Vehicle exhaust and fuel evaporation, and — increasingly, as regulation of traditional industrial sources has tightened — volatile chemical products (VCPs) such as cleaning agents, personal care products, and coatings, represent additional major anthropogenic categories, with residential buildings themselves now recognised as meaningful contributors of VOCs to ambient outdoor air (2,5).

Figure 2. Leading sectoral contributors to ozone and secondary organic aerosol (SOA) formation from anthropogenic VOCs



Source: sector-level ozone/SOA formation potential from anthropogenic VOC emissions (ref. 18).

Figure 2. Leading sectoral contributors to ozone and secondary organic aerosol (SOA) formation potential from anthropogenic VOC emissions (18).

Source category	Relative significance	Notes	Reference(s)
Vegetation, soils & oceans (biogenic)	Dominant by mass (≈65–90% of global emissions)	Isoprene, monoterpenes; also feeds into ozone/SOA formation	(3,18)
Industrial solvent use & processes	Leading anthropogenic contributor to O_3/SOA formation	Alkylbenzenes the single largest chemical contributor	(18)
Domestic combustion	Significant anthropogenic contributor	Household solid/liquid fuel combustion	(18)
Vehicle exhaust & fuel evaporation	Major urban source, especially BTEX compounds	Benzene, toluene, ethylbenzene, xylene prominent	(1,4)

Volatile chemical products (VCPs) & buildings	Growing share as traditional sources decline	Cleaning agents, coatings, personal care products; indoor-to-outdoor transfer	(2)
Petroleum refining & petrochemical facilities	Major documented industrial point source	Elevated local concentrations of hexane, benzene, toluene	(1)

Table 1. Consolidated source categories of VOC contamination reported in the reviewed literature.

4. Modes of Entry

VOCs move between environmental compartments primarily through atmospheric transport, but also through some industrial spills and leaks that carry them into soil and groundwater, and also via direct discharge into surface water (5,16). Because of their volatility, VOCs that reach soil or water do not remain there indefinitely: a substantial fraction of it re-volatilises back into the atmosphere, creating an ongoing exchange between compartments rather than the largely one-directional accumulation seen with more persistent pollutants such as PFAS (5,16,17). Indoor air represents a distinct and often underappreciated compartment: indoor VOC concentrations are frequently higher than outdoor levels because of continuous emission from building materials, furnishings, and consumer products combined with limited air exchange, meaning indoor exposure can dominate an individual's total VOC intake even in the absence of any nearby industrial source (4,15).

Biological uptake occurs principally through inhalation (the dominant route for both direct VOC toxicity and secondary ozone/SOA exposure), dermal contact (particularly for liquid-phase VOCs in consumer products), and ingestion of contaminated water (1,16). Figure 3 summarises this cascade from source through environmental compartments to biota and human exposure, including the distinct secondary pathway via ozone and SOA formation.

Figure 3. Sources, environmental compartments, and modes of entry of volatile organic compounds into biota and human exposure routes

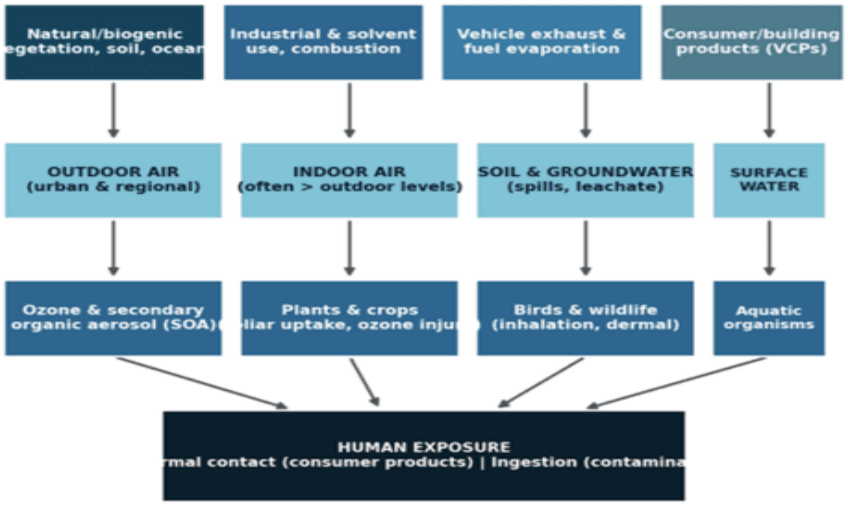


Figure 3. Schematic overview of sources, environmental compartments, and principal modes of entry of volatile organic compounds into biota and human exposure pathways, including the secondary ozone/SOA pathway (1,3,5,16,18).

5. Effects on Wildlife:

The widespread contamination of ecosystems by volatile organic compounds (VOCs) has caused disruptions across diverse wildlife populations. It was found that VOCs impair the physiology of both terrestrial and aquatic organisms, enter food chains via biomagnification, and have been implicated in gradual shifts in population dynamics that could contribute to the extinction risk of especially

vulnerable species. Wild animals often face dual exposure vectors: direct bioconcentration from contaminated physical environments, such as water or sediment, and indirect biomagnification via the continuous consumption of contaminated prey within localized trophic webs. Because apex predators consume organisms that have already concentrated these compounds, the internal tissue burden can multiply as it ascends the food chain, triggering severe systemic complications including reproductive failures, altered immune defenses, and population declines.(8)

5.1 Birds

Because of their uniquely efficient, highly vascularised respiratory anatomy, which can make them more sensitive to airborne contaminants than many mammalian species,Birds are considered an especially informative model for studying inhaled air pollutant toxicity (7). A controlled inhalation study exposing captive Japanese quail (Coturnix c. japonica) and American kestrels (Falco sparverius) to a mixture of VOCs (benzene and toluene) together with nitrogen dioxide and sulfur dioxide found increased plasma corticosterone — a physiological stress marker — in both species during exposure, alongside a trend toward increased hepatic biotransformation enzyme activity (EROD) in kestrels, though no significant alteration to T- and B-cell immune responses or immune organ histology was detected in either species at the doses tested (7). The authors noted that toxicological research on airborne contaminants in wildlife has historically lagged well behind aquatic toxicology research, despite birds' demonstrated physiological sensitivity to inhaled pollutants — an evidence gap that more recent reviews confirm remains only partially closed (7,8).

5.2 Terrestrial and aquatic invertebrates

Beyond birds, the broader living-organisms review found that VOC exposure degrades water quality indices in affected water bodies, with downstream physiological impacts on aquatic vertebrates and invertebrates, and documented toxicological effects severe enough in some ecotoxicological studies to raise concern about local population decline in sensitive species (8). Because many VOCs bioaccumulate and enter food chains via biomagnification in a manner structurally similar to other persistent organic pollutants, species at higher trophic levels — including predatory birds and larger aquatic vertebrates — face compounding exposure risk relative to primary consumers (8).

Taxon	Documented effects	Reference(s)
Birds (experimental inhalation exposure)	Elevated physiological stress hormone (corticosterone); trend toward increased hepatic biotransformation enzyme activity	(7)
Birds (general)	Heightened sensitivity to airborne contaminants due to respiratory anatomy; historically understudied relative to aquatic species	(7,8)
Aquatic vertebrates & invertebrates	Impaired physiology, degraded water quality exposure, population-level concern for sensitive species	(8)
Terrestrial invertebrates	Documented toxicological effects; contribute to food-chain biomagnification	(8)

Table 3. Summary of documented VOC-related effects across animal taxa.

5.3 Effects on Domestic animals

Given that indoor exposure represents a substantial source of these compounds, the resultant atmospheric load exerts profound physiological impacts on domestic companion species, including cats and dogs.

5.3.1 Respiratory Complications

Inhalation of volatile organic compounds (VOCs) is associated with pulmonary distress in domestic animals. Specifically, exposure to chemical agents such as formaldehyde and benzene has been documented to induce coughing, wheezing, and dyspnea in feline and canine populations. In the absence of rigorous air quality management, these initial respiratory insults can progress into chronic, more debilitating physiological conditions.

The comparatively smaller and more delicate airway architecture of domestic pets increases their vulnerability to even marginal concentrations of atmospheric pollutants relative to humans. Research suggests that suboptimal indoor air quality can trigger significant hypercellular responses within the canine respiratory tract, illustrating the heightened sensitivity of these companion species to inhaled toxins.(19)

5.3.2 Neurological Impacts

Prolonged, excessive VOC exposure exerts a detrimental influence on the central nervous system of pets. In dogs, contact with toluene and xylene can manifest as cognitive confusion, tremors, and systemic neurological degradation. Similarly, cats exhibit comparable neurotoxic symptoms, including disorientation and progressive muscular weakness, following environmental exposure. Because these neurodegenerative changes often occur gradually, pet owners may fail to identify subtle behavioral shifts as consequences of poor air quality. Consequently, the early-stage mitigation of VOC levels is critical to preventing the onset of irreversible neurological pathology in domestic animals.(19,20)

5.3.3 Systemic Organ Toxicity

Certain VOCs exhibit specific organotropism, targeting vital systems within the animal body. Carbon tetrachloride and trichloroethylene are recognized contributors to hepatic and renal impairment in dogs and cats. The liver, functioning as the primary site for chemical biotransformation, is particularly susceptible to the metabolic burden imposed by constant VOC inhalation. This persistent toxicological load can lead to severe systemic health complications, necessitating professional remediation services to reduce environmental exposure and alleviate the metabolic strain on the animal.(19,20)

6. Effects on Plant Health and Nutrition

Plants play a dual role as major biogenic VOC emitters and highly sensitive receptors of VOC-derived secondary pollution, mainly ground-level ozone. Alongside ozone, particulate matter, and nitrogen dioxide, VOCs impair plant germination, morphology, photosynthesis, and enzyme activity, reducing crop yield and nutritional quality (9). Since tropospheric ozone forms photochemically from VOCs and nitrogen oxides in sunlight, much evidence focuses on ozone specifically; a 40-year analysis of U.S. maize and soybean yields confirmed it as a persistent driver of crop yield loss, varying regionally by variety, farming practices, and local microclimatee (10).

At the physiological level, ozone exposure — and the oxidative stress it induces — directly alters plants' own biogenic VOC emissions, creating a feedback loop between air pollution and plant volatile chemistry. A controlled study of three tropical tree species from Atlantic Forest remnants in Brazil found that ozone exposure induced programmed cell death and hydrogen peroxide accumulation in leaf tissue, with species-specific differences in biogenic VOC response depending on each species' underlying carbon-allocation and defence strategy (11). A separate controlled study of oilseed rape found that ozone fumigation caused a rapid decrease in leaf monoterpene and sesquiterpene concentrations while increasing a range of oxygenated volatile species, nearly doubling the total carbon lost by leaves as volatiles — changes with plausible knock-on consequences for plant-plant and plant-insect chemical signalling, since these terpenoid compounds play a documented role in such communication (12).

Because biogenic VOCs also serve protective functions — including antifungal and antimicrobial activity and herbivore deterrence — ozone-driven disruption of normal BVOC emission profiles may compromise plants' natural chemical defences at the same time that direct oxidative damage affects growth and yield, a dual vulnerability distinct from the more straightforward dose-response toxicity documented for most other pollutant classes in this review series (11,12).

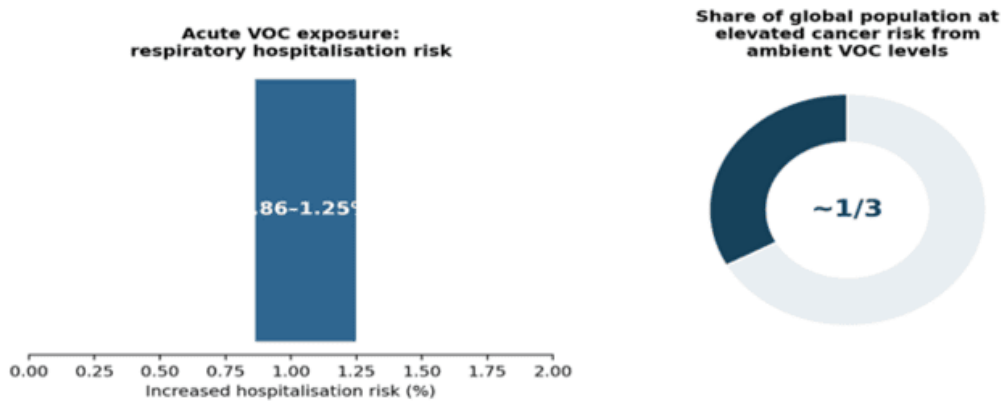
7. Effects on Human Health

Human exposure to VOCs occurs primarily through inhalation of outdoor and indoor air, with indoor concentrations frequently exceeding outdoor levels, alongside dermal contact with consumer products and, less commonly, ingestion of contaminated drinking water (1,4,15).

7.1 Carcinogenicity

Benzene and formaldehyde are widely evaluated VOCs classified as Group 1 human carcinogens by the International Agency for Research on Cancer. Benzene exposure is linked to leukaemia and haematologic malignancies, while formaldehyde is associated with DNA-protein crosslink formation, leukaemia, and nasopharyngeal cancer. Global modelling shows that a lifetime exposure to just 1 μg/m³ ambient concentration causes an estimated 13 extra lung and nasopharyngeal cancer cases per million people for formaldehyde, and 2.2–7.8 extra leukaemia cases per million people for benzene. Formaldehyde was identified as the top driver of cancer risk among 187 hazardous air pollutants in the United States, with a third of the global population facing elevated cancer risks from ambient VOC concentrations, as shown in Figure 4..(13,14)

Figure 4. Selected human health risk metrics associated with VOC exposure



Source: umbrella review and global VOC cancer-risk modelling study (refs. 13, 14).

Figure 4. Selected human health risk metrics associated with VOC exposure: acute respiratory hospitalisation risk and the share of the global population at elevated cancer risk from ambient VOCs (13,14).

7.2 Respiratory, cardiovascular, and neurological effects

An umbrella review spanning 35 countries linked occupational and environmental benzene exposure to increased micronucleus frequency, and environmental benzene specifically to childhood haematologic malignancies, asthma, and low birth weight. Additionally, styrene exposure was tied to dyschromatopsia and formaldehyde to sinonasal cancer. Short-term VOC exposure increases respiratory hospitalisation risks by 0.86–1.25%, while chronic exposure is linked to cardiovascular, neurological, and preterm birth outcomes.(13,14)

7.3 Indoor exposure

Because indoor VOC concentrations frequently exceed outdoor levels, indoor exposure assessment has become a major research focus. A study across 79 hotels in Hunan, China, detected benzene, toluene, xylene, and formaldehyde during both summer and winter sampling periods. By employing quantitative risk assessment methods — lifetime carcinogenic risk and non-carcinogenic hazard

quotients — the study characterized health risks for both guests and staff, illustrating the increasing application of setting-specific indoor assessments beyond traditional residential environments.(15)

Organ / system	Reported effects	Reference(s)
Haematologic system	Leukaemia and other haematologic malignancies (benzene, Group 1 carcinogen)	(13,14)
Respiratory system	Nasopharyngeal / lung / sinonasal cancer (formaldehyde) asthma; 0.86–1.25% increased hospitalisation risk	(13,14)
Cardiovascular system	Increased cardiovascular disease risk with long-term exposure	(14)
Nervous system	Neurological disorders associated with long-term exposure	(14)
Reproductive/developmental	Low birth weight, preterm birth	(13,14)
Genetic/cellular level	Elevated micronucleus frequency (genotoxicity biomarker); DNA-protein crosslink formation (formaldehyde)	(13)
Sensory systems	Dyschromatopsia (colour vision impairment) associated with styrene exposure	(13)

Table 4. Summary of documented VOC effects on human organ systems.

8. Effects on Aquatic Health

VOCs enter aquatic systems primarily via industrial discharge, spills, and runoff, with benzene, toluene, ethylbenzene, and xylene (BTEX), chlorinated solvents, and volatile pesticides among the compounds most commonly detected in contaminated water and posing risk to both aquatic ecosystems and human water users even at low concentrations (16). A review of VOC detection methods in aquatic samples emphasised that because VOCs threaten both aquatic ecosystem health and drinking water safety, effective and rapid detection technology is a prerequisite for adequate environmental management, noting that VOC contamination in water frequently originates from industrial sites and can subsequently migrate into groundwater and drinking water supplies (16). VOCs also occur naturally in aquatic systems, complicating environmental risk assessment: marine phytoplankton and freshwater cyanobacteria produce their own volatile compounds, including terpenoids and oxygenated species, some of which — such as β-cyclocitral — function as chemical deterrents used by cyanobacteria against algal competitors (17). This means aquatic VOC monitoring must distinguish anthropogenic contamination from a substantial and chemically overlapping natural background, a challenge broadly analogous to the biogenic/anthropogenic distinction that complicates atmospheric VOC source apportionment (3,17). The broader living-organisms review found that VOC contamination measurably deteriorates water quality indices and impairs the physiology of exposed aquatic organisms, with effects capable of altering population dynamics in sensitive species over time (8).

9. Effects on Climate Change

VOCs are not typically classified as greenhouse gases in their own right, but they exert a significant, well-established indirect effect on climate through their role as precursors to ground-level ozone and

secondary organic aerosol (SOA), both of which are short-lived climate forcers with documented radiative effects (18). A systematic quantitative review of VOC emissions from anthropogenic and natural sources found that both categories contribute to ozone and particulate matter formation via photochemical oxidation, with climate change itself expected to alter future biogenic VOC emission patterns as rising temperatures increase plant volatile output — a feedback loop in which climate change amplifies natural VOC emissions, which in turn contribute to further ozone and aerosol formation (18).

Volatile organic compounds (VOCs) occupy a unique position at the convergence of direct toxicology, air quality management, and climate science, representing a three-way intersection that is far less pronounced in most other pollutant classes. This distinct role arises because secondary organic aerosol (SOA) particles alter the Earth's radiative balance and reduce atmospheric visibility, while ground-level ozone has well-established adverse effects on both human health and agricultural productivity. Furthermore, regional projections for China indicate that climate-driven surges in biogenic VOC emissions could further elevate summertime ozone and SOA levels by mid-century under standard greenhouse gas scenarios, demonstrating how climate change and VOC-induced air pollution are poised to mutually reinforce one another in the future(18).

10.Effects on Environment and Ecosystems:

Beyond direct toxicity, VOC contamination alters the physical and chemical character of the environmental matrices it affects. In water bodies, VOC contamination measurably degrades water quality indices, a change with knock-on consequences for the broader aquatic community beyond the organisms directly exposed to toxicity (8). In soil and groundwater, spills and improper disposal of VOC-containing materials at industrial and commercial sites create persistent point-source contamination that, unlike the atmospheric VOC pool, can take considerably longer to dissipate given the slower re-volatilisation and degradation rates typical of subsurface environments — creating chronic secondary exposure risk for nearby groundwater users long after the original release (1,16).

Indoor environments represent a further distinct matrix-level concern: because indoor VOC concentrations frequently exceed outdoor levels due to continuous low-level emission from building materials and consumer products combined with limited air exchange, the built environment itself functions as a semi-persistent VOC reservoir distinct from either outdoor air or contaminated water/soil (4,15)

11. Regulation and Mitigation

Regulatory approaches to VOCs must contend with the dual toxicological identity described throughout this review: controlling VOCs simultaneously addresses direct toxicant exposure and secondary ozone/SOA formation, but because a substantial majority of global VOC emissions are natural rather than anthropogenic, regulation can only meaningfully target the anthropogenic fraction, making accurate source apportionment — of the kind demonstrated in the Harbin and Chinese national-level source-apportionment studies reviewed here — a prerequisite for effective policy design (1,5,18). In the United States, the Environmental Protection Agency classifies benzene, toluene, ethylbenzene, xylene, styrene, and formaldehyde as Hazardous Air Pollutants, with benzene and formaldehyde further designated Urban Air Toxic Pollutants, providing the regulatory basis for targeted emission controls on these specific high-priority compounds (13).

The reviewed literature converges on several further priorities: continued monitoring and control of volatile chemical products (VCPs) as their relative contribution to urban VOC emissions grows even as traditional industrial and vehicular sources decline under existing regulation; improved indoor air quality management given that indoor concentrations frequently exceed outdoor levels; sector-targeted controls on industrial solvent use, industrial processes, and domestic combustion given their disproportionate contribution to ozone and SOA formation potential; expanded wildlife toxicology

research to close the substantial evidence gap relative to human health and aquatic toxicology; and continued refinement of rapid, field-deployable VOC detection technology for both air and water monitoring (2,5,7,16,18).

12. CONCLUSION:

A One Health approach is crucial for addressing volatile organic compounds (VOCs) across human, animal, and environmental health. As direct toxicants and precursors to ground-level ozone and secondary organic aerosols, VOCs cause cascading biological harm, necessitating integrated, cross-disciplinary strategies that account for indoor/outdoor air quality, species-specific sensitivities, and climate feedbacks.

They alter environmental matrices, degrading water quality and impacting aquatic organisms. Subsurface industrial spills cause persistent contamination due to low re-volatilisation rates, while limited indoor air exchange concentrates emissions from building materials and consumer products. Biological uptake occurs via dermal contact, ingestion, and primarily inhalation, exposing organisms to both direct toxicants and secondary pollutants.

VOC contamination severely threatens ecosystems. Biomagnification imperils apex predators, while birds suffer due to sensitive, vascular respiratory systems. In domestic animals, indoor exposure triggers respiratory, neurological, and systemic disorders. Although vegetation naturally emits biogenic VOCs, plants are highly susceptible to VOC-induced ground-level ozone, which disrupts photosynthesis, morphology, and germination, reducing crop yields and quality. In humans, exposure drives mutagenic cancer risks alongside reproductive, nervous system, and cardiovascular disorders. Protecting planetary and public health requires advanced monitoring and strict regulation of anthropogenic emissions. Because biogenic sources dominate globally, precise source apportionment is vital to isolate human-derived fractions. Mitigation must focus heavily on sectors with high ozone and secondary organic aerosol formation potential—specifically industrial processes, solvent usage, domestic combustion, and volatile chemical products—within a unified, cross-disciplinary framework.

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ONE HEALTH APPROACH TO PESTICIDES

A Systematic Review of Sources, Exposure Pathways, and 360° Impacts on Environment, Animal, Plant, Aquatic, and Human Health

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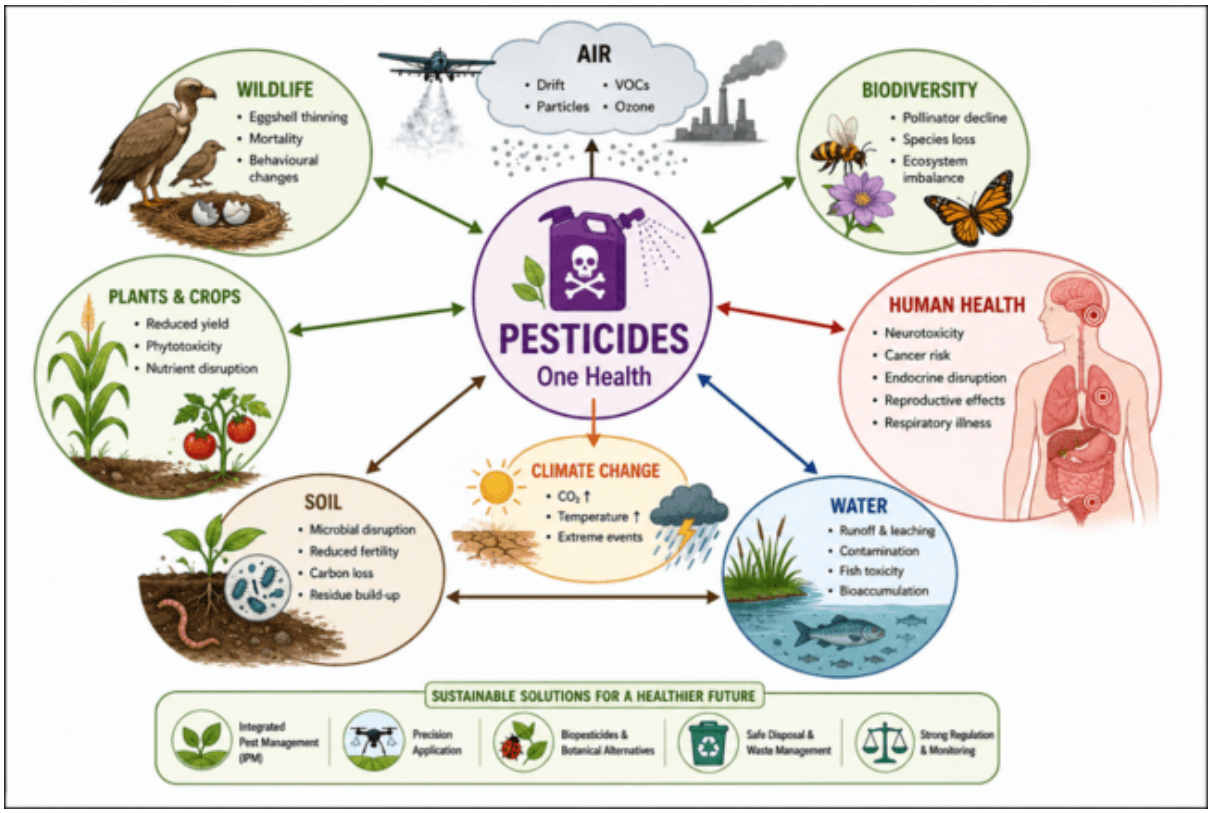
ABSTRACT

Pesticides have become a critical component of modern agriculture, yet their extensive use poses multifaceted risks to global ecosystems and public health. This systematic review investigates the "One Health" dimension of pesticide application, tracing their pathways from petrochemical-based synthesis and field application to their environmental fate and long-term impacts on plants, animals, and human health. The analysis highlights the shift from legacy organochlorines to "currently used pesticides" (CUPs), emphasizing the pervasive contamination of soil, water, and air. We examine mechanisms of toxicity—including neurotoxicity, endocrine disruption, and carcinogenicity—as well as the systemic biodiversity loss, contributions to climate change, and disruption of critical ecosystem services. By assessing international regulatory frameworks and sustainable mitigation strategies—such as Integrated Pest Management (IPM), precision application, and biopesticide adoption—this review provides a comprehensive overview for researchers and policymakers. Ultimately, the synthesis underscores the urgent need for harmonized, hazard-based regulatory reforms and a transition toward sustainable agricultural practices to protect the delicate balance of ecological and human health.

GRAPHICAL ABSTRACT



1.INTRODUCTION:

Pesticides are chemical or biological agents formulated to prevent, destroy, repel, or mitigate insects, weeds, fungi, rodents, and other organisms considered detrimental to agricultural production and public health. They are the most harmful and toxic among agrochemicals used vigorously. Just after a few decades of the onset of green revolution, the consumption of toxic chemical pesticides has

increased to such an extent that it has evolved into one of the biggest threats not only for the environment but also for human health. Excessive use of toxic chemical pesticides not only disturbs the ecological dynamics but also creates numerous health-related problems in human beings and other living organisms Since the mid-twentieth century, synthetic pesticides have underpinned the intensification of agriculture, enabling substantial gains in crop yield and playing a central role in vector-borne disease control. However, the same chemical properties that make pesticides effective against target pests — bioactivity, environmental mobility and, in some cases, persistence — also render them capable of harming non-target organisms, contaminating environmental compartments, and accumulating along food chains.

Global agriculture has undergone a gradual shift from legacy organochlorine pesticides (OCPs) such as DDT and hexachlorocyclohexane (HCH), now largely banned or restricted under international treaties, toward a newer generation of “currently used pesticides” (CUPs), including organophosphates, carbamates, pyrethroids, neonicotinoids, and modern fungicides and herbicides. CUPs were designed to have shorter environmental half-lives and reduced bioaccumulation potential; nevertheless, accumulating evidence shows that they still pose measurable risks to human health and ecosystems [1].

The primary focus of this analysis remains on the specific pesticide categories and active chemical agents that characterize the intensive agricultural landscape of South Asia — a region that serves as a critical model for pesticide reliance within emerging agrarian economies. Systematic field assessments and comprehensive residue-monitoring data consistently highlight a recurring profile of chemical consumption. Predominantly, organophosphates like chlorpyrifos, monocrotophos, profenofos, dimethoate, and malathion; carbamates including carbaryl and carbendazim; the pyrethroid cypermethrin; and the neonicotinoid imidacloprid represent the most pervasive contaminants. Furthermore, fungicides such as mancozeb, propiconazole, and bitertanol, alongside the herbicide glyphosate and elemental sulphur, emerge as the primary compounds detected across food supplies, water systems, and biological specimens, reflecting the highest volumes of environmental application [1-4,6].

2.METHODOLOGY

Literature was retrieved from PubMed/PMC and Scopus-indexed journals (ScienceDirect, Springer Nature, MDPI, Frontiers, ASCE Library) using combinations of the terms"currently used pesticides," "organophosphate," "neonicotinoid," "pyrethroid," "glyphosate," "pesticide exposure," "pesticide residue," "soil microbial community," "aquatic toxicity," "biodiversity," and "pesticide climate change," together with the individual active-ingredient names surveyed in this review. Records were restricted to freely accessible, full-text sources to ensure verifiability. From the results, some specific articles were selected to give the broadest possible topical coverage — comprehensive reviews for background and source data, and focused reviews for each specific health or ecological domain.

3.SOURCES

The overwhelming majority of currently used pesticides originate from petrochemical feedstocks. Active ingredients are synthesized from hydrocarbon precursors combined with chlorine, phosphorus,

sulphur, or nitrogen to yield organophosphate, organochlorine, carbamate, or pyrethroid molecules; formulated products additionally contain solvents, surfactants, and inert carriers, many of which are themselves derived from fossil fuels [8,17]. Key sources contributing pesticide loads to the environment include:

• Agricultural field application — foliar spraying, soil drenching, seed treatment, and granular soil application on cereals, pulses, oilseeds, cotton, fruits, and vegetables.

• Public health vector-control programmes — residual indoor spraying and larvicide application (e.g., organophosphates, pyrethroids).

• Household and urban pest control — domestic insecticide sprays, termiticides, and rodenticides.

• Horticultural and post-harvest treatment — fungicide dips and fumigants applied to stored produce.

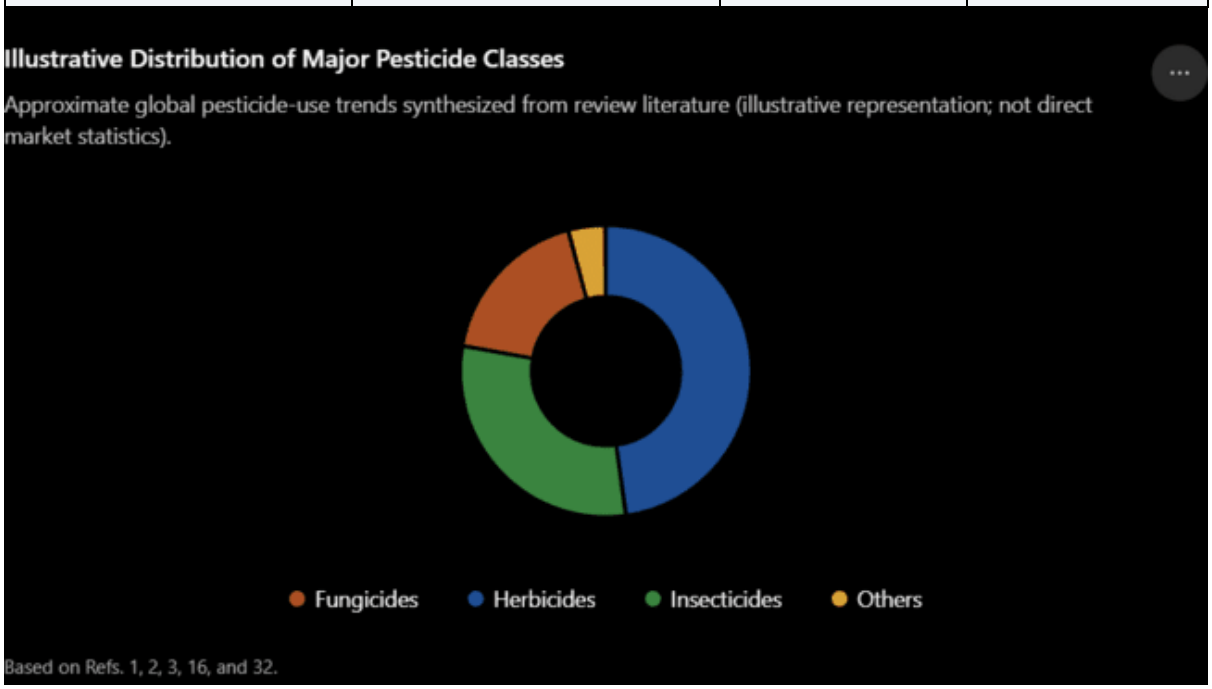
• Industrial manufacturing and formulation units, and improper disposal of expired stock or empty containers.

Among the classes surveyed, organophosphates (chlorpyrifos, monocrotophos, profenofos, dimethoate, phorate, malathion) remain dominant insecticides because of their broad-spectrum activity and comparatively low cost; the dithiocarbamate fungicide mancozeb and elemental sulphur are the most heavily used fungicidal/acaricidal inputs by volume; the neonicotinoid imidacloprid is, after glyphosate, the second most widely used pesticide globally; and glyphosate remains the most extensively applied herbicide worldwide [3,5,15].

Table 1. Major Pesticide Classes Currently in Widespread Agricultural Use

Class	Representative Active Ingredients	Primary Target	Typical Crops/Uses
Organophosphates	Chlorpyrifos, monocrotophos, profenofos, dimethoate, phorate, malathion	Insects, mites	Cotton, rice, vegetables, fruit orchards
Carbamates	Carbaryl, carbendazim	Insects; carbendazim also fungicidal	Vegetables, cereals, fruit
Synthetic pyrethroids	Cypermethrin	Insects	Cotton, vegetables, stored grain
Neonicotinoids	Imidacloprid	Sap-sucking insects (aphids, whiteflies)	Cotton, rice, vegetables; seed treatment

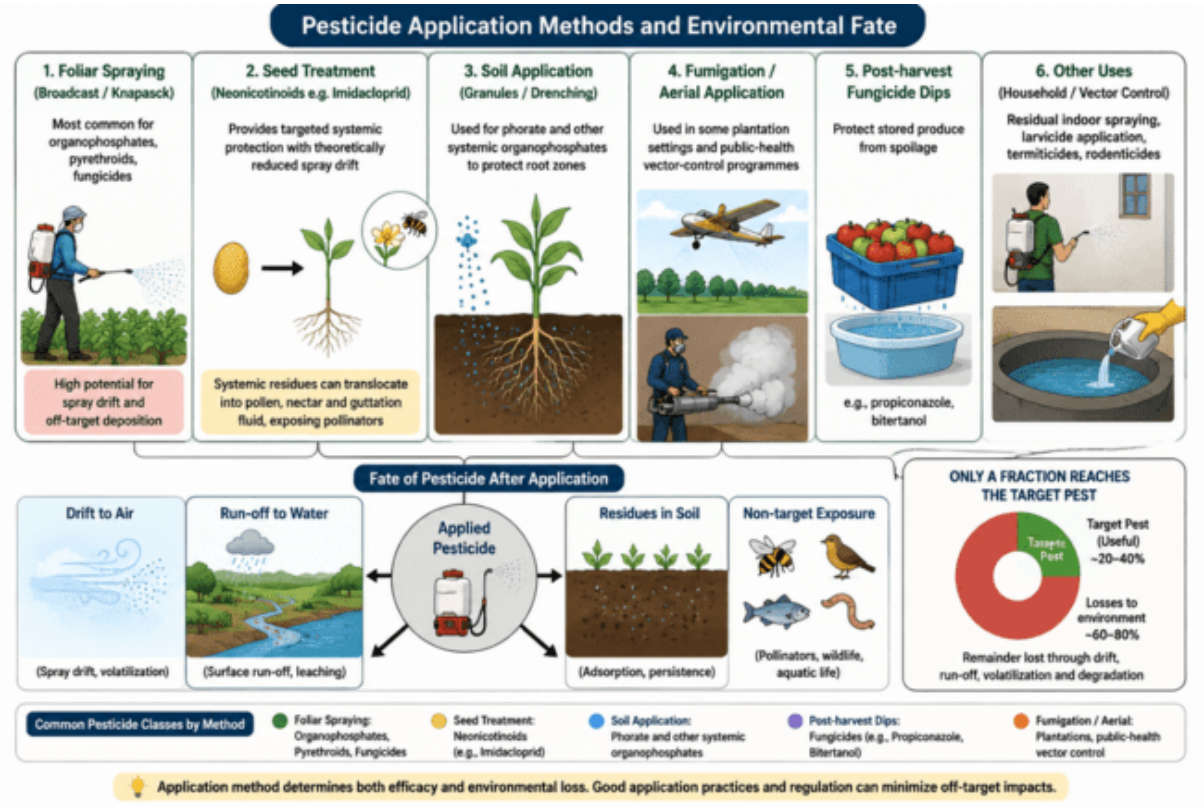
Dithiocarbamate/triazole fungicides	Mancozeb, propiconazole, bitertanol	Fungal pathogens	Potato, grapes, cereals, vegetables
Elemental/inorganic	Sulphur, copper compounds	Fungi, mites	Grapes, vegetables, orchard crops
Organophosphonate herbicide	Glyphosate	Broad-spectrum weeds	Tea, plantation crops, non-crop areas



Illustrative distribution of major pesticide classes used worldwide. Herbicides constitute the largest proportion of pesticide use, followed by insecticides and fungicides

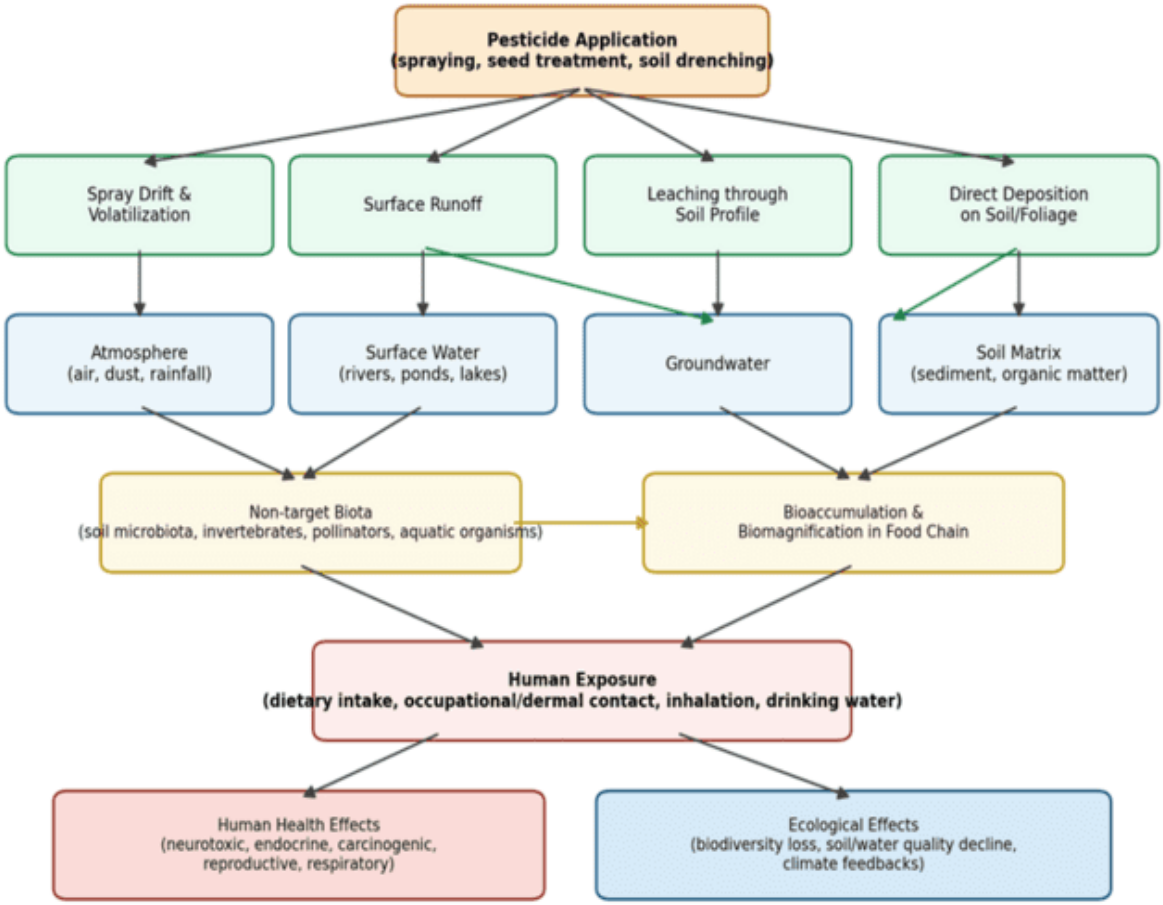
4.Mode of Application & ENTRY

Application methods strongly influence both efficacy and environmental loss fraction. Broadcast and knapsack foliar spraying remains the most common method for organophosphates, pyrethroids and fungicides, but has a high potential for spray drift and off-target deposition. Seed treatment with neonicotinoids such as imidacloprid offers targeted systemic protection with theoretically reduced spray drift, yet systemic residues can still translocate into pollen, nectar and guttation fluid, exposing pollinators [5,18]. Soil application (granules or drenching) is used for phorate and other systemic organophosphates to protect root zones. Fumigation and aerial application are used in some plantation and public-health vector-control settings. Post-harvest fungicide dips (e.g., propiconazole, bitertanol) protect stored produce from spoilage. Irrespective of method, only a fraction of applied active ingredient reaches the target pest; the remainder disperses into air, soil, and water, or is lost through drift, run-off, and volatilization [13,17].



Pesticides enter environmental compartments through four principal, often overlapping, pathways: (i) spray drift and volatilization into the atmosphere; (ii) surface run-off following rainfall or irrigation, transporting residues into streams, rivers and ponds; (iii) leaching through the soil profile into groundwater, particularly for water-soluble compounds; and (iv) direct deposition onto soil and non-target vegetation during and after application [13,17]. From these initial compartments, pesticides are taken up by soil and aquatic biota, bioaccumulate and biomagnify along food chains, and ultimately reach humans through dietary intake, occupational and dermal contact, inhalation of contaminated air or dust, and consumption of contaminated drinking water [1,6,32].

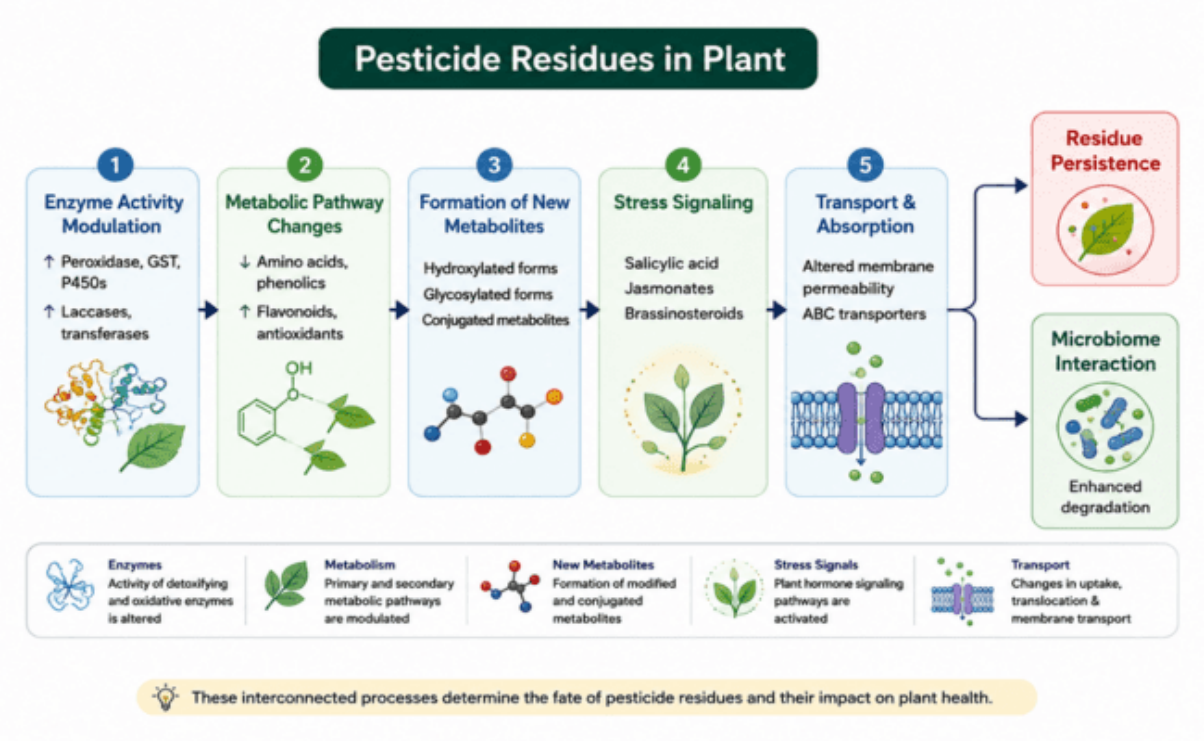
Figure 1. Pathways of Pesticide Entry into the Ecosystem and Human Exposure



5.Effects on Plants and Nutritional Quality

Pesticides Are Absorbed By Crops Through Foliar Or Root Uptake And, Beyond Controlling Target pests, can alter plant biochemistry, gene expression, and cellular signalling. Reviews of pesticide-induced plant stress describe disruption of carbohydrate, amino acid, and fatty-acid metabolism, along with generation of reactive oxygen species and induction of enzymatic and non-enzymatic antioxidant responses, that collectively affect germination, growth, and final yield [25,26]. Metabolomic studies further show that pesticide exposure can reduce or alter amino acids, organic acids and sugars, and modulate flavonoids, phenolic acids, alkaloids and terpenoids — compounds central to plant defence, flavour, and nutritional value [24].

From a food-safety standpoint, residues that persist beyond permissible limits in fruits, vegetables, and cereals raise direct concerns for consumers, while comparative studies of organically versus conventionally grown crops suggest that non-use of synthetic pesticides is associated with a lower incidence of detectable pesticide residues, although differences in some nutrient concentrations (e.g., protein) are driven more by fertilization regime than pesticide use itself(27). Excessive or improperly timed herbicide use can additionally cause phytotoxicity to the treated crop itself and residual injury to sensitive rotational crops planted afterward [27].



6.EFFECTS ON ANIMALS:

6.1 Terrestrial Wildlife and Livestock

Birds, regarded as sentinel species for environmental quality, are particularly vulnerable to pesticide exposure; documented effects include eggshell thinning, reduced egg production, and elevated mortality, with resident bird populations in some regions showing higher organochlorine residue burdens than migratory species [28]. In agricultural settings, domestic livestock face regular exposure to insecticides and herbicides that disrupt endocrine function, yet the subsequent ramifications for their reproductive health and overall productivity continue to be relatively neglected in research[33]. Companion animals such as dogs and cats can experience neurological symptoms, vomiting, dermatitis, and seizures following exposure to organophosphate and pyrethroid household and garden insecticides [31]

6.2 Aquatic Organisms:

Fish and other aquatic animals are exposed to pesticides dermally, through the gills, orally by drinking contaminated water, or via consumption of contaminated prey (secondary poisoning); exposure severity depends on a compound's bioavailability, bioconcentration, biomagnification potential, and environmental persistence [28]. Sub-lethal exposure produces altered behaviour, impaired predator avoidance, reduced cold/heat tolerance, weight loss and impaired reproduction, and monitoring of major rivers has found pesticide residues detectable in the large majority of fish, surface-water, and aquifer samples examined [28]. Mechanistic reviews document histopathological damage (gill, liver, pancreas, intestinal epithelium), acetylcholinesterase inhibition across brain, liver and muscle tissue, and oxidative stress with associated lipid peroxidation and DNA damage in exposed fish [29,30]. Organochlorines and pyrethroids are considered particularly toxic to aquatic invertebrates and fish because of strong sorption to bottom sediments, while organophosphates and carbamates, though highly toxic, are more rapidly metabolized and less persistent in water [28].

7. Effects on Human Health

Human exposure to CUPs occurs via occupational contact during mixing and application, residues in food, and contamination of drinking water. Systematic reviews eport consistent associations between chronic pesticide exposure and non-communicable diseases, including cancer, neurological disorders, endocrine disruption, and respiratory illness, with the strongest evidence among agricultural workers exposed at high intensity or for prolonged periods [9]. Further links suggests that pesticide contact to carcinogenesis, reproductive toxicity, metabolic disruption, and systemic oxidative stress, particularly in low- and middle-income countries where regulatory oversight and occupational protection are limited [10].

7.1 Neurotoxicity

Organophosphates and carbamates act principally by inhibiting acetylcholinesterase, disrupting cholinergic neurotransmission; chlorpyrifos, malathion and carbendazim have specifically been linked to neurodevelopmental deficits in children, DNA damage, and disrupted cell-cycle regulation [1]. Pyrethroids such as cypermethrin act on voltage-gated sodium channels and, while generally considered less acutely toxic than organophosphates, have also been associated with neurobehavioral and cognitive effects, especially where co-exposure with organophosphates occurs, as is common in agricultural and residential settings [11]. Children are considered particularly vulnerable because of their developing nervous systems and comparatively greater exposure per unit body weight [12].

7.2 Endocrine Disruption and Cancer

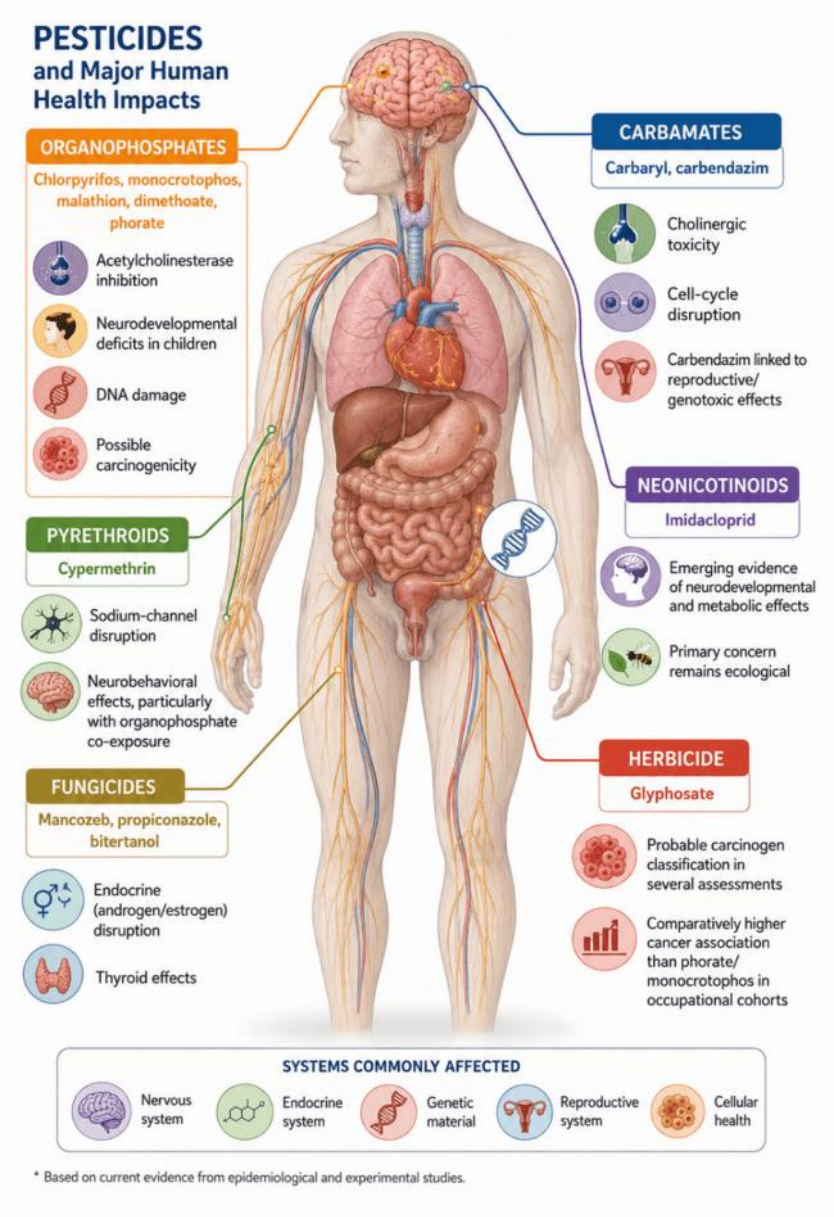
Several currently used fungicides and insecticides — including bitertanol, propiconazole, mancozeb, terbuthylazine, cypermethrin and malathion — have been shown to interfere with androgen and estrogen receptor signalling and aromatase activity, classifying them as potential endocrine-disrupting chemicals [1]. Glyphosate exposure has similarly been associated with elevated cancer risk in occupational cohorts, generally to a greater degree than phorate or monocrotophos, according to comparative public-health analyses [4].

7.3 Dietary Exposure

Food-basket surveys analyzing ninety-four CUPs across cereals, pulses, vegetables, fruits, animal-based foods and water found chlorpropham, chlorpyrifos and carbendazim to have the highest detection frequencies, with chlorpyrifos and chlorpropham detected in roughly a third and a quarter of samples respectively; hazard quotients were highest for chlorpropham in potatoes and chlorpyrifos in rice, indicating that cereals and pulses carry a disproportionate share of dietary pesticide burden [1]. Similar residue-monitoring work on green leafy vegetables identified imidacloprid, chlorpyrifos, profenofos and cypermethrin as the most commonly detected pesticides in fresh produce [6].

Table 2. Summary of Documented Human Health Effects by Pesticide Class

Pesticide	Representative Compounds	Principal Human Health Concerns
Organophosphates	Chlorpyrifos, monocrotophos, malathion, dimethoate, phorate	Acetylcholinesterase inhibition; neurodevelopmental deficits in children; DNA damage; possible carcinogenicity [1,9]
Carbamates	Carbaryl, carbendazim	Cholinergic toxicity; cell-cycle disruption; carbendazim linked to reproductive/genotoxic effects [1]
Pyrethroids	Cypermethrin	Sodium-channel disruption; neurobehavioral effects, particularly with organophosphate co-exposure [11]
Neonicotinoids	Imidacloprid	Emerging evidence of neurodevelopmental & metabolic effects; primary concern remains ecological [5,18]
Fungicides	Mancozeb, propiconazole, bitertanol	Endocrine (androgen/estrogen) disruption; thyroid effects [1]
Herbicide	Glyphosate	Probable carcinogen classification in several assessments; higher cancer association than phorate/monocrotophos in occupational cohorts [4]



8. Effects on Biodiversity

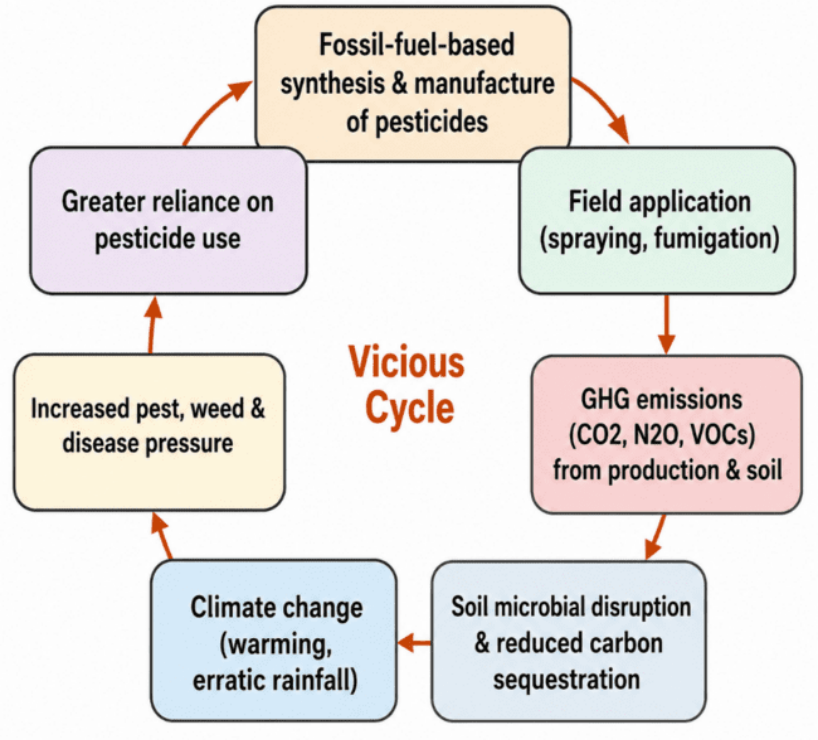
Pesticide drift and run-off routinely reach areas well beyond the intended application zone, exposing beneficial insects, birds, aquatic organisms and soil fauna that were never the intended target [13]. Insecticide exposure reduces floral visitation rates and pollinator resilience, and can destabilize pollination services with consequences for both wild plant communities and crop yield; herbicide drift, even where it does not directly harm pollinators, can delay flowering and reduce floral resource availability [13]. Neonicotinoids such as imidacloprid, now the most widely used insecticide class globally after glyphosate among herbicides, have drawn particular scrutiny for sub-lethal effects on bee foraging, navigation, and colony health, alongside bioaccumulation in stream organisms with sub-lethal effects on amphibians [18]. Soil biodiversity is similarly affected: pesticide residues were the second-most important driver, after soil physicochemical properties, of soil biodiversity patterns across hundreds of European sites, altering archaea, bacteria, fungi, protists, nematodes and arthropod communities and the functions they perform [14]. At the landscape level, cumulative biodiversity loss of pollinators, natural pest predators, earthworms, and aquatic invertebrates undermines the ecological processes on which sustainable agricultural production itself depends [17,33].

9. Effects on Climate Change

Pesticides contribute to climate change across their full life cycle — manufacture, transport, application, and post-application breakdown — while climate change itself is projected to increase pest, weed, and disease pressure and therefore pesticide demand, forming a self-reinforcing feedback loop [7,22 of climate cluster]. Because nearly all synthetic pesticides are derived from fossil-fuel feedstocks, their production is energy-intensive: manufacturing one kilogram of pesticide active ingredient has been estimated to require roughly ten times the energy needed to produce a kilogram of nitrogen fertilizer, and several individual pesticides carry a manufacturing carbon footprint exceeding 40 kilograms of CO2-equivalent per kilogram of product — for comparison, glyphosate manufacture alone has been estimated at approximately 31 kilograms of CO2-equivalent per kilogram [7,8].

Beyond manufacturing, fumigant pesticides can directly or indirectly increase soil nitrous oxide emissions — a greenhouse gas roughly 300 times more potent than carbon dioxide over a century timescale — by stimulating soil microbial processes, and some fumigants are themselves potent greenhouse gases [7]. Volatile organic compounds released from pesticide application also act as tropospheric ozone precursors, an additional short-lived climate pollutant [7]. Compounding this, disruption of soil microbial communities by pesticide residues reduces the soil's capacity to sequester carbon, undermining a key natural climate mitigation service [7,14].

Figure 2. The Pesticide-Climate Change Feedback Cycle



10.Effects on ENVIRONMENT

Across compartments, pesticide contamination reduces the capacity of ecosystems to deliver services such as pollination, natural pest control, nutrient cycling, and water purification [40]. Because air,

water and soil compartments are interconnected — atmospheric deposition returns residues to soil and water, run-off connects fields to aquatic systems, and leaching connects surface and groundwater — contamination in one compartment routinely propagates to others, producing environment-wide effects extending well beyond the treated field [13,17].

10.1 Air

Volatilization during and after application releases pesticide active ingredients and volatile organic compound (VOC) by-products into the atmosphere; these VOCs act as precursors to ground-level ozone, itself a potent greenhouse gas and respiratory irritant, while spray drift can carry residues far beyond the treated field, exposing rural populations and non-target ecosystems to airborne contamination [23,24 of climate cluster].

10.2 Water

Agricultural run-off and leaching are the dominant pathways of surface- and groundwater contamination; national river and stream surveys have detected one or more pesticide residues in the overwhelming majority of fish, surface-water, and aquifer samples tested, with compounds used for both agricultural and non-agricultural (e.g., lawn/garden) purposes commonly found [28]. Contaminated water threatens aquatic food webs and can compromise drinking-water quality where treatment infrastructure is inadequate [13].

10.3 Soil

Soil is both a reservoir and a transformation medium for pesticides. A continental-scale European survey examining sixty-three pesticides across 373 sites found residues in 70% of sampled sites, with pesticide load emerging as the second-strongest driver of soil biodiversity patterns after inherent soil properties; effects included suppression of beneficial taxa such as arbuscular mycorrhizal fungi and bacterivorous nematodes, and disruption of phosphorus and nitrogen cycling functions [14,38]. Combined applications of multiple pesticides have been shown to have compounding negative effects on key soil processes, including nutrient cycling and aggregate formation, beyond what any single compound would cause alone [17]. Because soil microbial communities underpin decomposition, nutrient cycling and carbon storage, their disruption has cascading consequences for long-term soil fertility and agricultural sustainability [15,16].

Table 3. Environmental Components Effects

Component	Contamination Route	Consequences
Air	Spray drift, volatilization, VOC emission	Ground-level ozone precursor; off-target deposition; inhalation exposure of rural populations
Surface water	Run-off, spray drift onto water bodies	Residues detected in most rivers/streams surveyed; toxicity to fish and invertebrates; food-web disruption
Groundwater	Leaching through soil profile	Contamination of drinking-water aquifers, especially with soluble compounds

Soil	Direct deposition, residue persistence, sediment binding	Suppressed microbial diversity and function; impaired nutrient cycling; reduced carbon sequestration capacity

11. Regulations and Mitigation Strategies

11.1 International Regulatory Frameworks

The Stockholm Convention globally bans the production, use, and trade of toxic, persistent, and bioaccumulative chemicals, subject to limited exemptions. The Rotterdam Convention provides an information-sharing framework to help countries manage hazardous chemical imports. Complementing these, the FAO/WHO Code of Conduct and Joint Meeting on Pesticide Residues set international residue limits and establish pesticide management guidance.(21,23)

11.2 Domestic Legislative Frameworks

National pesticide usage is generally regulated by specific legislation requiring mandatory registration of all active ingredients and formulations before manufacturing, sale, or import, alongside establishing technical bodies for quality control and enforcement. These laws empower a central technical committee to restrict or ban toxic chemicals, leading to the gradual phase-out of many hazardous products. Persistent legacy compounds like DDT are restricted to narrow public-health exemptions, such as vector control, in line with the Stockholm Convention while safer alternatives are scaled up. However, legal reviews identify frequent implementation hurdles, including fragmented regulatory jurisdictions, slow review cycles, and discrepancies between hazard identification and active restriction, prompting calls for modernized frameworks aligned with precautionary principles and international treaties(19-22).

11.3 Mitigation and Sustainable Alternatives

Recommended mitigation strategies converge on several complementary approaches:

- Integrated Pest Management (IPM): combining biological control, crop rotation, resistant cultivars, and judicious, need-based chemical application to reduce total pesticide load.
- Biopesticides and botanical alternatives: microbial agents, neem-based formulations, and plant-growth-promoting rhizobacteria that degrade or substitute for synthetic residues while supporting soil health [16,37].
- Precision application technologies: electrostatic and targeted sprayers that reduce drift and per-hectare active-ingredient use, lowering both exposure and the energy/carbon footprint of application [8].
- Residue monitoring and stewardship: Routine surveillance of food, water, and soil residues to enforce maximum residue limits and identify emerging contamination hotspots [1].
- Nanotechnology-enabled formulations: nanocarriers designed to improve targeting efficiency and reduce the total quantity of active ingredient required, an area of active research as a lower-impact alternative to conventional pesticide delivery [16].

- Harmonized hazard-based regulation: aligning domestic registration criteria with the Stockholm and Rotterdam Conventions and the FAO Code of Conduct, and phasing out highly hazardous pesticides on a fixed timeline [21,23].

12.CONCLUSION:

Thus, though, the pesticides have a major role in impacting the quantity of harvest of the farming whose need is increasing day by day due tonthe increasing need because of the rapidly growing population, ultimately the measures raken to increase the quantity has led to the drastic reduction in its quality, to the extent thst overconsumption, might even led to health hazardous effects.They remain indispensable to contemporary crop protection, yet the accumulated evidence reviewed here demonstrates that their benefits are inseparable from measurable costs to human health, plant physiology and nutritional quality, terrestrial and aquatic animal life, and the integrity of air, water, soil, and biodiversity.

Their contribution to greenhouse gas emissions across the full production-to-degradation life cycle further embeds pesticide dependence within the climate crisis, creating a feedback loop in which a warming climate is projected to intensify pest pressure and, in turn, pesticide demand. Addressing these interlinked challenges will require coordinated action across three fronts: strengthened, hazard-based and harmonized regulation consistent with international conventions; accelerated adoption of integrated pest management, biopesticides, and precision application technologies; and sustained investment in residue surveillance and toxicological research to keep pace with newly introduced active ingredients. A transition toward more judicious, evidence-based pesticide use — rather than either unconditional reliance or wholesale elimination — offers the most realistic pathway to reconciling food security with human and environmental health.

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COMPONENT-CONTAMINANT-MATRIX:

Planetary Component	Pollutant #1	Pollutant #2	Pollutant #3	Pollutant #4	Pollutant #5	Other Prevalent Pollutants
AIR / ATMOSPHERE	**PM2.5** **Prevalence:** Among the two highestmeasured pollutants (~50% share of exceedances). **Main source:** Industry >50%, vehicles 27%, crop/biomass burning 17%, domestic cooking 13-15%; over half of PM2.5 mass forms secondarily from SO2 + ammonia reactions. **Permissible limit:** CPCB NAAQS: 40 µg/m³ annual, 60 µg/m³ 24-hr. WHO AQG 2021: 5 µg/m³ annual, 15 µg/m³ 24hr **Also contaminates:** Into soil and water via atmospheric fallout; infiltrates indoor air.	**PM10** **Prevalence:** Highestmeasured pollutant nationally (~30-50%) **Main source:** Road dust and construction/demolition activity (>26%), biomass burning (20%); Industrial (~20%). **Permissible limit:** CPCB NAAQS: 60 µg/m³ annual, 100 µg/m³ 24-hr. WHO AQG 2021: 15 µg/m³ annual, 45 µg/m³ 24hr **Also contaminates:** Settles on soil, water bodies and plant surfaces.	**NO2** **Prevalence:** Relatively stable to rising trend; (~22-25%) **Main source:** Industrial sector (5055%), transportation (30-35%), Agriculture & Biomass burning (10-15%) **Permissible limit:** CPCB NAAQS: 30-40 µg/m³ annual, 80 µg/m³ 24-hr. WHO AQG: 10 µg/m³ annual, 10-25 µg/m³ 24hr **Also contaminates:** Contributes to acid rain (soil/water acidification) and secondary PM2.5/ozone formation.	**SO2** **Prevalence:** Declining national trend, but persistent hotspots near coal plants (~34%) **Main source:** Coalbased thermal power plants (60-70%), Industries & Smelting (20-25%), Transportation & Offroad Marine (5-8%) **Permissible limit:** CPCB NAAQS: 50 µg/m³ annual (20 µg/m³ eco-sensitive), 80 µg/m³ 24-hr. WHO AQG: 40 µg/m³ 24-hr **Also contaminates:** Causes acid rain affecting soil/water chemistry; reacts with ammonia to form secondary PM2.5 (ammonium sulfate).	**O3** **Prevalence:** Rising summer-season levels in urban/periurban India, (~15-40%) **Main source:** Photochemical reaction of NOx + VOCs under sunlight — traffic and industrial precursor emissions. **Permissible limit:** CPCB NAAQS: 100 µg/m³ 8-hr. WHO AQG: 100 µg/m³ 8-hr **Also contaminates:** Damages crops/vegetation (yield loss), degrades rubber and building materials.	• **Carbon Monoxide (CO) (combustion gas)** — vehicular/industrial exhaust • **Ammonia (NH3) (gaseous/agrochemical)** — fertilizer use, livestock • **Benzene (VOC, carcinogen class)** — vehicle exhaust, fuel evaporation • **Lead (Pb)** (heavy metal, airborne) — battery recycling, some industry • **Black Carbon (soot)** (carbonaceous particulate) — biomass/diesel combustion
WATER	**Sewage derived organic & pathogenic load** **Prevalence:** 75-80% of samples exceed the safe limit. **Main source:** Untreated urban sewage (45-50%), industrial effluent (~40%), Agricultural/Animal. **Permissible limit:** BIS 10500:2012 & WHO guideline: 0 (Nondetectable) **Also contaminates:** Surrounding soil, plants, air quality.	**Nitrate (NO_3^-)** **Prevalence:** 19.8–20% of samples exceed the safe limit. **Main source:** Nitrogenous fertilizers (75-80%), septic-tank/sewage leakage (20-40%), Soil erosion (15-20%). **Permissible limit:** BIS 10500:2012 — 45 mg/L. WHO guideline: 50 mg/L as NO3 **Also contaminates:** Leaches into surface water causing eutrophication; accumulates in soil.	**Fluoride (F^-)** **Prevalence:** 9.04% of samples exceed the safe limit. **Main source:** Natural rock–water interaction with fluoride-bearing minerals (geogenic), intensified by groundwater overextraction. **Permissible limit:** BIS 10500:2012 — acceptable 1.0 mg/L, permissible 1.5 mg/L. WHO guideline: 1.5 mg/L **Also contaminates:** Absorbed into soil and taken up by crops in high-fluoride zones.	**Arsenic (As)** **Prevalence:** 3.55% of samples exceed the safe limit; concentrations up to ~200 µg/L reported in parts of Bihar and Uttar Pradesh. **Main source:** Geogenic release from alluvial aquifer sediments (Indo-Gangetic/Brahmaputra plains) **Permissible limit:** BIS 10500:2012 — 0.01 mg/L / 10 µg/L. WHO guideline: 10 µg/L **Also contaminates:** Accumulates in irrigated paddy soil and rice grain (notably West Bengal); enters surface water.	**Uranium (U)** **Prevalence:** 6.60% of samples exceed the safe limit, concentrated in Punjab (~42% of affected samples) and Rajasthan (~30%), plus Haryana, Gujarat, Telangana. **Main source:** Geogenic release from phosphatic rock formations, compounded by phosphate-fertilizer use and falling water tables from overextraction. **Permissible limit:** AERB/BIS provisional guidance — 30 µg/L. WHO guideline: 30 µg/L **Also contaminates:** Largely confined to groundwater; localized soil enrichment near source rock.	• **Chloride & Salinity (EC)** (inorganic salts) — coastal intrusion, overextraction • **Heavy metals** — Pb, Cd, Cr, Hg — unregulated industrial discharge • **Pharmaceutical & pesticide residues** (emerging contaminants) — agricultural runoff, effluent discharge

SOIL	**Heavy Metals (Cd, Pb, Cr, As, Hg)** **Prevalence:** ~31% of India's agricultural land affected by soil degradation (CPCB, 2022-23); **Main source:** Untreated industrial effluent & sludge discharge (~40-45%), sewage/wastewater irrigation (~25-30%), mining & smelting activity (~20%). **Permissible limit:** CPCB uses site-specific screening levels. Commonly cited Indian agri-soil limits: Cd 3-6 mg/kg, Pb 250-500 mg/kg. WHO/FAO agricultural guideline: Cd 3 mg/kg, Pb 100 mg/kg. **Also contaminates:** Leaches into groundwater; absorbed by crops entering the food chain; resuspended as airborne dust.	**Pesticide Residues (organochlorines/organophosphates)** **Prevalence:** Residues detected in ~60% of vegetable samples across Indian cities (IITR study). **Main source:** Direct/overuse in agriculture (majority share), surface runoff accumulation, atmospheric spray drift. **Permissible limit:** India — FSSAI/Insecticides Act sets crop-level residue tolerances (no universal soil limit). WHO/Codex — similarly crop-specific MRLs; no universal soil threshold exists internationally. **Also contaminates:** Leaches into groundwater; absorbed by soil fauna (earthworms); bioaccumulates up the food chain.	**Plastics & Microplastics** **Prevalence:** widespread but largely unquantified. India generates an estimated 26,000+ tonnes/day of plastic waste (CPCB), a large share entering soil via dumping. **Main source:** Agricultural mulch/greenhouse film, urban solid-waste dumping & landfill leachate, sewage sludge applied as fertilizer. **Permissible limit:** No India or WHO permissible soil limit yet established (emerging/unregulated contaminant). **Also contaminates:** Leaches into groundwater; ingested by soil organisms/livestock; taken up by food crops.	**Excess Nitrogen & Phosphate (fertilizer residue)** **Prevalence:** Contributes to the ~31% of agricultural land affected by soil degradation nationally (CPCB, 2022-23). **Main source:** Over-application of chemical N-P-K fertilizers (dominant), livestock manure, sewage-sludge/wastewater irrigation. **Permissible limit:** No BIS/WHO universal soil-nutrient contamination limit; India uses Soil Health Card advisories for optimum levels. **Also contaminates:** Runs off into surface water causing eutrophication; leaches into groundwater as nitrate.	**E-waste-derived Heavy Metals & POPs** **Prevalence:** India generates ~1.75 million tonnes of e-waste annually (MoEFCC) **Main source:** Informal e-waste dismantling/recycling (dominant at hotspots), improper landfilling, electronics-manufacturing waste. **Permissible limit:** No dedicated soil standard; E-Waste (Management) Rules, 2022 regulate handling, not soil concentration. WHO has no specific soil guideline either. **Also contaminates:** Groundwater near dumpsites; airborne dioxins from informal burning; local food-chain bioaccumulation.	• **Radioactive/NORM residues** (naturally occurring radioactive material) — mining tailings, phosphate fertilizer processing • **Soil salinity & waterlogging** — over-irrigation, poor drainage • **Petroleum hydrocarbons** — oil spills, fuel storage leaks • **Coal fly ash** — thermal power plant ash pond disposal
CLIMATE	**Carbon Dioxide (CO_2)** **Prevalence:** Largest share of India's GHG portfolio; India ~8.2% of global GHG emissions among the largest absolute emitters. **Main source:** Power generation/coal combustion (~44-45%), Industry (24-31%), Transport (~12%). **Target (India NDC/IPCC):** India's NDC — 45% cut in emissions intensity by 2030 (vs 2005) & net-zero by 2070. IPCC 1.5°C pathway requires global CO_2 to reach net-zero by ~2050. **Also contaminates:** Drives ocean acidification; raises global temperatures affecting all ecosystems.	**Methane (CH_4)** **Prevalence:** ~16% of global GHG total (2024), mostly agriculture/waste; part of India's ~8% AFOLU + 4% waste-sector share. **Main source:** Livestock enteric fermentation & paddy cultivation (agriculture), municipal solid-waste landfills, coal-mining/fossil-fuel leakage. **Target:** Global Methane Pledge (COP26) — 30% cut in global methane by 2030 vs 2020 (India pursues efficiency measures via its NDC). **Also contaminates:** Precursor to ground-level ozone, harming air quality and crop yields.	**Nitrous Oxide (N_2O)** **Prevalence:** Rising with fertilizer use; part of India's ~8% AFOLU emissions share. **Main source:** Nitrogenous fertilizer application (dominant), livestock manure management, industrial/combustion sources. **Target:** No India-specific N_2O ceiling; IPCC AR6 flags N_2O reduction as necessary for 1.5°C pathways; addressed indirectly via fertilizer-efficiency policy. **Also contaminates:** Depletes stratospheric ozone; leaches into water as nitrate.	**Black Carbon (Short-Lived Climate Pollutant)** **Prevalence:** (15-20%)driven by biomass burning and diesel use. **Main source:** Biomass/crop-residue burning (10-12%), diesel vehicle(15-20%) exhaust, residential solid-fuel cooking (45-50%). **Target:** No binding numeric limit; addressed under the Climate & Clean Air Coalition's voluntary SLCP goals and India's National Clean Air Programme (20-30% PM2.5 cut by 2026 vs 2017, indirectly targeting black carbon). **Also contaminates:** Deposits on Himalayan glaciers	**Hydrofluorocarbons (HFCs)** **Prevalence:** Fast-growing due to rising AC/refrigeration demand; small share of total GHG mass but very high per-tonne global warming potential. **Main source:** Refrigeration & air-conditioning (dominant), foam-blowing industry, aerosol propellants. **Target:** Kigali Amendment to the Montreal Protocol (ratified by India, 2021) — freeze HFC consumption by 2028, cut 85% by 2047 from baseline. **Also contaminates:** Purely atmospheric impact; no direct soil/water pathway.	• Sulfate aerosols — cooling effect but damages air quality/health; coal/industry-derived • Tropospheric ozone — short-lived climate pollutant & air/crop pollutant • Perfluorocarbons (PFCs) — aluminium smelting industry • Land-use/irrigation-driven water vapour feedback — large-scale irrigation & deforestation

ANIMALS (Wildlife & Ecosystems)	**Veterinary Pharmaceuticals (Diclofenac & NSAIDs)** **Prevalence:** Caused a 95-99% collapse in South Asian Gyps vulture populations since the 1990s (peer-reviewed ecotoxicology studies). **Main source:** Veterinary treatment of livestock whose carcasses are scavenged (dominant), illegal diversion of human-formulation diclofenac, cross-toxicity from substitute NSAIDs (aceclofenac, ketoprofen). **Permissible limit:** India banned veterinary diclofenac (2006); zero-residue is the	**Organochlorine Pesticides (DDT, HCH, Endosulfan)** **Prevalence:** Detected in nearly all sampled tissues of Indian raptors/vultures across multiple studies — confirmed nationwide bioaccumulation. **Main source:** Ongoing agricultural use/runoff — DDT is still used for vector control (dominant), industrial discharge, long-range atmospheric drift. **Permissible limit:** India bans DDT for agriculture (retained only for malaria vector control). WHO/Stockholm Convention lists DDT, HCH and endosulfan as Persistent Organic	**Heavy Metals (Hg, Pb, Cd) in Wildlife Tissue** **Prevalence:** Detected in liver/kidney tissue of Indian vultures and other wildlife; generally sub-acute but rising near industrial belts. **Main source:** Industrial effluent discharge into water bodies (dominant), mining runoff, ingestion of lead ammunition/fishing weights. **Permissible limit:** No India-specific wildlife-tissue standard; researchers reference international ecotox benchmarks (e.g., liver Pb >2 µg/g wet-wt flagged as elevated exposure).	**Plastic & Marine Debris Ingestion** **Prevalence:** Widespread in coastal/marine wildlife (turtles, seabirds, fish); the Ganga is documented among the largest global river contributors of ocean plastic. **Main source:** Mismanaged urban/coastal solid waste (dominant), fishing gear ("ghost nets"), riverine plastic transport to the sea. **Permissible limit:** No permissible-limit framework exists for ingested plastic load in wildlife — an unregulated, emerging issue in both India and internationally. **Also contaminates:** Microplastics enter	**Endocrine-Disrupting Chemicals (Industrial POPs)** **Prevalence:** Detected in some Indian riverine fish/bird studies; nationwide monitoring remains limited. **Main source:** Industrial effluent — dyes, plasticizers (dominant), pesticide runoff, untreated sewage discharge. **Permissible limit:** No India-specific ecotoxicological limit; Stockholm Convention POPs list guides restriction of specific chemicals (PCBs, certain phthalates). **Also contaminates:** Affects reproductive/hormonal systems across	• Cyanide/mining effluent — fish kills near mining zones • Noise & light pollution — behavioural disruption to wildlife • Antibiotic-resistance genes — livestock waste runoff • Oil spills — coastal/marine fauna impact
	vulture-safe standard. No separate WHO wildlife-exposure limit exists (WHO does not set ecotoxicological standards). **Also contaminates:** Enters soil/water via carcass decomposition; affects other scavengers (kites, crows).	Pollutants (POPs) for elimination. **Also contaminates:** Bioaccumulates in soil, fish, milk and human tissue.	**Also contaminates:** Biomagnifies up the food chain to apex predators and humans.	the marine food chain, eventually reaching seafood consumed by humans.	species; enters water and soil.	

PLANTS (Crops & Vegetation)	**Ground-level Ozone (O3)** **Prevalence:** India projected to suffer crop-yield loss of up to 20% by 2030 from ozone; some densely-populated states already show ~50% relative yield loss for specific crops. **Main source:** Photochemical reaction of vehicular NOx + VOCs (transport, dominant precursor), industrial precursor emissions, biomass-burning precursors. **Permissible limit:** No India-specific crop-protective ozone standard (NAAQS 100 µg/m³ 8-hr is health-based). WHO AQG (100 µg/m³ 8-hr) is also health-based — actual crop-damage threshold is far lower (~40 ppbv AOT40). **Also contaminates:** Also a major air/human-health pollutant (see AIR table); damages forests and natural vegetation.	**Heavy Metals (Cd, Pb, As, Cr) via Soil/Irrigation** **Prevalence:** Elevated levels reported in vegetables across Indian industrial-belt and sewage-irrigated farmland (multiple regional studies). **Main source:** Irrigation with untreated/industrial wastewater (dominant near urban-industrial belts), contaminated-soil root uptake, atmospheric deposition on leaves. **Permissible limit:** FSSAI food-crop limits — e.g., Pb ~2.5 mg/kg, Cd ~0.2 mg/kg in vegetables. Codex/WHO — broadly comparable (Pb 0.1-0.3 mg/kg, Cd 0.05-0.2 mg/kg depending on vegetable). **Also contaminates:** Enters the human food chain directly; disrupts soil microbiota.	**Fluoride** **Prevalence:** Documented uptake in tea, vegetables and cereal crops across fluoride-endemic groundwater zones (Rajasthan, Gujarat, Andhra Pradesh). **Main source:** Irrigation with fluoride-contaminated groundwater (dominant in endemic belts), industrial emissions (brick kilns, aluminium smelting) settling on foliage, geogenic soil fluoride. **Permissible limit:** No dedicated India/WHO plant-tissue fluoride limit; drinking/irrigation-water limits (BIS 1.5 mg/L, WHO 1.5 mg/L) are used as the indirect benchmark. **Also contaminates:** Passes into the food chain (tea leaves accumulate fluoride strongly); affects livestock grazing on contaminated fodder.	**Acid Deposition (SO2/NOx-derived)** **Prevalence:** Localized crop/forest damage reported near thermal-power-plant clusters and industrial belts (e.g., Singrauli, Korba). **Main source:** Coal-based thermal power plants (dominant), industrial smelting, vehicular NOx. **Permissible limit:** No dedicated vegetation-protection standard; CPCB NAAQS for SO2 (50 µg/m³ annual) and NO2 (30-40 µg/m³ annual) are health-based, not crop-based; WHO limits are similarly health-focused. **Also contaminates:** Acidifies soil and surface water, indirectly stressing root systems.	**Pesticide Residues (direct application)** **Prevalence:** Residues found in ~60% of vegetable/fruit samples tested across Indian city surveys (IITR). **Main source:** Direct crop spraying/over-application (dominant), drift onto neighbouring non-target plants, contaminated irrigation water. **Permissible limit:** FSSAI MRLs vary by pesticide-crop combination (Insecticides Act 1968 & FSS Contaminants Regulations 2011). Codex/WHO MRLs serve as the international reference, often stricter for export crops. **Also contaminates:** Residue passes to consumers via food; harms pollinator insects (bees).	• Excess nitrogen deposition — canopy nutrient imbalance from vehicular/industrial NOx • Herbicide drift — damages non-target crops • Industrial dust/soot deposition — reduces photosynthesis • Microplastic uptake — via contaminated soil/irrigation water (emerging area)

HUMANS (Public Health)	**PM2.5 / Ambient Air Pollution Exposure** **Prevalence:** India records among the world's highest ambient PM2.5 exposure levels; linked to well over a million deaths annually across Global Burden of Disease-style estimates. **Main source:** Industry & vehicles (see AIR table for %), biomass/crop burning, domestic solid-fuel cooking. **Permissible limit:** WHO AQG 2021 (5 µg/m³ annual) & CPCB NAAQS (40 µg/m³ annual) exposure limits; no blood biomarker exists — health-effect thresholds are used instead.	**Lead (Pb)** **Prevalence:** ~275 million Indian children have blood lead levels ≥5 µg/dL, the WHO intervention threshold (Pure Earth/UNICEF, 2020); pooled mean BLL in Indian children ~10.4 µg/dL (ICMR-led meta-analysis, 2025). **Main source:** Informal lead-acid battery recycling/repair (dominant in hotspot states like Bihar/Jharkhand), lead-adulterated spices (e.g., turmeric with lead chromate), industrial emissions/lead paint & pottery. **Permissible limit:** WHO intervention/reference level — 5 µg/dL	**Arsenic** **Prevalence:** Millions exposed via contaminated drinking water in Ganga-Brahmaputra floodplain states; concentrations up to ~200 µg/L reported in parts of Bihar/UP (20x the safe limit). **Main source:** Contaminated groundwater consumption (dominant), food grown with arsenic-affected irrigation water (notably rice), minor occupational exposure. **Permissible limit:** BIS/WHO drinking-water limit — 10 µg/L (0.01 mg/L). No standardized Indian blood/urine biomonitoring limit, though urinary arsenic >100 µg/L is used internationally	**Fluoride** **Prevalence:** Dental/skeletal fluorosis is endemic across 20+ Indian states; historically tens of millions estimated affected (Rajasthan, Gujarat, AP, Telangana worst-hit). **Main source:** Fluoride-contaminated groundwater consumption (dominant), fluoride-rich food/tea in endemic zones, industrial exposure (aluminium, phosphate-fertilizer workers). **Permissible limit:** BIS 1.5 mg/L, WHO 1.5 mg/L (drinking water). No routine blood/urine standard — diagnosis relies on	**Pesticide Residues (occupational + dietary)** **Prevalence:** Detected in blood/adipose-tissue samples in multiple Indian biomonitoring studies, particularly among farming communities; also reported in some regional breast-milk samples. **Main source:** Occupational exposure during spraying (farmers/agricultural workers, dominant), dietary intake via treated produce, residential household-pesticide use. **Permissible limit:** WHO/FAO set Acceptable Daily Intake (ADI) values per pesticide; India's	• Mercury — dental amalgam, fish consumption, artisanal gold-mining zones • Endocrine disruptors (phthalates, BPA) — food packaging, plastics • Radon — indoor exposure, geology-dependent • Cadmium via tobacco smoking — direct inhalation exposure
	Also contaminates: Same sources contaminate soil/water via atmospheric fallout (see AIR table).	blood lead (no level considered fully "safe"). India has no distinct national blood-lead action level; follows WHO guidance via ICMR. **Also contaminates:** Same sources contaminate soil (battery-recycling sites) and food (spices)	as an elevated-exposure marker. **Also contaminates:** Same groundwater sources; rice-grain accumulation (West Bengal).	clinical/skeletal fluorosis criteria. **Also contaminates:** Same groundwater/food sources as fluoride in water and plants.	FSSAI regulates via crop-level MRLs rather than a blood-level standard. **Also contaminates:** Same agricultural/food/soil pathways as pesticide contamination elsewhere in this matrix.	

FOOD (Diet & Supply Chain)	**Pesticide Residues** **Prevalence:** (IITR: ~60%); repeatedly flagged in spices/produce in FSSAI surveillance. **Main source:** Excess/improper agricultural application (dominant), post-harvest treatment, contaminated irrigation water. **Permissible limit:** FSSAI (FSS Contaminants, Toxins & Residues Regulations, 2011) sets crop-pesticide-specific MRLs. Codex Alimentarius/WHO MRLs are the international reference, sometimes stricter for export markets. **Also contaminates:** Soil, groundwater, and human tissue via dietary intake.	**Heavy Metals (Pb, Cd, As, Hg) in Food** **Prevalence:** Lead flagged repeatedly in spices (turmeric adulterated with lead chromate); arsenic accumulation documented in rice from contaminated-irrigation belts. **Main source:** Crop uptake from contaminated soil/irrigation water (dominant), deliberate adulteration for colour/weight (lead chromate in turmeric), industrial contamination of aquatic food (fish). **Permissible limit:** FSSAI sets limits by food category (e.g., Pb ~2.5 mg/kg in most foods/spices; As ~1.1 mg/kg in rice). Codex/WHO sets comparable but sometimes stricter limits (Codex rice-arsenic limit: 0.2 mg/kg). **Also contaminates:** Same soil/water contamination sources feed back into food.	**Aflatoxins & Mycotoxins** **Prevalence:** Frequently detected above safe limits in stored grains, groundnuts and spices in Indian market surveys, especially under poor storage/humidity. **Main source:** Fungal (Aspergillus) growth during improper storage/humidity (dominant), pre-harvest field contamination in drought-stressed crops, inadequate drying/warehousing infrastructure. **Permissible limit:** FSSAI — Aflatoxin B1 ~30 µg/kg (groundnuts/others), total aflatoxins 15-20 µg/kg in various commodities. Codex/WHO — Aflatoxin M1 (milk) 0.5 µg/kg; broadly comparable with some commodity-specific variance. **Also contaminates:** Contaminates animal feed (passing into meat/milk); spreads via soil-borne fungal spores.	**Veterinary Drug & Antibiotic Residues** **Prevalence:** Nitrofuran metabolites detected in poultry eggs (Dec 2025 surveillance); repeated FSSAI findings of antibiotic residues in poultry, dairy and aquaculture products. **Main source:** Illegal/excess antibiotic use in poultry & livestock farming (dominant), aquaculture antibiotic use, non-compliance with pre-slaughter/milking withdrawal periods. **Permissible limit:** FSSAI sets per-drug, per-commodity residue limits (e.g., Chlortetracycline 0.1 mg/kg in milk); nitrofurans are banned outright (zero tolerance). Codex/WHO sets comparable MRLs; several antibiotics are banned globally in food animals. **Also contaminates:** Drives antimicrobial resistance in humans and the environment via livestock-waste runoff into soil/water.	**Food Adulterants (synthetic dyes, starch, urea, detergent)** **Prevalence:** FSSAI's Pan-India milk survey (2023) found ~4% of 10,126 samples adulterated with starch/salt/foreign fat/sucrose; regional surveys show far higher local failure rates (e.g., ~47% of Punjab dairy samples failed over 2022-25). **Main source:** Economically-motivated dilution/adulteration in the unorganized dairy/spice supply chain (dominant), industrial dyes (metanil yellow, Sudan dyes) in spices, synthetic substitutes in oils/ghee. **Permissible limit:** FSSAI sets zero-tolerance for adulterants like detergent, excess starch, and banned industrial dyes under the FSS Act, 2006. Codex/WHO similarly prohibit non-food-grade additives and industrial dyes in food. **Also contaminates:** Adulterant disposal/runoff can locally affect soil and water near processing units.	• Packaging-derived microplastics & phthalates — leaching from plastic packaging/storage • Nitrate/nitrite — cured/processed meats and leafy vegetables • Trans fats & reused cooking oils — repeated deep-frying oil reuse • Radioactive contamination — rare, near mining/industrial zones

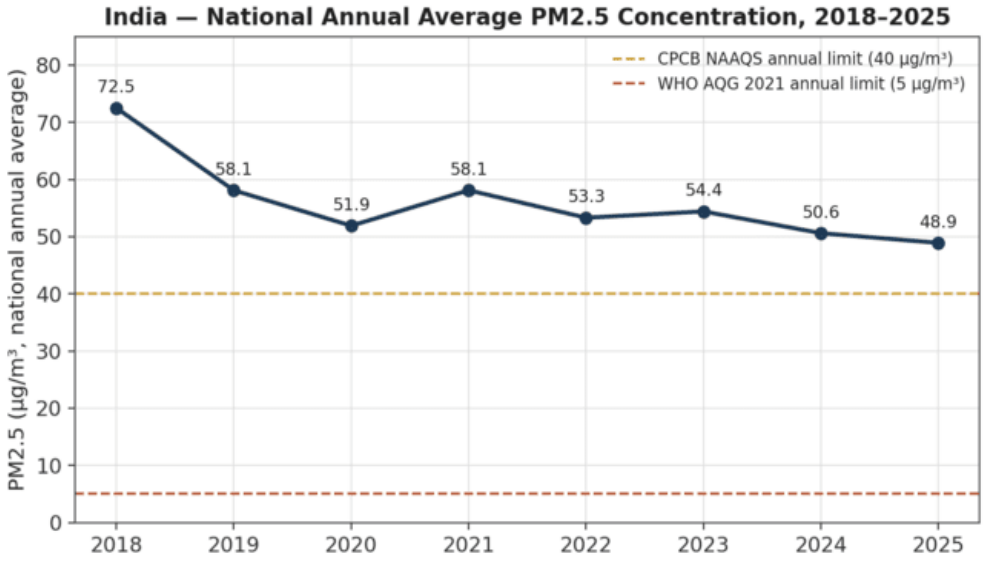
TREND ANALYSIS

1)Major Air Pollutants

Trends of the Five Major Air Pollutants in India (Past Decade)

1 PM2.5 (Fine Particulate Matter)

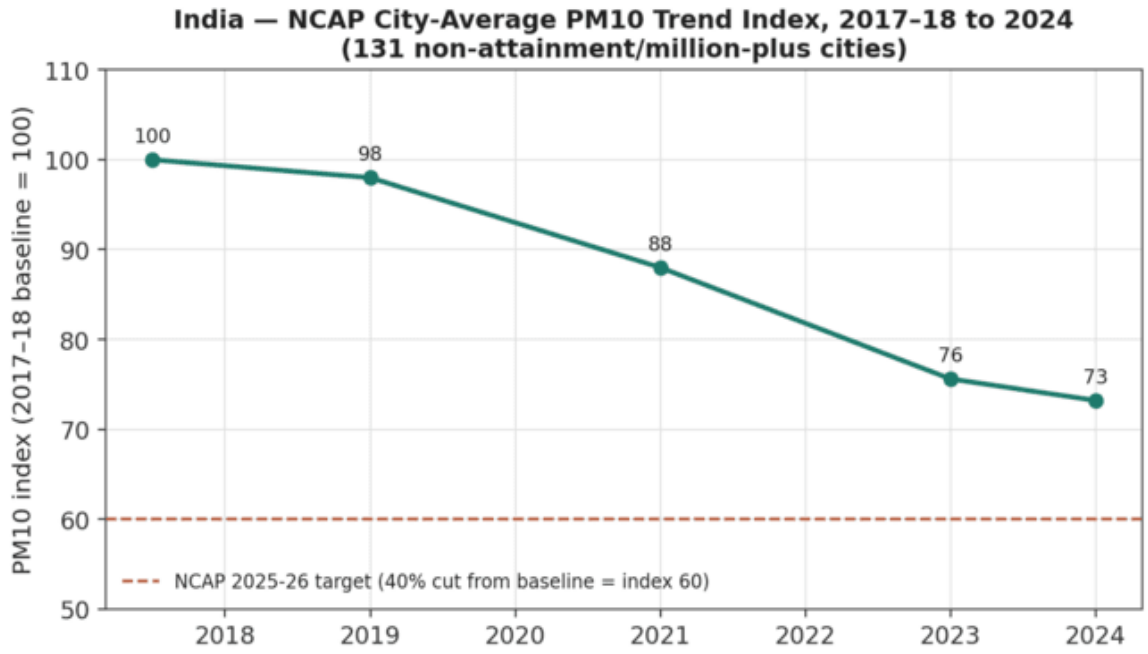
PM2.5 is consistently the pollutant with the highest share of NAAQS exceedances in India. The national annual average, as tracked by IQAir's World Air Quality Reports (compiled from thousands of government and independent monitoring stations), fell sharply during the 2020 COVID-19 lockdowns before rebounding, and has shown a general — though uneven — decline since 2021.



2 PM10 (Coarse Particulate Matter)

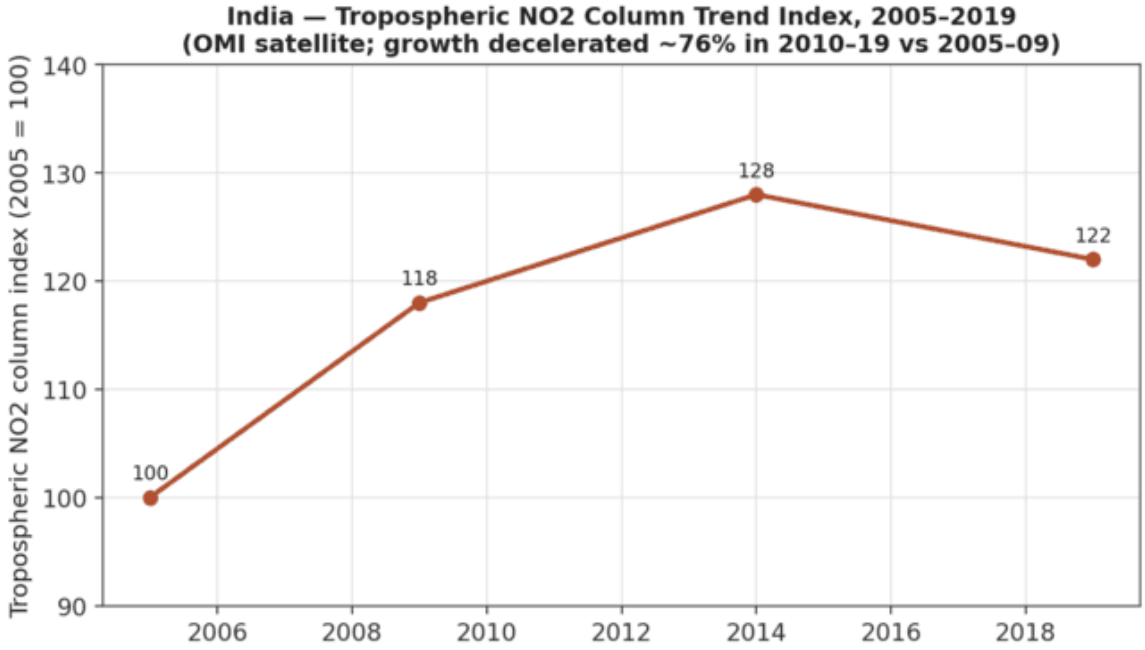
PM10 is the pollutant with the highest overall measured prevalence nationally and is the principal metric tracked under the National Clean Air Programme (NCAP), launched by the Ministry of Environment, Forest and Climate Change (MoEFCC) in January 2019 with a 2017–18 baseline. The original target was a 20–30% reduction by 2024; this was revised in 2022 to a 40% reduction (or attainment of the 60 µg/m³ NAAQS) by 2025–26.

- India achieved a reported 26.84% nationwide reduction in particulate matter levels between 2019 and 2024, with NCAP cities collectively showing a 24.45% improvement.



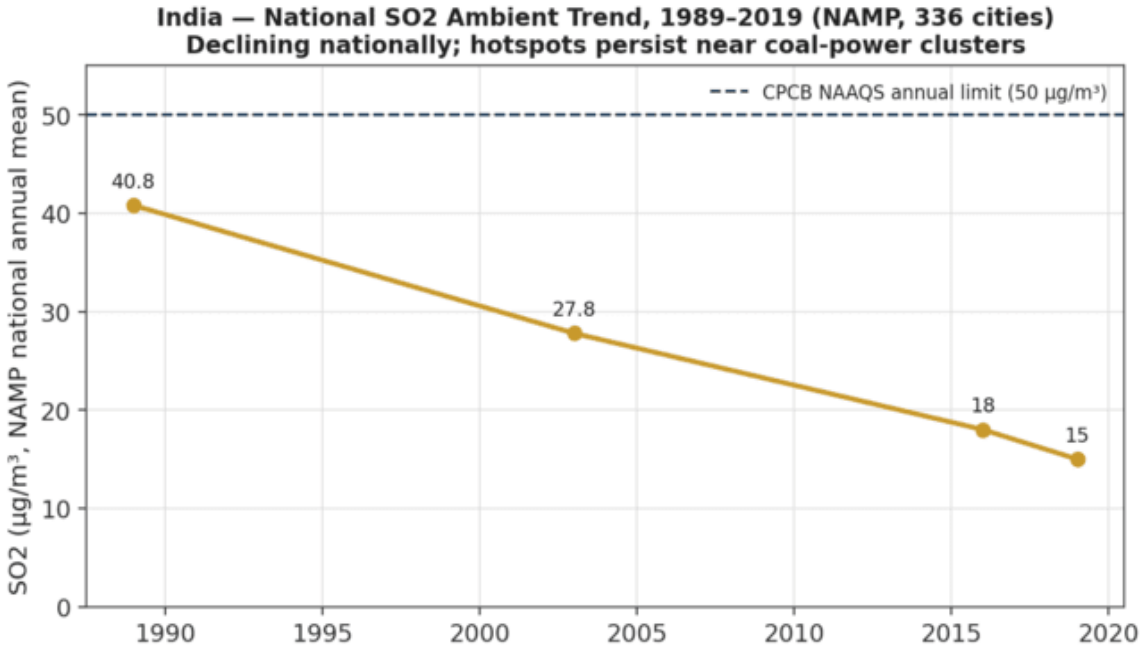
3 Nitrogen Dioxide (NO2)

Ground-based CPCB monitoring (CAAQM/NAMP) found NO2 generally meeting the NAAQS nationally with no statistically significant five-year trend over 2015–2019. However, satellite-derived tropospheric NO2 column data present a longer and more sensitive record: a 2023 peer-reviewed analysis of OMI satellite data (2005–2019) found tropospheric NO2 rising 12.5–29.6% across most of India between 2005 and the early 2010s, with the rate of increase decelerating by roughly 76% in 2010–2019 relative to 2005–2009 — indicating a plateauing rather than a reversal.



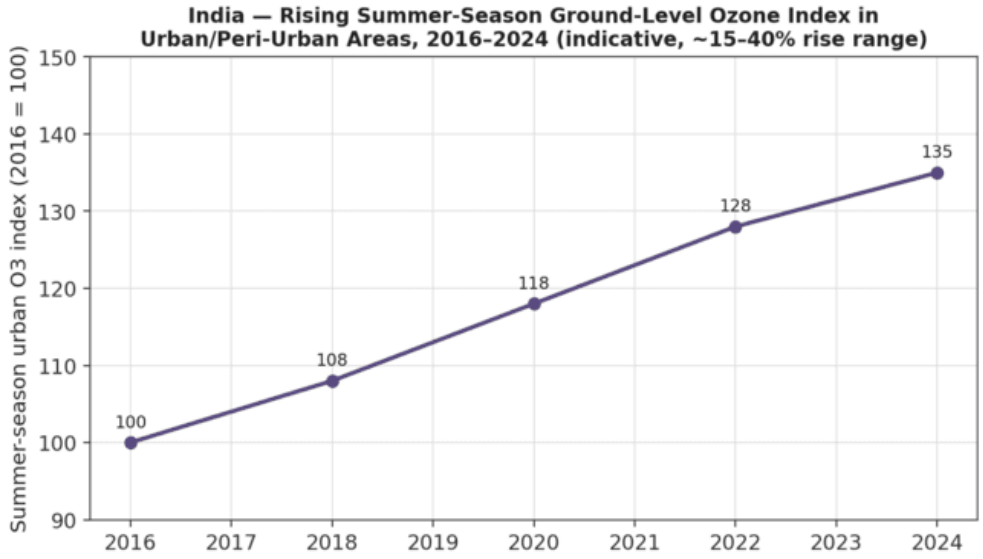
4 Sulphur Dioxide (SO2)

SO2 shows the clearest long-run decline of the five pollutants at the national scale, driven by cleaner fuel standards and (more recently) flue-gas desulfurization mandates at coal-fired power plants, even though localized hotspots persist near thermal power and smelting clusters.



5 Ground-Level Ozone (O3)

O3 is a secondary pollutant formed photochemically from NOx and VOC precursors under sunlight, and is the only one of the five pollutants that CPCB and independent researchers report as rising in urban and peri-urban India, particularly during the summer season, even while its 8-hour NAAQS is still generally met on an annual-average basis.

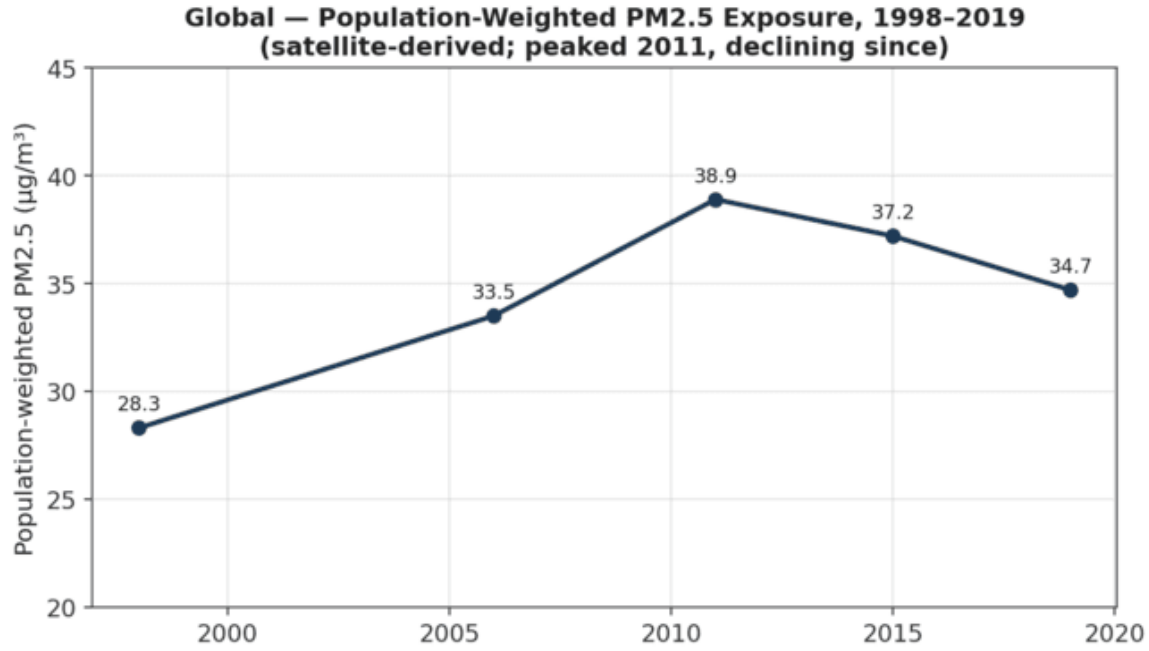


Global Multi-Decadal Trends

This section places the same five pollutants in a global context across roughly three decades: the mid-1990s, mid-2000s, mid-2010s, and the present decade. Because no single international agency publishes one unbroken 30-year global average for every pollutant, each subsection draws on the longest-running peer-reviewed satellite or emissions-inventory record available for that specific pollutant, with the exact reported years and sources stated under each chart.

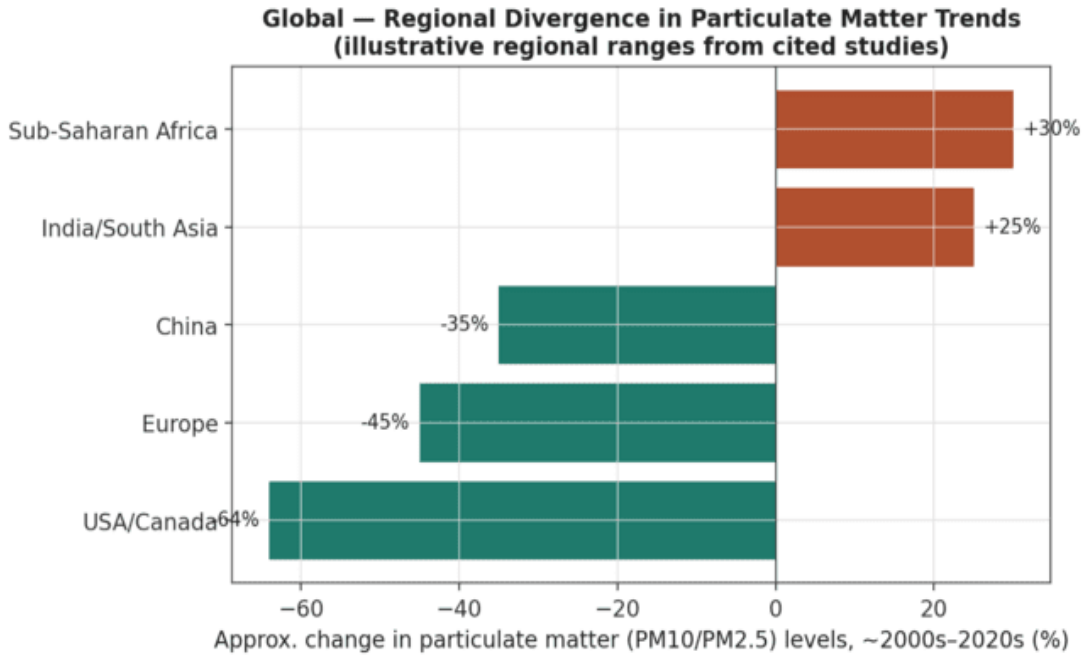
1 Global PM2.5

The most authoritative long-run global series is the population-weighted PM2.5 exposure estimate derived from satellite aerosol optical depth data (Nature Communications, 2023), covering 1998–2019. Global population-weighted PM2.5 rose from 28.3 µg/m³ in 1998 to a peak of 38.9 µg/m³ in 2011, before declining to 34.7 µg/m³ by 2019 — a reversal driven mainly by air-quality controls in China, partly offset by continued increases in South Asia and parts of Africa.



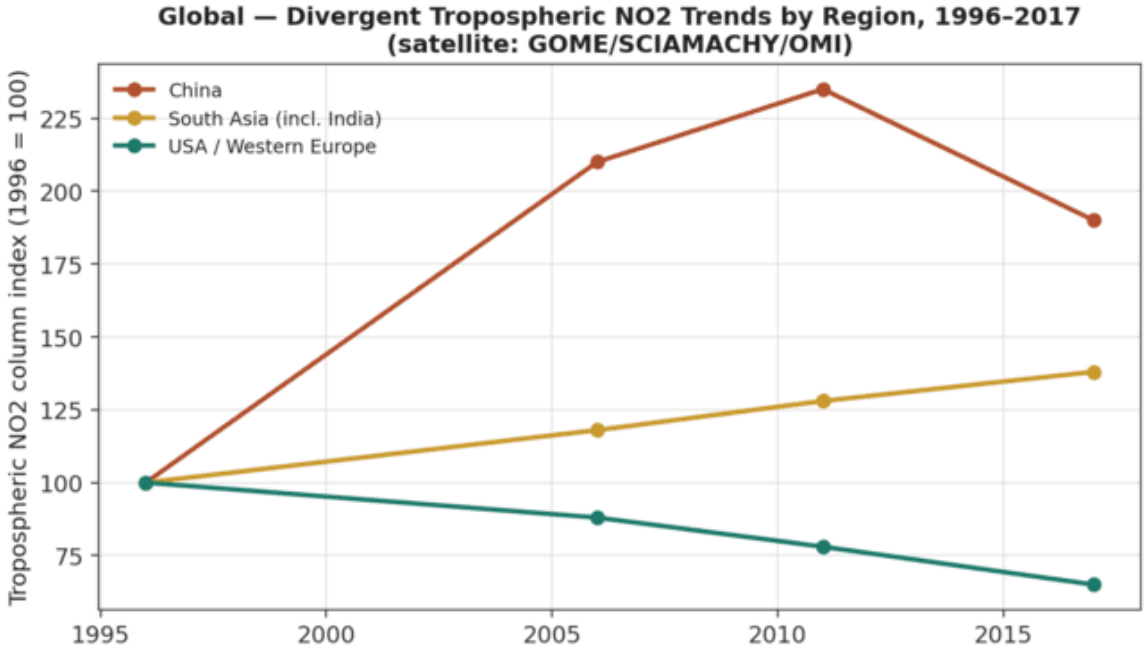
2. Global PM10 / Particulate Matter

No single unbroken global PM10 time series across 1996–2026 exists in the published literature; instead, the clearest documented pattern is a strong regional divergence. North America and Europe achieved large multi-decade reductions through vehicle and industrial emission controls, while China's particulate levels rose sharply into the early 2010s before falling after 2013 due to its Air Pollution Prevention and Control Action Plan, and India and Sub-Saharan Africa have continued to see rising levels over the same period.



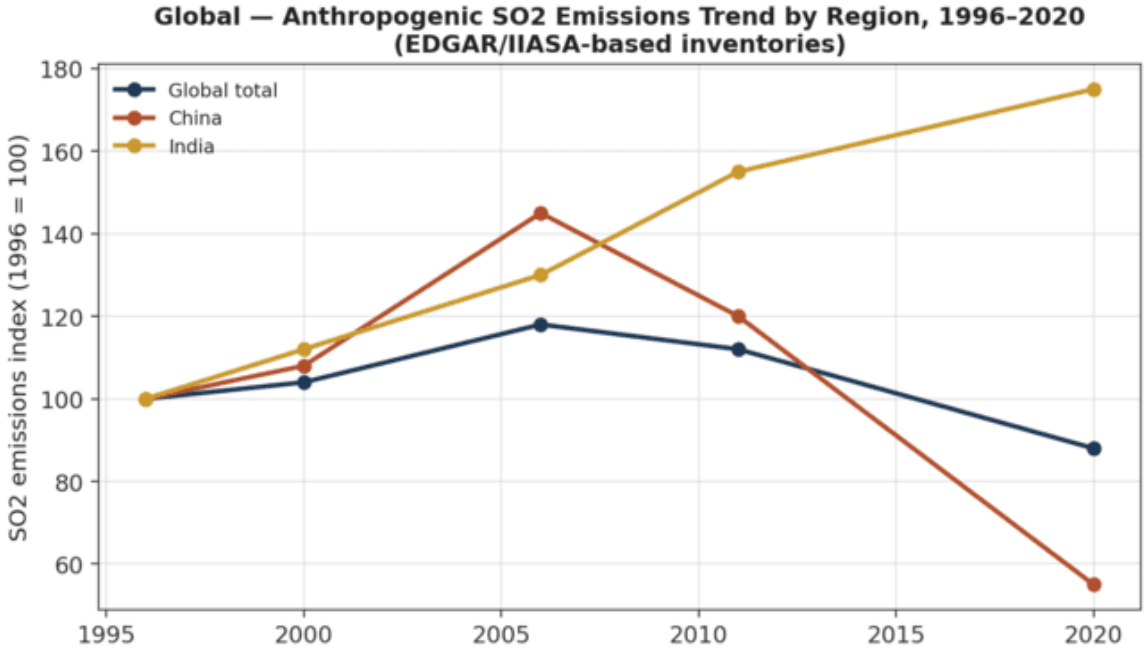
3 . Global NO2

Long-running satellite instruments (GOME, SCIAMACHY, and OMI) provide tropospheric NO2 column trends from 1996 onward. Combined GOME/SCIAMACHY data for 1996–2006 found NO2 columns rising by roughly 11%/year over China, 1.76%/year over South Asia (including India), 2.3%/year over the Middle East, and 2.4%/year over South Africa, while declining over the eastern United States (about −2%/year) and Europe (about −0.9%/year) in the same period. China's growth reversed after roughly 2011 due to emissions-control policy, while South Asia's NO2 columns continued rising through the 2010s, and developed-world regions (US, Netherlands, UK) recorded a roughly 49% cumulative decline over the two decades to 2017–19.



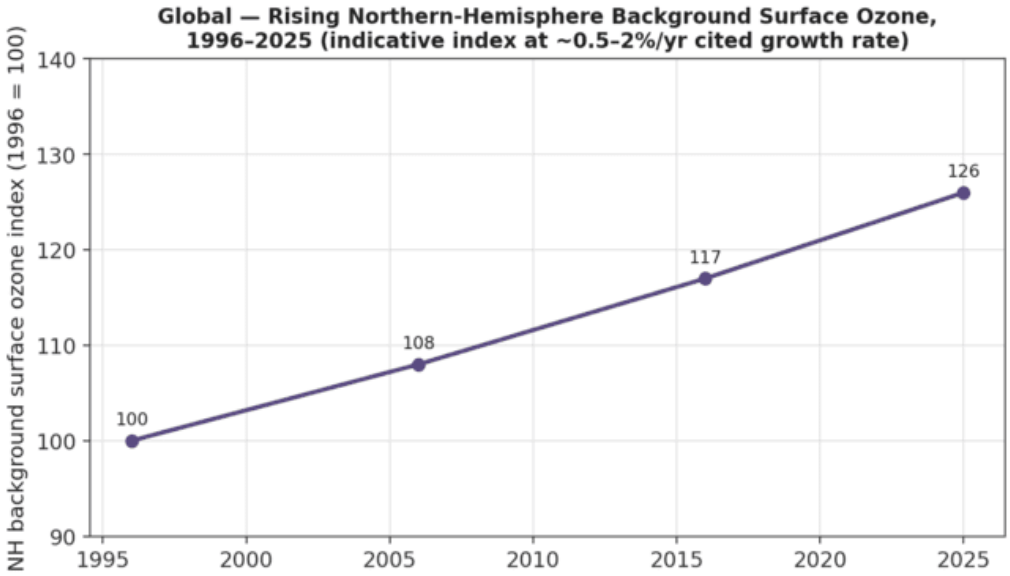
4 . Global SO2

Global anthropogenic SO2 emission inventories (EDGAR-based, IIASA) show total emissions rising through the early-to-mid 2000s, driven overwhelmingly by China's coal-based industrial expansion, before China's emissions plateaued and then fell sharply after roughly 2007–2011 following flue-gas desulfurization mandates. India's SO2 emissions, by contrast, grew more than 40% between 2005 and 2010 alone and showed no sign of slowing as of the most recent inventory years reviewed, later becoming the subject of India's own coal-plant FGD mandate from 2015 onward. European and US SO2 emissions fell by roughly 40–50% between 2000 and 2015.



5 . Global O3 (Tropospheric / Background Surface Ozone)

Background surface ozone at Northern Hemisphere mid-latitudes has been rising for decades, estimated in peer-reviewed reviews at roughly 0.5–2% per year over the past three decades, alongside a longer-run rate of about 0.60 ppb/year measured for 1980–2000. A 2024 global satellite/ozonesonde analysis found total column ozone increasing at 0.09 Dobson Units per year from 1980–2020 due to rising precursor (NOx, VOC) emissions and changing tropopause dynamics, even as stratospheric ozone itself has been recovering under the Montreal Protocol.



Summary Table — Direction of Trend

Pollutant	India (past decade)	Global (past ~3 decades)
PM2.5	Declining since 2021 peak, still ~10x WHO guideline	Rose to 2011 peak, declining since (but rising in South Asia/Africa)
PM10	Declining in most NCAP cities but majority still exceed NAAQS	Divergent: falling in US/EU/China (post-2013), rising in South Asia/Africa
NO2	Rising 2005–2011, decelerating/plateauing 2011–2019	Rising in developing regions, falling in US/EU since 1996
SO2	Declining nationally; hotspots persist near coal plants	China plateaued/declined post-2007–11; India still rising; US/EU declining
O3	Rising, especially summer-season urban/peri-urban levels	Rising background levels globally for several decades

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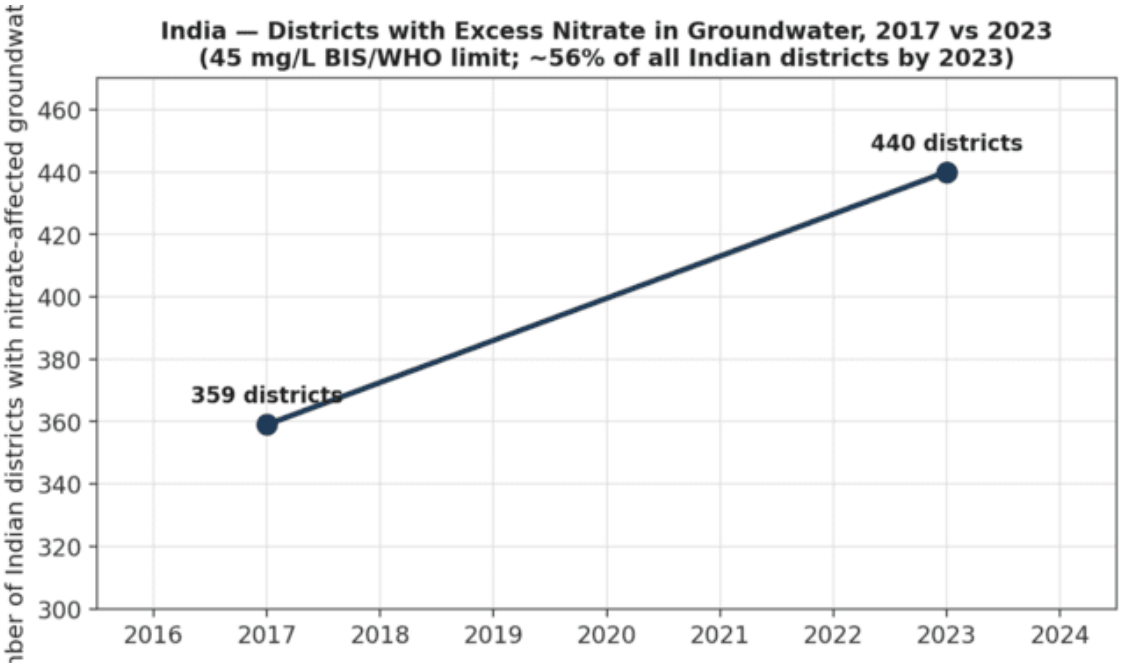
2)MAJOR WATER POLLUTANTS

Trends of the Five Major Water Contaminants in India

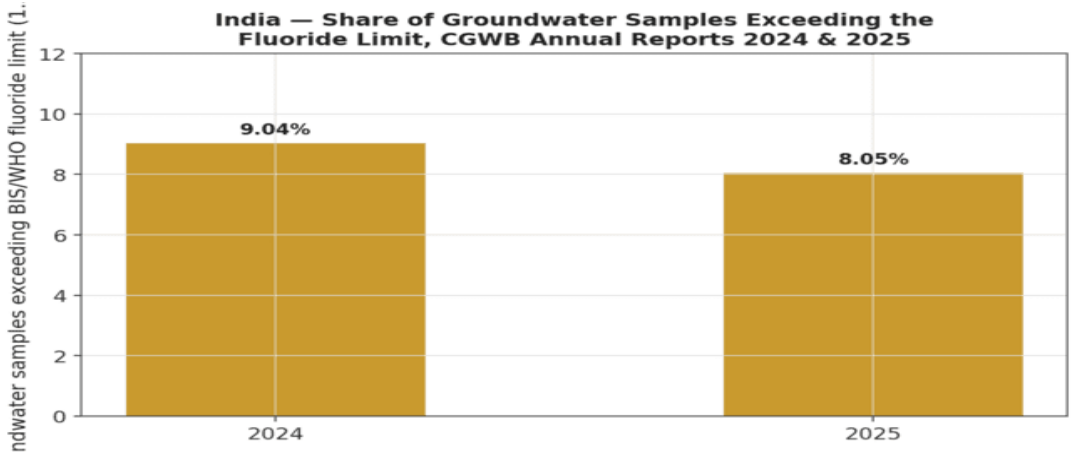
1. Nitrate

Nitrate, mainly from nitrogen-based fertiliser runoff, septic/sewage leakage, and animal waste, is the most widespread groundwater contaminant in India by sample count. CGWB's Annual Ground Water Quality Reports show the number of nitrate-affected districts rising markedly over roughly a decade, and roughly a fifth of national samples now exceed the safe limit.



2 Fluoride

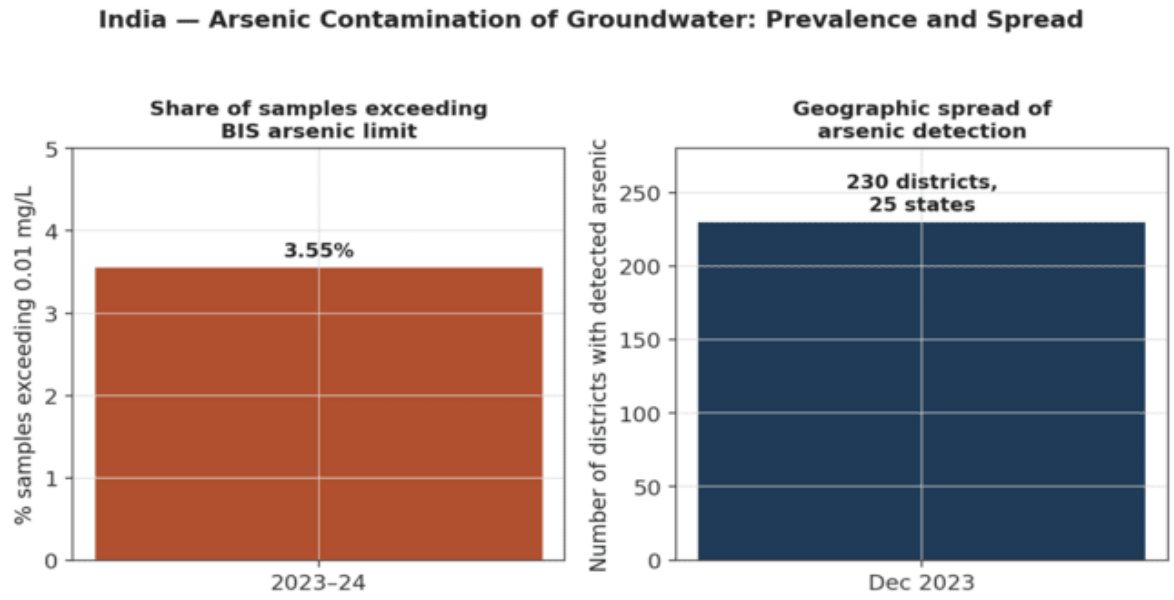
Fluoride contamination is predominantly geogenic (naturally occurring from fluoride-bearing rock weathering), concentrated in the arid and semi-arid belts of Rajasthan, Haryana, Karnataka, Andhra Pradesh, and Telangana, and is linked to dental and skeletal fluorosis.



The share of national samples exceeding the fluoride limit eased slightly between the two most recent CGWB assessments — 9.04% in the 2024 report to 8.05% in the 2025 report — though this reflects the specific samples drawn in each survey year rather than a confirmed multi-year downward trend, and CGWB emphasises that fluoride's largely geogenic origin means it does not show large systematic change over time.

3 Arsenic

Arsenic contamination is concentrated in the alluvial aquifers of the Ganga-Brahmaputra plains — West Bengal, Bihar, Uttar Pradesh, Jharkhand, and Assam — and is linked to skin lesions and cancer risk with long-term exposure.

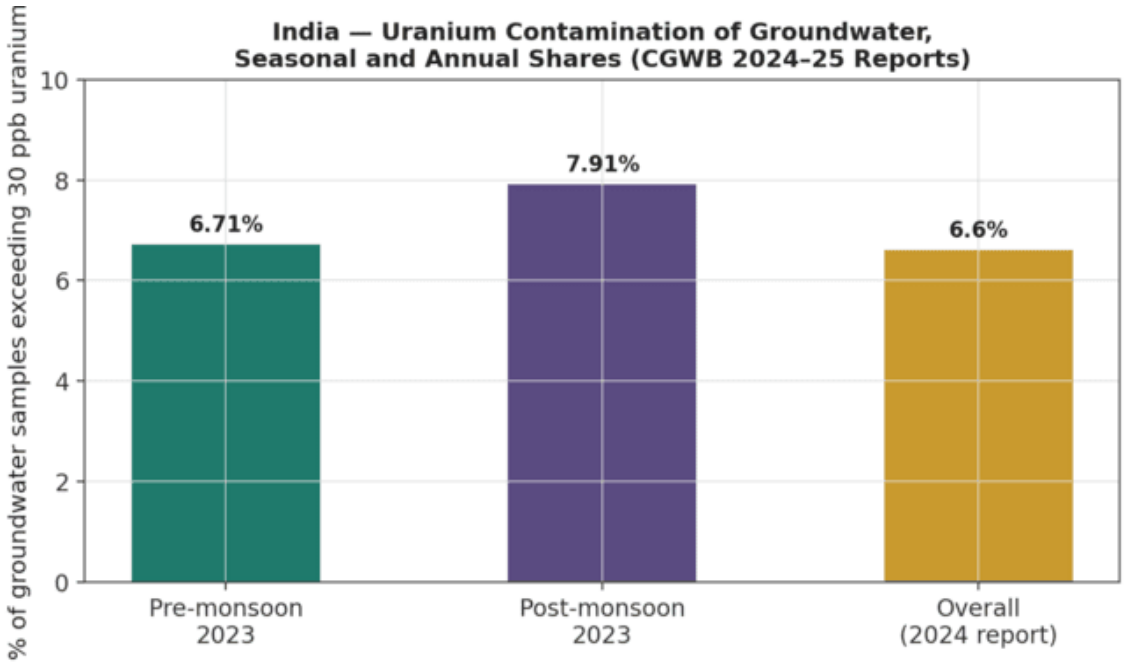


Source: CGWB, Annual Ground Water Quality Report 2024; Ministry of Jal Shakti statement to Parliament, December 2023 (reported by Deccan Herald and The Hindu/PressReader).

- 3.55% of the 15,259 national samples (May 2023) exceeded the 0.01 mg/L (10 µg/L) arsenic limit.
- As of December 2023, arsenic had been detected in groundwater across 230 districts in 25 states — the Ministry of Jal Shakti described this contamination as mostly geogenic and not showing significant year-on-year change.

4 Uranium

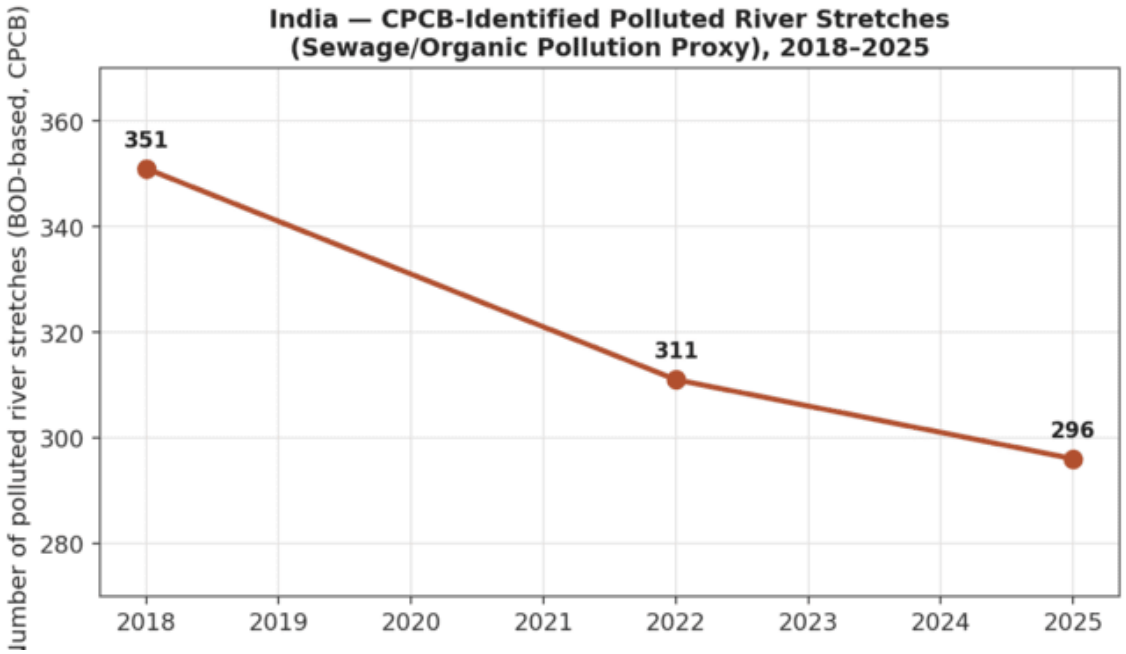
Uranium contamination, linked to kidney damage on chronic exposure, is concentrated in over-exploited, critical, and semi-critical groundwater-stress zones — notably Punjab, Rajasthan, Haryana, Gujarat, Tamil Nadu, Andhra Pradesh, and Karnataka — and CGWB has specifically flagged an association between falling water tables (from over-extraction) and rising uranium mobilisation.



Source: CGWB, Annual Ground Water Quality Report 2025 (pre-/post-monsoon 2023 trend-station data) and Annual Ground Water Quality Report 2024 (overall 2023 sampling).

5 Sewage-Derived Organic and Pathogenic Load (River BOD)

India's surface-water sewage/pathogen burden is tracked nationally by CPCB through Biochemical Oxygen Demand (BOD), an indicator of organic pollution mainly from untreated sewage and industrial effluent discharge; CPCB has published a periodic national count of 'polluted river stretches' where BOD exceeds the bathing-water criterion of 3 mg/L. This is the clearest decade-scale national indicator available for this contaminant class.



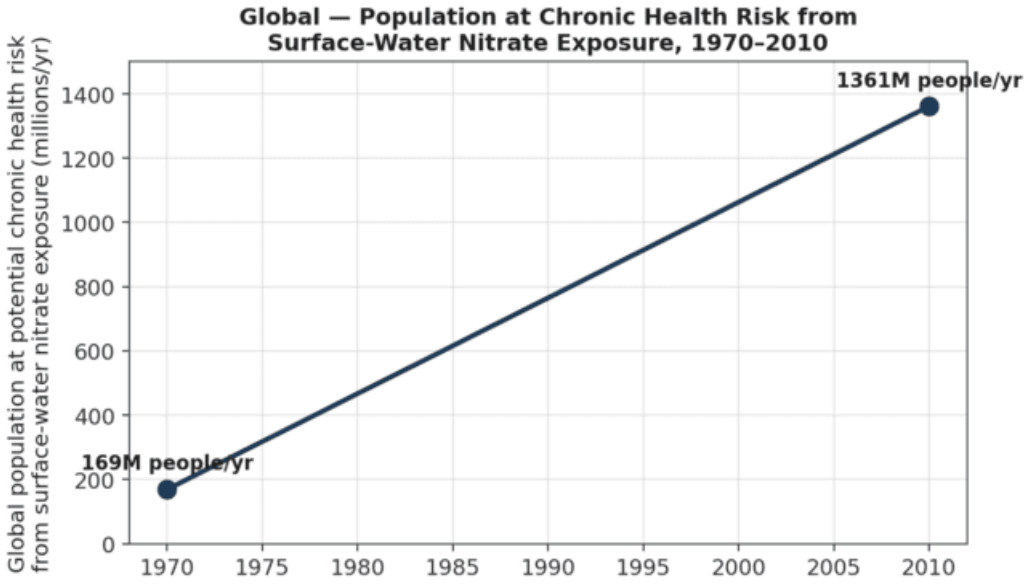
Source: CPCB, 'Polluted River Stretches for Restoration of Water Quality' reports (using BOD data from 2016–17, 2018, and subsequent assessment cycles through 2022 and 2024–25), and CPCB data as reported by Vajiram & Ravi and Energy Tracker Asia.

- In September 2018, CPCB identified 351 polluted river stretches nationally (using BOD data from 2016 and 2017) across 31 states/UTs.
- By its 2022 assessment, the number had fallen to 311 polluted stretches on 279 rivers, and by the 2024–25 assessment cycle to 296 stretches on 271 rivers — a roughly 15.7% decline since 2018.
- However, the most severely polluted (Priority I, BOD > 30 mg/L) category fell only from 46 to 37 stretches over the same period, and 108 stretches showed no change in priority class at all between 2018 and 2022, indicating persistent hotspots even as the national count improves.

Global Multi-Decadal Trends of the Five Contaminants

1 Global Nitrate

The most rigorous long-run global assessment (Wang et al., Environmental Science & Technology, 2023) models surface-water nitrate exposure and population health risk from 1970 to 2010 using global spatially explicit nitrate concentration and population data.

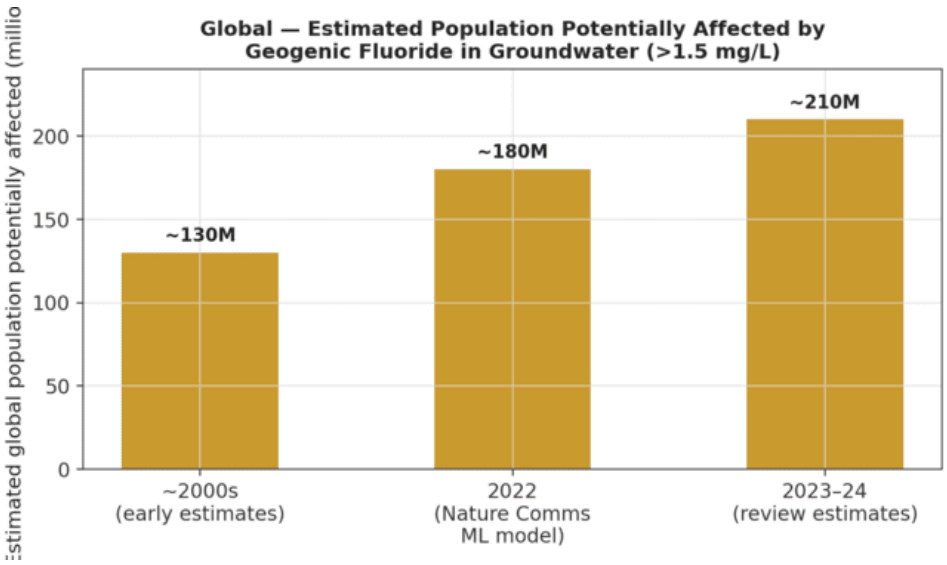


Source: Wang, J., Liu, X., Beusen, A. H. W., Middelburg, J. J., 'Surface-Water Nitrate Exposure to World Populations Has Expanded and Intensified during 1970–2010', Environmental Science & Technology 57, 2023 (open access via PMC).

Global populations at potential chronic health risk from surface-water nitrate exposure rose roughly eightfold, from 169 million persons/year in 1970 to 1,361 million persons/year in 2010, while populations at acute health risk rose tenfold, from 6 to 60 million persons/year, over the same 40 years. The study also found a geographic shift: populations at chronic risk were once concentrated in high-income Europe and North America but are now dominated by middle-income countries in Asia and Africa — consistent with global fertiliser-nitrogen use shifting toward East and South Asia after 1990.

2 Global Fluoride

No single continuous global time series exists for fluoride; the estimates below are drawn from the most-cited point estimates published at different times, including a 2022 machine-learning global hazard map.

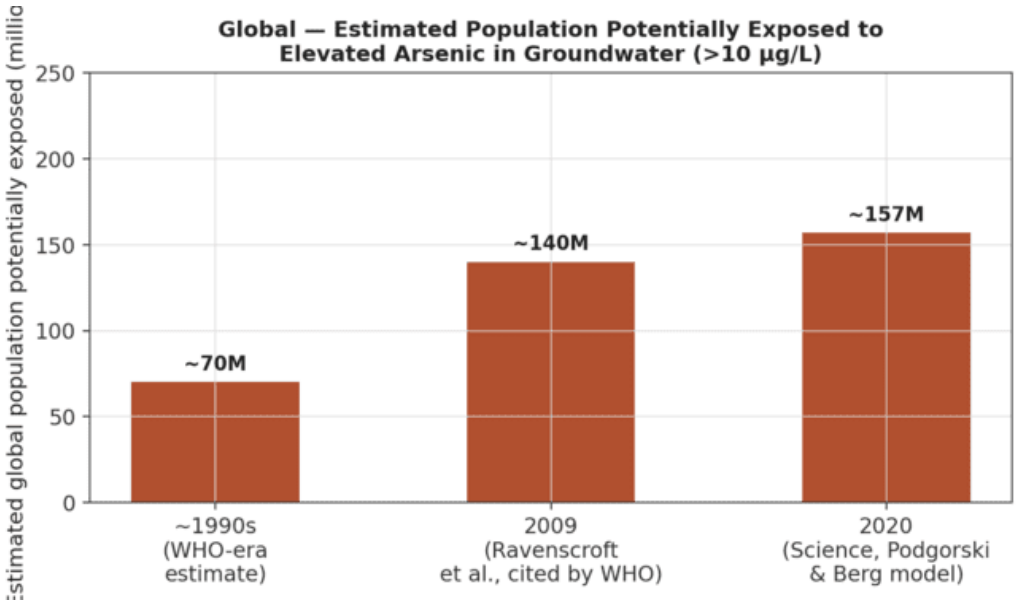


Source: Podgorski, J., Berg, M., 'Global analysis and prediction of fluoride in groundwater', Nature Communications 13, 2022 (open access); 'Fluoride contamination in groundwater: A global review', ScienceDirect, 2023–24; earlier estimates as compiled in 'Worldwide Occurrences of Fluoride in Groundwater', Fluoride in Groundwater (Groundwater Project), 2022.

Estimates of the global population potentially exposed to fluoride above the WHO guideline of 1.5 mg/L have grown from roughly 130 million in early 2000s reviews toward approximately 180 million in the 2022 global machine-learning hazard model (Podgorski & Berg, Nature Communications), and 200 million or more in subsequent 2023–24 review estimates — reflecting both genuinely expanding detection (over 100 countries now report fluoride contamination, versus far fewer decades ago) and improved global mapping resolution, which the source studies note makes it difficult to separate a true rise in contamination from a rise in monitoring coverage.

3 Global Arsenic

Similarly, arsenic exposure estimates have been revised upward repeatedly as detection has expanded from the original Bangladesh/West Bengal 'mass poisoning' cases (first flagged by WHO in 2000) to a global prediction model.

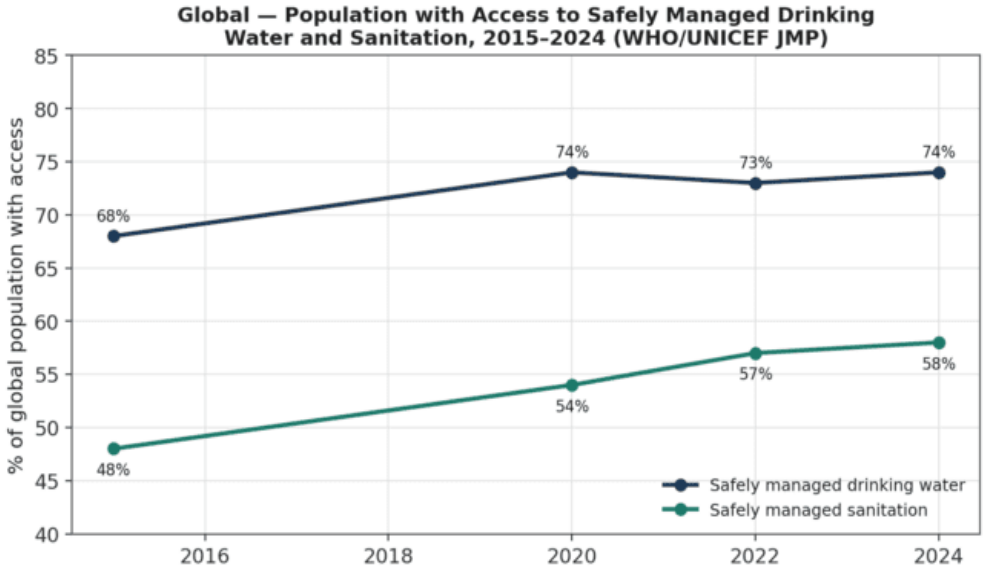


Source: Podgorski, J., Berg, M., 'Global threat of arsenic in groundwater', Science 368, 2020 (peer-reviewed); Ravenscroft, P. et al. (2009), as cited in WHO Arsenic Fact Sheet (who.int); Smith, A. H. et al., 'Contamination of drinking-water by arsenic in Bangladesh: A public health emergency', Bulletin of the World Health Organization 78, 2000.

Early WHO-era estimates from the 1990s put the global population exposed to unsafe arsenic levels at roughly 70 million, concentrated overwhelmingly in Bangladesh and West Bengal, India. By 2009, the widely-cited Ravenscroft et al. assessment (used in the current WHO arsenic fact sheet) put the figure at approximately 140 million. The most recent global statistical model (Podgorski & Berg, Science, 2020), built from over 800,000 measurements, estimates 94 to 220 million people are potentially exposed worldwide, with 94% of that population in Asia — a range whose midpoint (roughly 157 million) is shown in the chart.

4 Global Sewage-Derived Contamination (Access to Safely Managed Water and Sanitation)

The clearest continuous global decadal indicator for pathogen/sewage-related water contamination is the WHO/UNICEF Joint Monitoring Programme's tracking of the share of the world's population with 'safely managed' drinking water and sanitation — a service level introduced under the Sustainable Development Goals framework and tracked consistently since 2015.

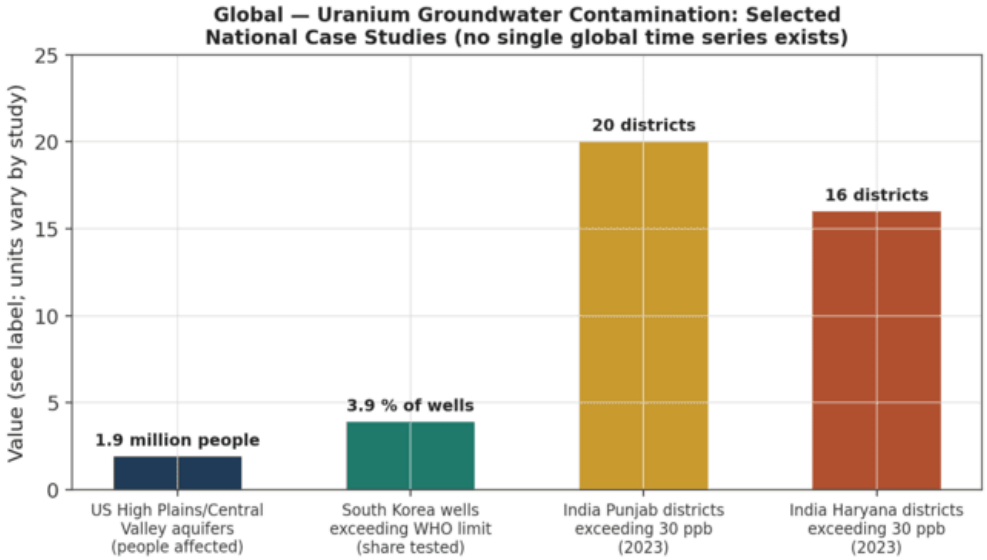


Source: WHO/UNICEF Joint Monitoring Programme (JMP) for Water Supply, Sanitation and Hygiene: 'Progress on household drinking water, sanitation and hygiene, 2000–2020', 'Progress...2000–2022: Special focus on gender', and 'Progress...2000–2024' reports, data.unicef.org / washdata.org.

- Global access to safely managed drinking water rose from 68% in 2015 to 74% in 2024 — 961 million people gained access over that period, though 2.1 billion people still lacked it as of 2024.
- Global access to safely managed sanitation rose from 48% in 2015 to 58% in 2024 — 1.2 billion people gained access, though 3.4 billion people still lacked it as of 2024.
- Global open defecation — the most severe end of the sanitation/pathogen-exposure spectrum — fell by more than half between 2015 and 2024, from 10% (784 million people) to 4% (354 million people) of the world's population.

5 Global Uranium

No global population-exposure model comparable to those for arsenic or fluoride yet exists for uranium in groundwater; the literature instead consists of scaling national and regional case studies, which nonetheless show a consistent pattern — uranium in drinking water was rarely measured before 1980, and detected contamination has expanded steadily as testing has spread from a handful of countries to dozens over the past three decades.



Source: Nolan, J., Weber, K. A., 'Natural Uranium Contamination in Major U.S. Aquifers Linked to Nitrate', Environmental Science & Technology Letters, 2015; Shin, W. J. et al. (2016) South Korea well survey, cited in 'Emerging health risks...of uranium contamination', ScienceDirect, 2020; Coyte, R. M. et al. (2018) India Rajasthan/Gujarat well survey, cited in the same ScienceDirect review; CGWB Annual Ground Water Quality Report 2024 (Punjab/Haryana district counts).

In the United States, the High Plains and Central Valley aquifers alone show uranium exceeding the WHO/EPA guideline (30 µg/L) across roughly 22,375 km², affecting an estimated 1.9 million residents living within 1 km of contaminated groundwater. In South Korea, 160 of 4,140 tested wells (about 3.9%) exceeded the WHO guideline, concentrated in plutonic bedrock regions. In India, CGWB's most recent assessment found 20 districts in Punjab and 16 in Haryana exceeding the 30 ppb provisional limit, consistent with earlier well-level surveys (Coyte et al., 2018) that found uranium up to 2,074.8 µg/L in Rajasthan and Gujarat aquifers.

Summary Table — Direction of Trend

Contaminant	India (past decade)	Global (past ~3 decades)
Nitrate	Rising: districts affected up from 359 (2017) to 440 (2023)	Rising sharply: chronic-risk population up ~8x, 1970–2010
Fluoride	Roughly stable/geogenic; 9.04% (2024) to 8.05% (2025) of samples	Estimates rising as detection expands: ~130M to ~200M+
Arsenic	Stable/geogenic; 3.55% of samples, 230 districts (2023-24)	Estimates rising as detection expands: ~70M to ~94-220M
Uranium	Rising with over-extraction; 6.71% pre- to 7.91% post-monsoon	Expanding geographic detection; no single global series yet
Sewage/BOD (pathogen proxy)	Improving: polluted river stretches down from 351 (2018) to 296 (2025)	Improving: safely managed sanitation up from 48% (2015) to 58% (2024)

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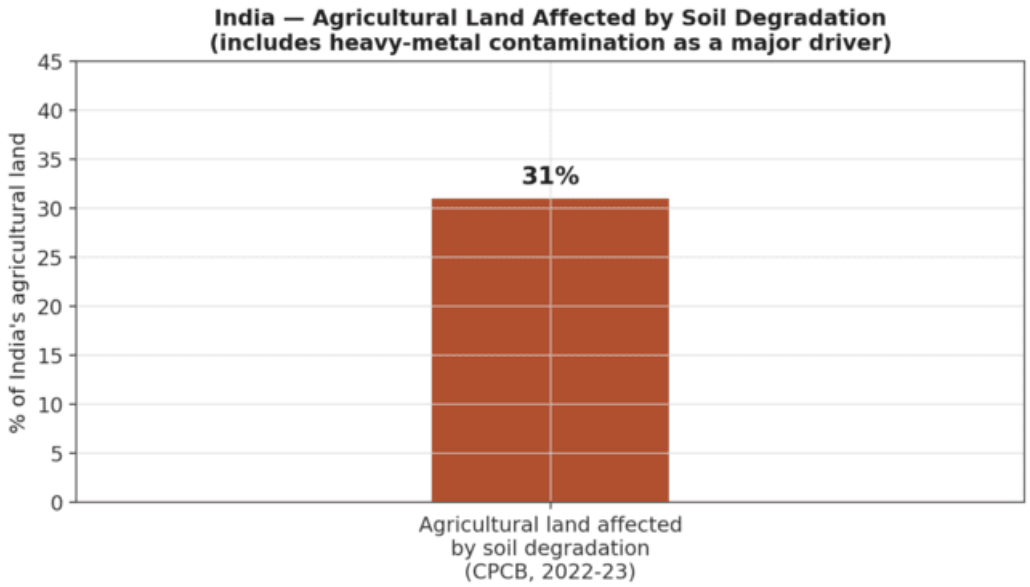
MAJOR SOIL POLLUTANTS:

Trends of the Five Major Soil Contaminants in India

1. Heavy Metals (Cd, Pb, Cr, As, Hg)

India has no single national soil-quality standard or continuous monitoring network comparable to its air and groundwater networks; CPCB instead uses site-specific screening levels, and most quantitative evidence comes from peer-reviewed regional studies rather than an annual government series. The clearest available national-scale figure is CPCB's estimate of the share of agricultural land affected by soil degradation, of which heavy-metal contamination (from industrial effluent, sewage-irrigation, and mining) is a major driver.

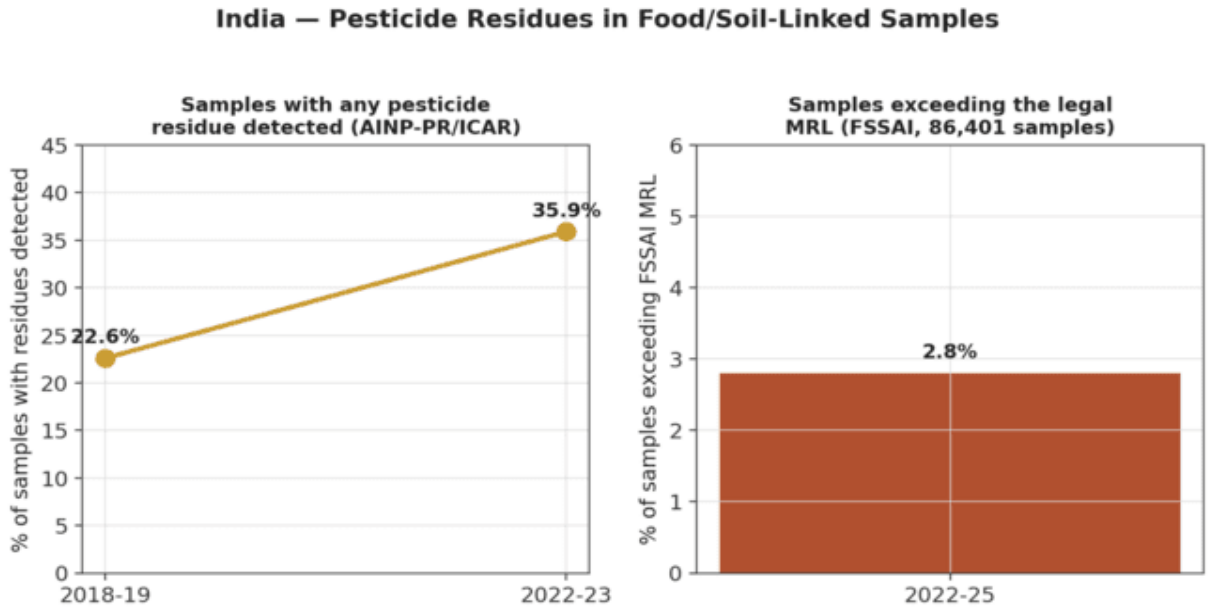


Source: CPCB, cited in national soil degradation statistics (2022-23); 'Pollution assessment of heavy metals in soils of India and ecological risk assessment: A state-of-the-art', ScienceDirect, 2018 (review of 1991–2018 Indian studies).

CPCB estimates that approximately 31% of India's agricultural land is affected by soil degradation (2022–23), with multiple industrial-belt studies documenting cadmium, lead, and chromium levels exceeding India's natural soil background levels near industrial effluent and sewage-irrigation sites. A 2018 peer-reviewed review of Indian soil studies from 1991–2018 found zinc and lead levels exceeding Indian, Canadian, Swedish, and Polish soil guideline values across multiple soil types, and identified cadmium and arsenic as the metals of greatest ecological concern nationally, with cadmium showing the highest potential ecological risk index of any metal studied.

2. Pesticide Residues

Pesticide residues are tracked nationally through two separate official monitoring efforts: the ICAR All India Network Project on Pesticide Residues (AINP-PR), which reports the share of samples with any detectable residue, and FSSAI's National Monitoring Plan, which reports the share of samples exceeding the legal Maximum Residue Limit (MRL). These measure different things and are shown separately.

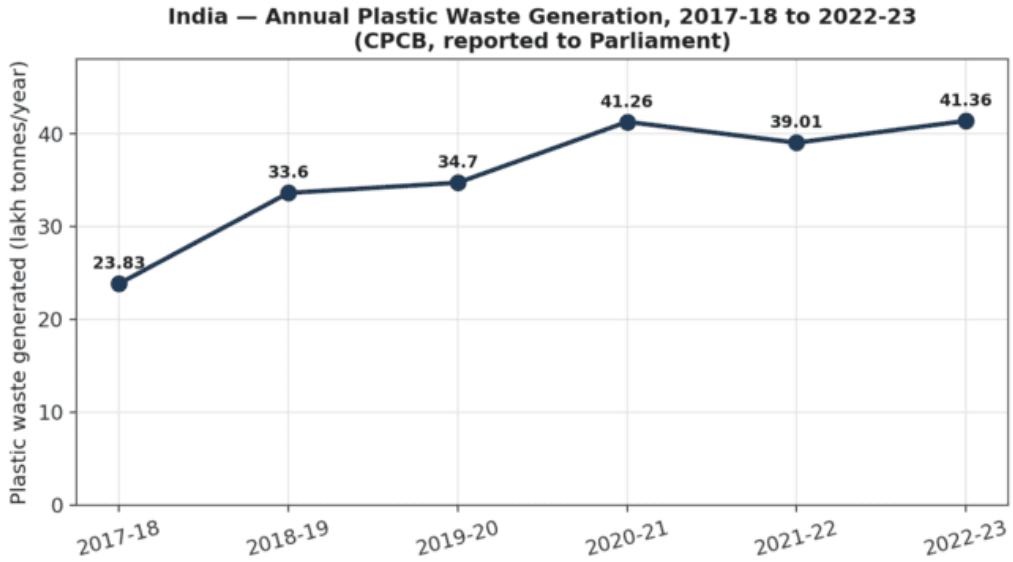


Source: All India Network Project on Pesticide Residues (ICAR), data obtained via RTI and reported by Down To Earth, April 2024; FSSAI National Monitoring Plan data, reported to the Rajya Sabha, August 2025.

- The share of food/produce samples with any detectable pesticide residue rose from 22.6% in 2018-19 to 35.9% in 2022-23, according to five years of AINP-PR data obtained under the Right to Information Act.
- Separately, FSSAI's National Monitoring Plan found that of 86,401 samples tested nationally for pesticide residues during 2022-25, 2.8% exceeded the legal MRL — indicating that while detectable residues are increasingly common, samples breaching the enforceable safety limit remain a small minority.

3. Plastics and Microplastics

Plastic waste entering soil (via dumping, landfill leachate, agricultural mulch film, and sewage sludge used as fertiliser) is tracked through CPCB's annual reports on the implementation of the Plastic Waste Management Rules, 2016, based on data submitted by State Pollution Control Boards.

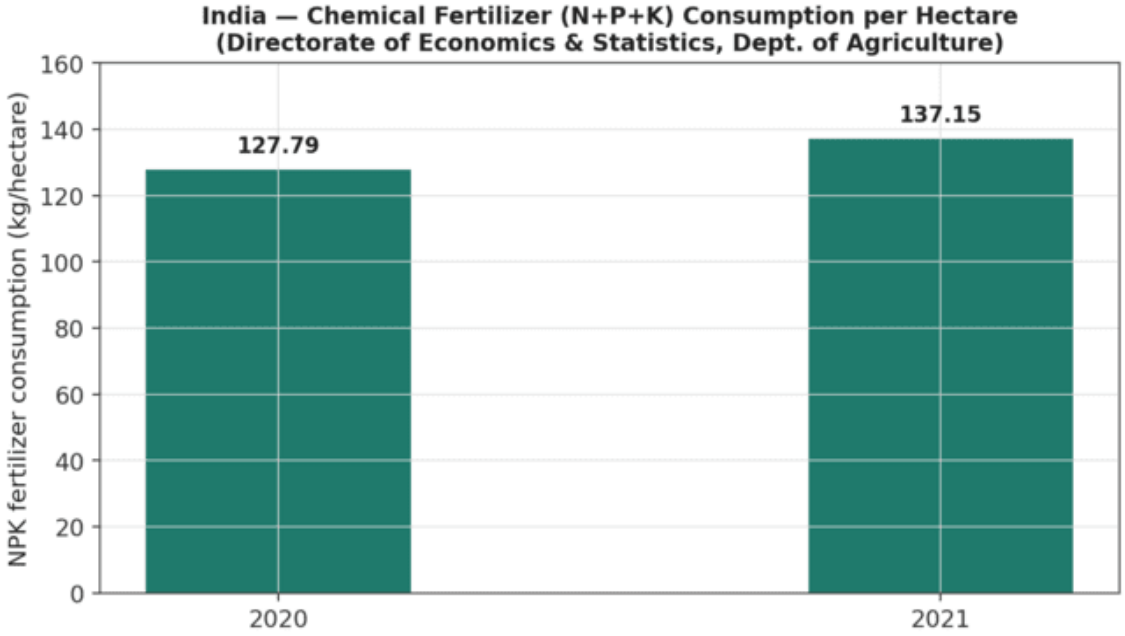


Source: CPCB Annual Reports on Implementation of Plastic Waste Management Rules, 2016 (2018-19 to 2022-23 editions); Ministry of Environment, Forest and Climate Change, replies to Parliament (Rajya Sabha/Lok Sabha), as reported by Deccan Herald, CEED India, and Telecom Talk.

India's estimated annual plastic waste generation rose from 23.83 lakh tonnes in 2017-18 to 41.36 lakh tonnes in 2022-23 — a rise of roughly 74% over five years, with a temporary dip in 2021-22 (39.01 lakh tonnes) that coincided with COVID-19-related disruptions before rising again. Per-capita plastic waste generation is separately reported to have risen from around 700 grams in 2016-17 to over 2.5 kilograms by 2020. CPCB's own study on the impact of plastic waste disposal at Lucknow dumpsites found that dumped plastic waste degrades soil and groundwater quality through the leaching of additives, colourants, stabilisers, and fillers.

4. Excess Nitrogen and Phosphate (Fertiliser Residue)

Over-application of nitrogen-phosphorus-potassium (NPK) chemical fertiliser is tracked through the Department of Agriculture & Farmers Welfare's per-hectare consumption statistics.

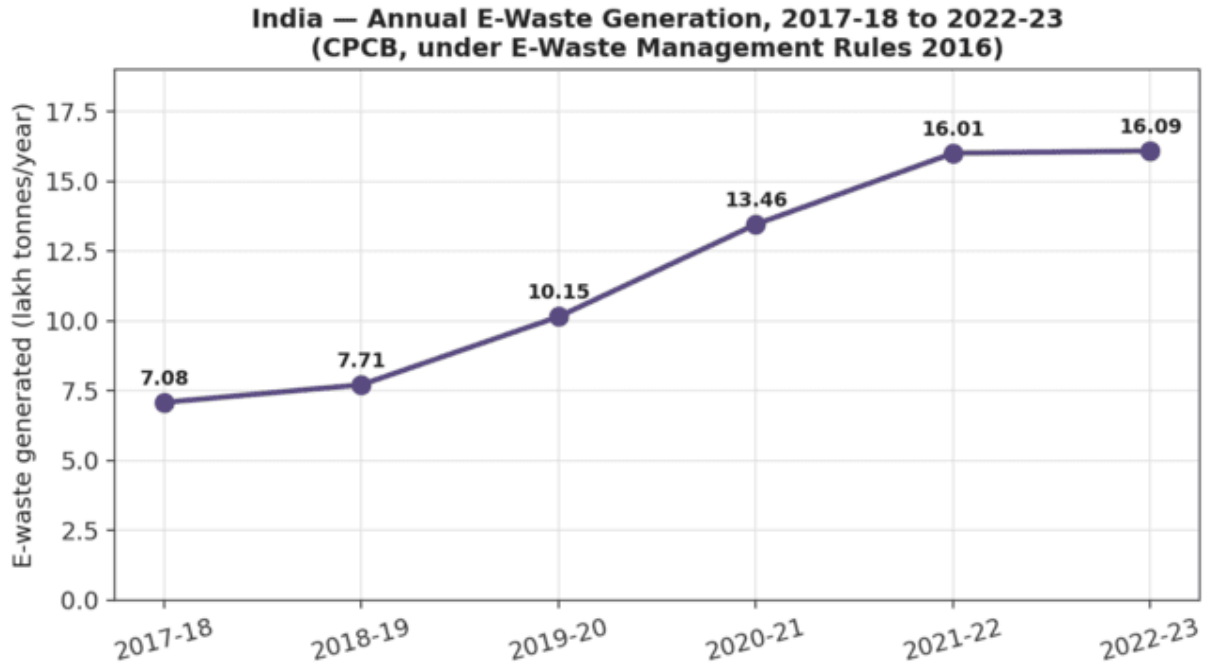


Source: Directorate of Economics and Statistics, Department of Agriculture and Farmers Welfare, Government of India, via CEIC Data; Impact Study of the Soil Health Card Scheme, MANAGE, Hyderabad; government data shared in the Rajya Sabha, reported by Factly, 2021.

- National NPK fertiliser consumption per hectare rose from 127.79 kg/ha in 2020 to 137.15 kg/ha in 2021 — continuing a trend in which India's chemical fertiliser consumption rose by about 16% between 2015-16 and 2020-21.
- India's current NPK application ratio is approximately 6.7:2.4:1, heavily skewed toward nitrogen against an agronomically recommended ratio of roughly 4:2:1, according to the Soil Health Card Scheme impact study — a skew CPCB and the Ministry of Agriculture link to declining soil micronutrient balance and long-term soil health decline in intensively farmed states such as Punjab, Haryana, and Telangana.

5. E-Waste-Derived Heavy Metals and POPs

E-waste is tracked nationally through CPCB's e-waste generation estimates, based on producer sales data and average product lifespans under the E-Waste (Management) Rules, 2016. Improper dismantling and informal recycling — concentrated at hotspots such as Moradabad and Seelampur — release heavy metals and persistent organic pollutants directly into soil.



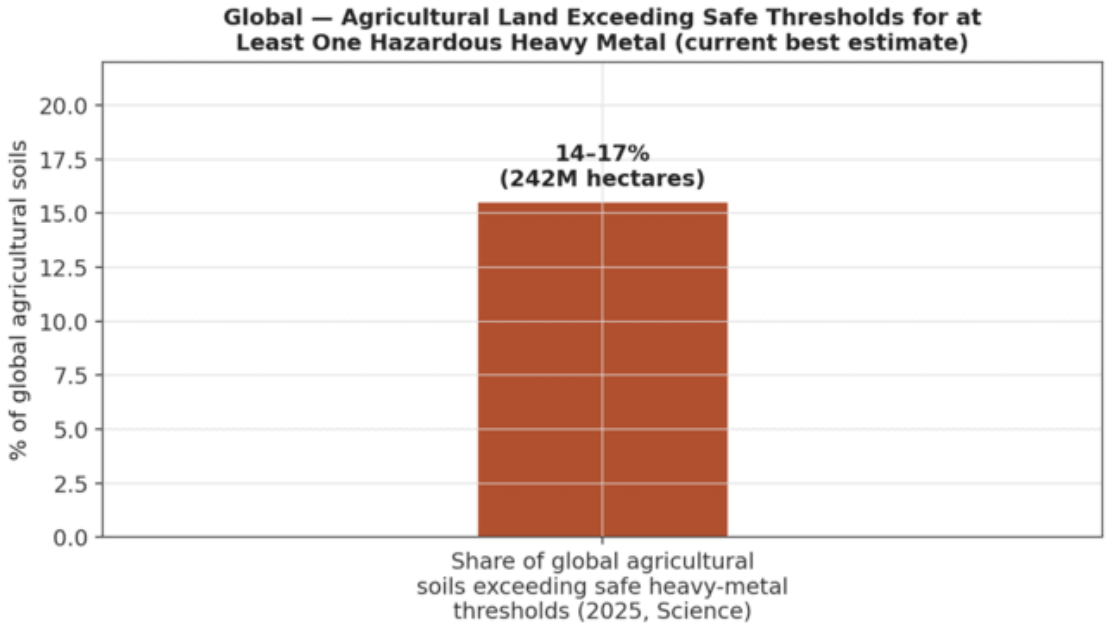
Source: CPCB e-waste generation data, reported to the Rajya Sabha by the Ministry of Environment, Forest and Climate Change (December 2024) and via Down To Earth (January 2021); Dataful/CPCB compiled state-level e-waste datasets, August 2025.

India's estimated e-waste generation rose from 7.08 lakh tonnes in 2017-18 to 16.09 lakh tonnes in 2022-23 — more than doubling in five years, a faster growth rate than plastic waste over the same period. Formal collection and processing has lagged well behind generation throughout: only 3.5% of 2017-18's e-waste and 10% of 2018-19's e-waste was formally collected, though the formally processed share has since risen to about 26% (2020-21) as Extended Producer Responsibility rules have taken effect.

Global Multi-Decadal Trends of the Five Contaminants

1. Global Heavy Metals in Agricultural Soil

The first truly global, machine-learning-based assessment of soil heavy-metal contamination was published in Science only in April 2025 (Hou et al., Tsinghua University), analysing more than 1,000 regional datasets. No earlier study attempted a comparable global estimate, so this section presents the current best global figure rather than a trend line.

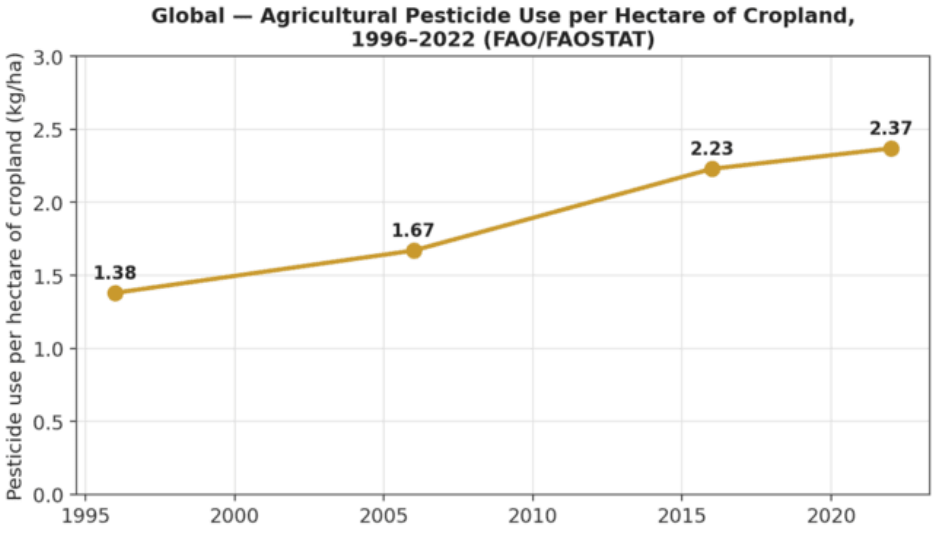


Source: Hou, D. et al., 'Global soil pollution by toxic metals threatens agriculture and human health', Science, April 17, 2025, as reported by Down To Earth and Climate Fact Checks.

The study found that 14–17% of the world's agricultural soils (approximately 242 million hectares — an area close to the size of China's entire agricultural land) exceed safe thresholds for at least one hazardous heavy metal, potentially affecting between 900 million and 1.4 billion people living in high-risk regions. Cadmium is the most widespread pollutant, exceeding safe levels in 9% of soils globally, with hotspots in northern and central India, Pakistan, Bangladesh, southern China, and parts of Africa and Latin America — directly overlapping with the cadmium/arsenic concern flagged in Indian studies in Section 1.1. The researchers caution that the true global extent is likely underestimated due to sparse data from sub-Saharan Africa and northern Russia.

2.Global Pesticide Use

FAO's FAOSTAT Pesticides Use domain provides a continuous, comparable global series from 1990 onward, based on active-ingredient application data submitted by member countries.

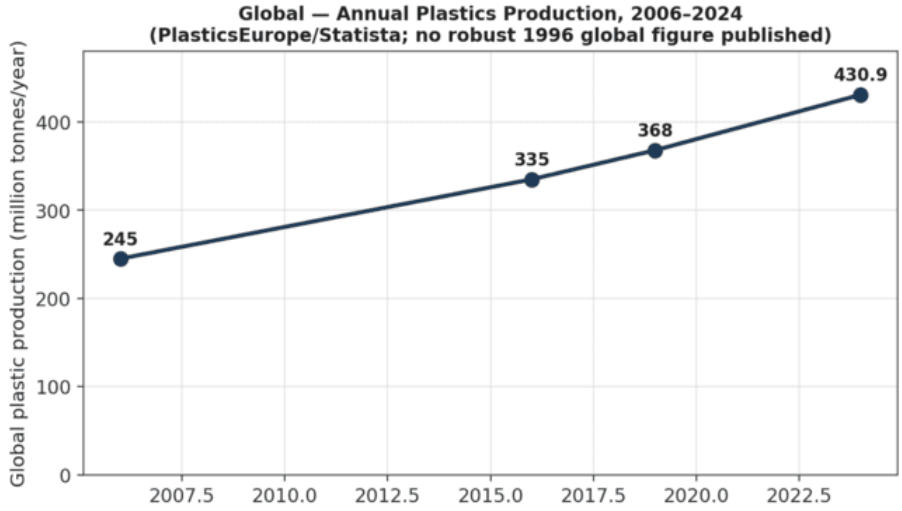


Source: FAO, FAOSTAT Pesticides Use and Pesticides Indicators domain (1990–2023 data), as compiled by Statista and cited in FAO's 'Pesticides use, pesticides trade and pesticides indicators' analytical briefs.

Global pesticide use per hectare of cropland rose steadily from 1.38 kg/ha in 1996 to 1.67 kg/ha in 2006, 2.23 kg/ha in 2016, and 2.37 kg/ha in 2022 — an increase of roughly 72% over that span, and FAO notes total global use more than doubled between 1990 and the 2020s. Growth has been most pronounced in the Americas (a 210% rise in agricultural pesticide use between 1990 and 2022) and parts of Asia, while Africa and Europe have shown the slowest growth.

3.Global Plastics Production

PlasticsEurope's Market Research Group has published the most widely cited continuous global plastics production series; a robust directly-sourced figure for 1996 specifically was not found in available official releases, so the chart begins at the earliest well-documented year, 2006.

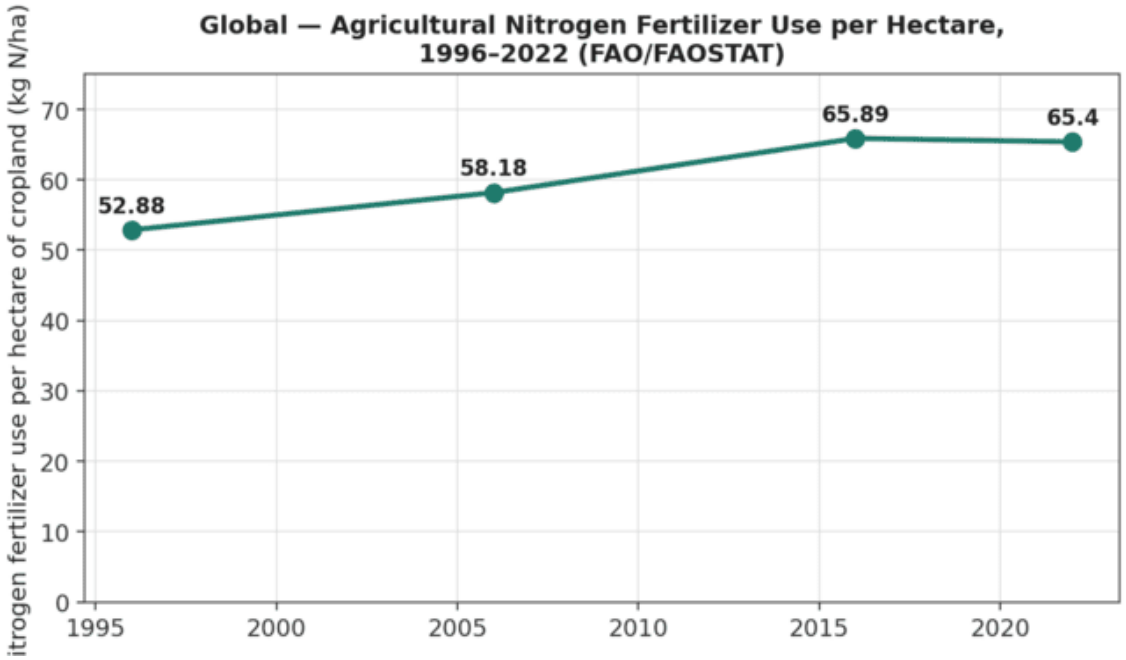


Source: PlasticsEurope, 'The Compelling Facts About Plastics' (2006 edition) and 'Plastics – the Facts' (2020 edition); Statista compilation 'Annual production of plastics worldwide from 1950 to 2024'; Geyer, R., Jambeck, J. R., Law, K. L., 'Production, use, and fate of all plastics ever made', Science Advances, 2017 (open access).

Global plastics production rose from 245 million tonnes in 2006 to 335 million tonnes in 2016, 368 million tonnes in 2019, and surpassed 430.9 million tonnes in 2024 — a rise of roughly 76% in under two decades. The landmark Geyer et al. (2017) lifecycle study found that of the roughly 6,300 million tonnes of plastic waste generated by 2015 (out of 8,300 million tonnes of virgin plastic ever produced), only 9% had been recycled, 12% incinerated, and 79% had accumulated in landfills or the natural environment — much of which ultimately reaches soil.

4. Global Nitrogen Fertiliser Use

FAO's FAOSTAT Fertilizers by Nutrient domain provides a continuous global series on nitrogen fertiliser application per hectare of cropland from 1990 onward.

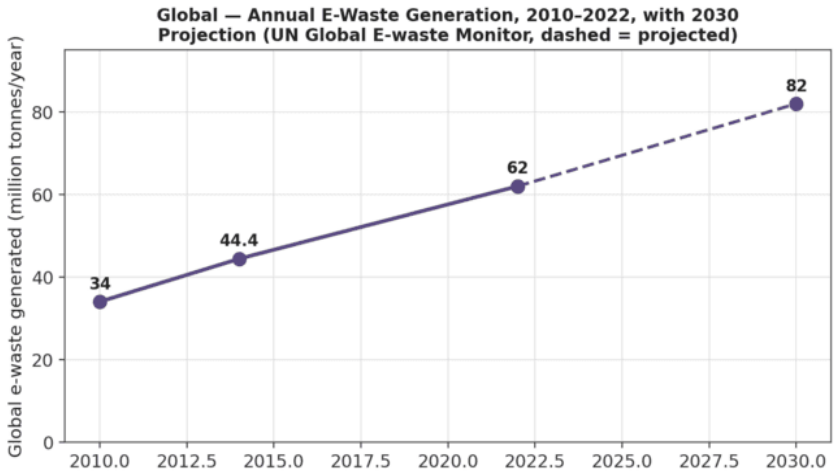


Source: FAO, FAOSTAT Land, Inputs and Sustainability: Fertilizers by Nutrient domain, as compiled by Statista and Our World in Data (2025).

Global nitrogen fertiliser use per hectare of cropland rose from 52.88 kg N/ha in 1996 to 58.18 kg N/ha in 2006, 65.89 kg N/ha in 2016, and 65.4 kg N/ha in 2022 — an increase of roughly 24% since 1996, with growth largely plateauing over the most recent decade. Global nitrogen-use efficiency (the share of applied nitrogen actually taken up by crops) has been estimated at only 42–47% in recent decades, meaning more than half of applied nitrogen is lost to soil, water, and air rather than absorbed by plants.

5. Global E-Waste Generation

The UN's Global E-waste Monitor (produced by UNITAR and the International Telecommunication Union) has published four editions since 2014 and provides the clearest continuous global e-waste series, though it does not extend back to 1996.



Source: Global E-waste Monitor 2024 (UNITAR/ITU), March 2024, as reported by Waste Management World and the Circular Electronics Partnership.

Global e-waste generation rose from 34 million tonnes in 2010 to 44.4 million tonnes in 2014 and a record 62 million tonnes in 2022 — an 82% increase since 2010 — and is projected to reach 82 million tonnes by 2030. In 2022, only 22.3% of that e-waste was formally collected and recycled, leaving the great majority to be informally processed or dumped, releasing heavy metals and POPs into soil at both formal and informal recycling and disposal sites worldwide.

Summary Table — Direction of Trend

Contaminant	India (past decade)	Global (past ~3 decades)
Heavy metals	31% of agricultural land degraded (2022-23); no continuous series	14-17% of global agri-soils affected (2025 estimate); no continuous series
Pesticide residues	Detected-residue share up from 22.6% (2018-19) to 35.9% (2022-23)	Use per hectare up from 1.38 (1996) to 2.37 kg/ha (2022), +72%
Plastics/microplastics	Waste generation up from 23.8 (2017-18) to 41.4 lakh t (2022-23)	Production up from 245 (2006) to 430.9 million t (2024), +76%
Excess N/P (fertiliser)	NPK use up from 127.8 (2020) to 137.2 kg/ha (2021); N-skewed ratio	N use per hectare up from 52.9 (1996) to 65.4 kg N/ha (2022), +24%
E-waste (heavy metals/POPs)	Generation up from 7.1 (2017-18) to 16.1 lakh t (2022-23), +127%	Generation up from 34 (2010) to 62 million t (2022), +82%

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11. PlasticsEurope, 'The Compelling Facts About Plastics' (2006) and 'Plastics – the Facts 2020'; Statista, 'Annual production of plastics worldwide from 1950 to 2024'.
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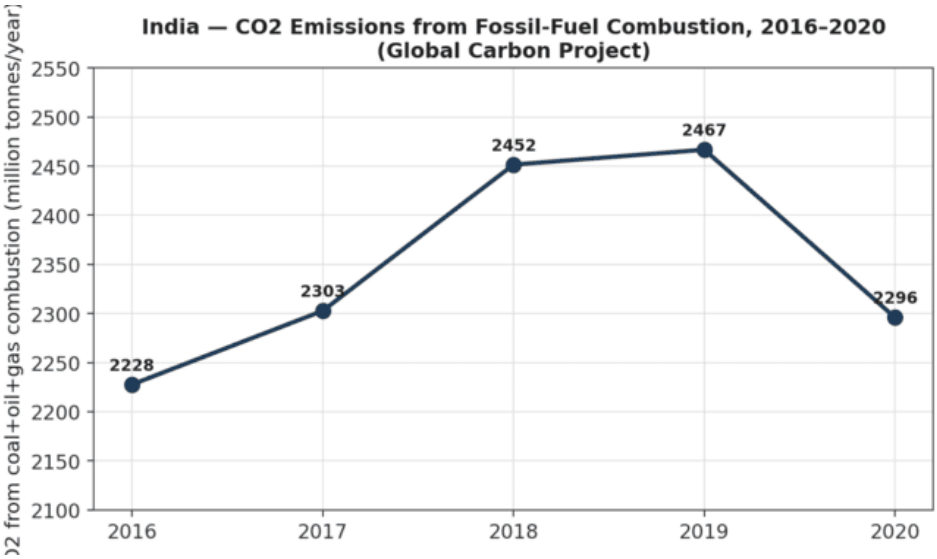
MAJOR CLIMATE CONTAMINANTS

Trends of the Five Major Climate Pollutants in India

1 Carbon Dioxide (CO2)

CO2 is by far the largest contributor to India's GHG inventory — 80.53% of total emissions in the 2020 inventory year, per BUR-4. The Global Carbon Project's annual fossil-fuel emissions accounting provides a clean, continuous, methodologically consistent series for coal, oil, and gas combustion in India across the requested 2016–2020 span.

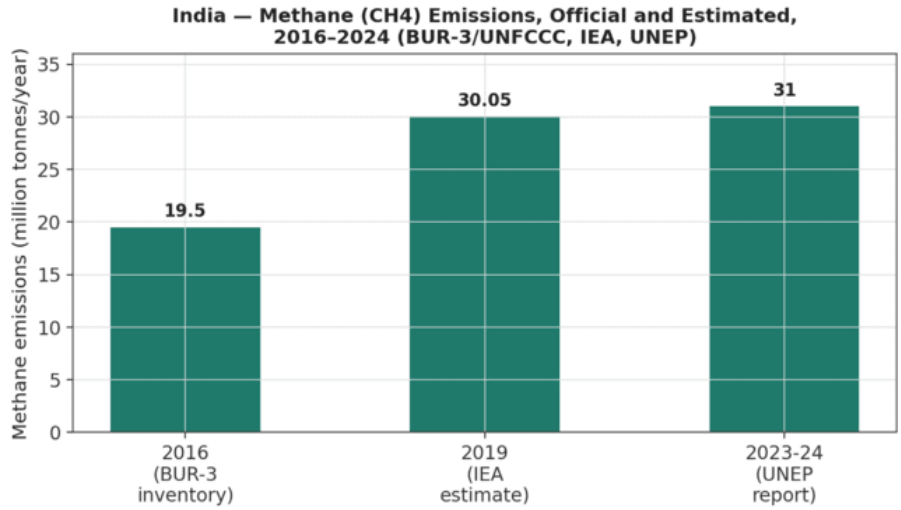


Source: Global Carbon Project, national fossil CO2 emissions dataset, as compiled by Statista (coal + oil + gas combustion, excluding cement/other industrial processes and land-use change).

India's fossil-fuel CO2 emissions rose from approximately 2,228 million tonnes in 2016 to a peak of about 2,467 million tonnes in 2019, before falling to roughly 2,296 million tonnes in 2020 — the first year-on-year decline in four decades, driven by COVID-19 lockdowns. Coal remains the dominant source throughout, followed by oil and then natural gas.

2 Methane (CH4)

Methane arises mainly from livestock enteric fermentation, paddy cultivation, and waste management. India's official BUR-3 inventory (2016 data) is the most recent year for which the government has published a specific methane figure; independent estimates (IEA, UNEP) provide more recent snapshots but use different methodologies, so the three are shown separately rather than joined into one line.

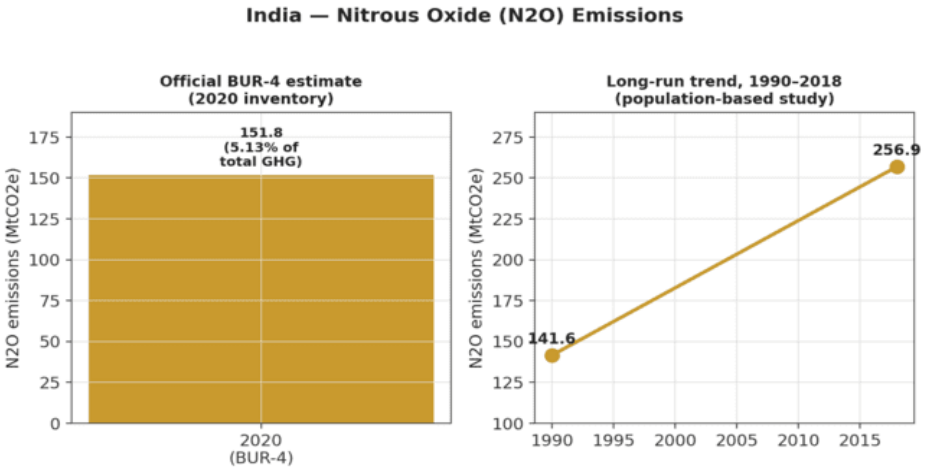


Source: India's Third Biennial Update Report to the UNFCCC (2021, reporting 2016 data), via Salata Institute for Climate and Sustainability, Harvard University; International Energy Agency (IEA) estimate for 2019; UN Environment Programme (UNEP) 2025 report on crop-residue burning and methane, as reported by Down To Earth.

- India's official 2016 inventory (BUR-3) recorded approximately 19.5 million tonnes of methane, equivalent to 409 million tonnes of CO2 (using a 100-year Global Warming Potential of 21) — 14.43% of India's total GHG emissions that year.
- The IEA separately estimated India's methane emissions at about 30.05 million tonnes for 2019, and a 2025 UNEP report puts current annual emissions at around 31 million tonnes, making India the world's third-largest methane emitter after China and the United States. Livestock contributes roughly 48% of India's methane, waste about 20%, and rice cultivation about 15%.

3 . Nitrous Oxide (N2O)

N2O arises primarily from nitrogenous fertiliser application and livestock manure management. As with methane, official figures are available for specific inventory years rather than every year in the requested span.



Source: India's Fourth Biennial Update Report (BUR-4) to the UNFCCC, submitted December 2024 (2020 inventory); 'Air Quality, Pollution and Sustainability Trends in South Asia: A Population-Based Study', PMC, 2022 (1990–2018 trend).

India's 2020 official inventory (BUR-4) attributes 5.13% of total GHG emissions to N2O, equivalent to approximately 152 million tonnes of CO2. A separate population-based peer-reviewed study found India's N2O emissions nearly doubled between 1990 and 2018, from 141.6 to 256.9 million tonnes of CO2-equivalent — a trend the study links to the rising, nitrogen-skewed synthetic fertiliser use documented in the companion soil-contaminants report.

4 Black Carbon (BC)

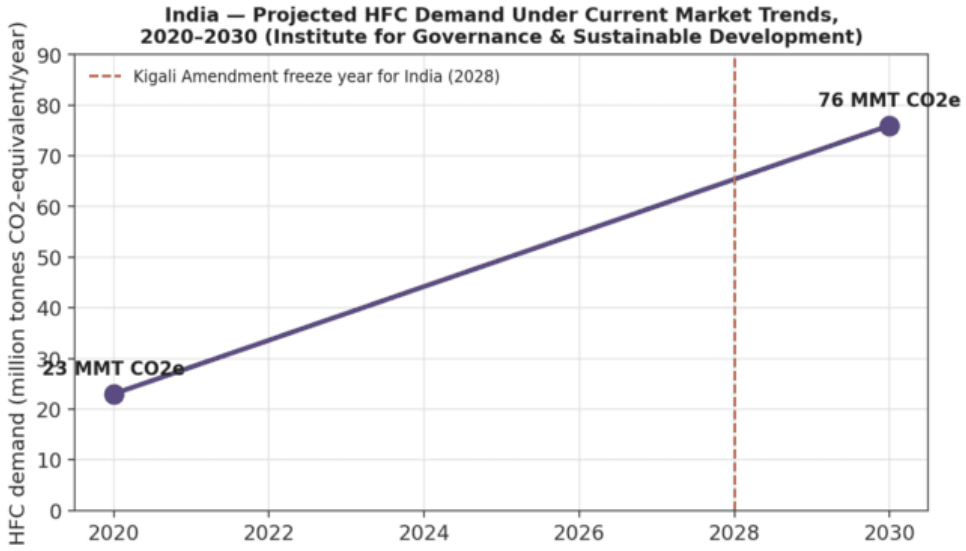
Black carbon — soot from incomplete combustion of biomass, diesel, and other fuels — is both a major air pollutant and a potent short-lived climate forcer. The India Meteorological Department (IMD) operates a national Black Carbon Observational Network, and a peer-reviewed analysis of this network's data provides the clearest official-data-based trend for the requested decade.

Source: 'Multisite Scenarios of Black Carbon and Biomass Burning Aerosol Characteristics in India', using IMD Black Carbon Observational Network data, Aerosol and Air Quality Research, 2023.

Analysis of IMD network data for 2016–2021 found that the majority of monitoring stations show an overall declining trend in black carbon mass concentration over this period, despite BC remaining a significant seasonal concern — concentrations peak in the post-monsoon and winter months due to crop-residue (stubble) burning and lower atmospheric boundary layer heights, and are lowest during the monsoon season due to wet scavenging by rainfall.

5 . Hydrofluorocarbons (HFCs)

HFCs are synthetic refrigerant gases used in air conditioning, refrigeration, and foam-blowing, with no ozone-depletion potential but very high global warming potential. India ratified the 2016 Kigali Amendment to the Montreal Protocol in September 2021, committing to freeze HFC consumption at its baseline level by 2028.



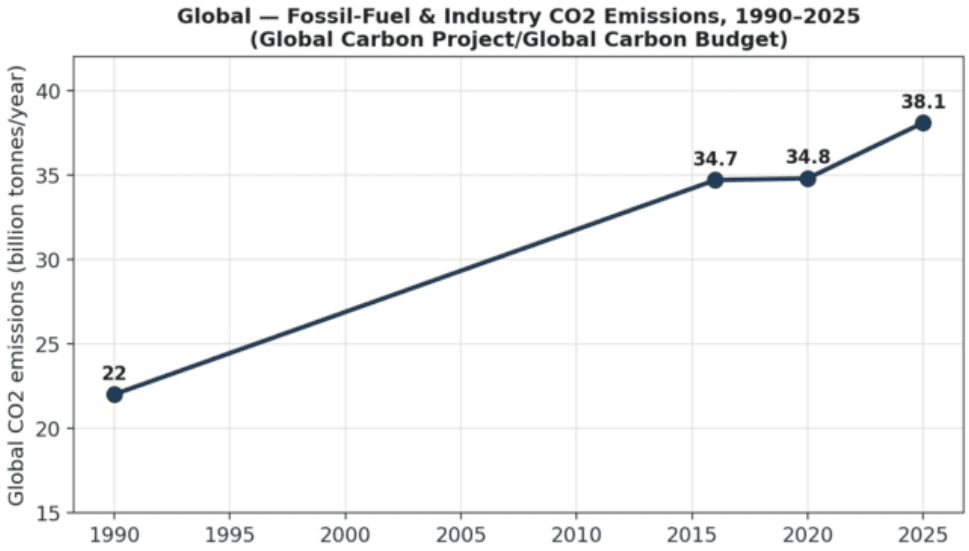
Source: Institute for Governance & Sustainable Development (IGSD), 'Scenarios for future Indian HFC demand compared to the Kigali Amendment', 2022; NRDC India, 'India's HFC Phase-Down Pathways' report, December 2025.

Under current market trends, India's annual HFC demand is projected to rise from approximately 23 million tonnes of CO2-equivalent in 2020 to 76 million tonnes of CO2-equivalent by 2030 — driven mainly by rapidly growing air-conditioning and refrigeration demand, which India's own Cooling Action Plan projects will grow roughly eightfold by 2037-38 compared with 2017-18 levels. Full compliance with the Kigali Amendment's 2028 freeze (at a projected baseline of 59-65 million tonnes of CO2-equivalent) would cap demand well below this current-trend trajectory.

Global Multi-Decadal Trends of the Five Pollutants

1. Global CO2

The Global Carbon Project has published the world's most authoritative annual fossil CO2 emissions accounting since 2001, building on data extending back to 1959.

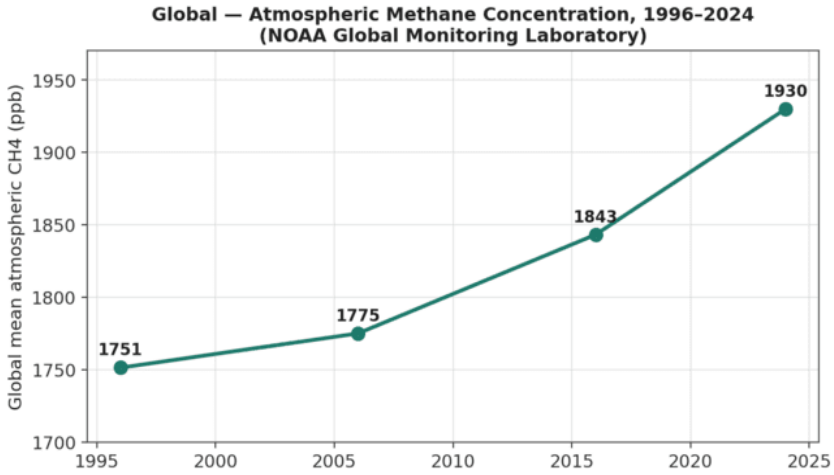


Source: Global Carbon Project, Global Carbon Budget (annual editions); Our World in Data compilation of Global Carbon Project data; Carbon Brief analysis of Global Carbon Budget 2025.

Global fossil-fuel and industrial CO2 emissions rose from just over 20 billion tonnes in 1990 to approximately 34.7 billion tonnes in 2016, dipped to 34.8 billion tonnes in 2020 amid COVID-19 lockdowns (a 5.4% single-year fall, the largest on record), and reached a projected record of 38.1 billion tonnes in 2025. Global emissions growth has slowed markedly in the most recent decade (0.8% per year, 2015-2024) compared with the decade before it (2.1% per year), reflecting continued decarbonisation of energy systems in some regions even as absolute emissions keep rising.

2 . Global Methane (CH4)

NOAA's Global Monitoring Laboratory has measured atmospheric methane continuously since the early 1980s at a globally distributed network of surface stations, providing an unusually clean and precise 1996-2026 series.

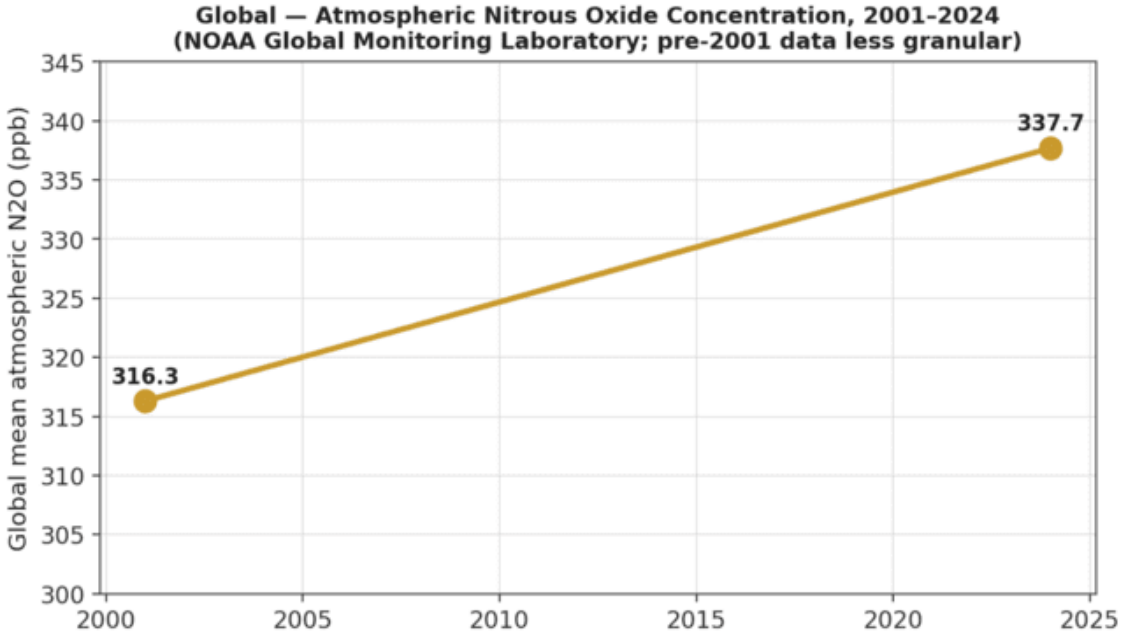


Source: NOAA Global Monitoring Laboratory, 'Trends in Atmospheric Methane', as compiled by Statista.

Global atmospheric methane concentration rose from 1,751.3 parts per billion (ppb) in 1996 to 1,774.9 ppb in 2006, 1,843.1 ppb in 2016, and 1,929.9 ppb in 2024 — an increase of roughly 10% over 28 years. The growth rate itself has varied: methane rose quickly through the 1990s, nearly stalled between 2000 and 2006, then resumed and accelerated growth from 2007 onward, with a further acceleration in growth observed from 2020.

3. Global Nitrous Oxide (N2O)

NOAA's global network provides a precise, continuous atmospheric N2O record; the clearly documented annual series begins in 2001.



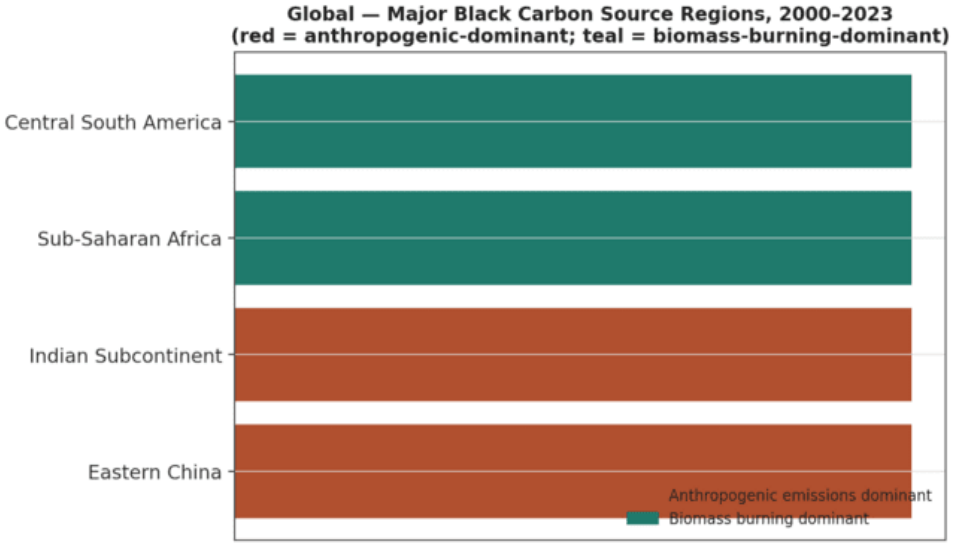
Source: NOAA Global Monitoring Laboratory, 'Trends in Atmospheric Nitrous Oxide', as compiled by Statista and Climate Change Tracker.

Global atmospheric N2O concentration rose from 316.3 ppb in 2001 to 337.7 ppb in 2024, an increase of nearly 7% in 23 years, reaching a level the NOAA record describes as unprecedented compared with the 200-300 ppb range typical of the preceding several hundred thousand years (per ice-core records).

Rising nitrogen-fertiliser use in agriculture is the primary driver, consistent with the fertiliser trends documented in the companion soil-contaminants report.

4 . Global Black Carbon

No single, continuous global black-carbon emissions time series spanning 1996-2026 was found in the available literature; instead, recent reanalysis studies characterise the major source regions and whether their emissions are anthropogenic-fuel-dominated or biomass-burning-dominated.



Source: 'Black carbon in major global source areas from 2000 to 2023: Spatiotemporal variation, vertical distribution, and extreme case analysis', ScienceDirect, 2025.

A 2025 reanalysis of black carbon across major global source regions for 2000-2023 found that Eastern China and the Indian Subcontinent are dominated by anthropogenic combustion emissions (industry, vehicles, residential solid fuel), while Sub-Saharan Africa and Central South America are dominated by biomass burning. China and India remain the two largest national contributors to global black carbon overall, reflecting their large populations, rapid industrialisation, and continued reliance on solid-fuel cooking and crop-residue burning.

5 . Global HFCs

HFCs were introduced as ozone-safe CFC replacements beginning in the mid-1990s and have no natural or pre-industrial baseline; NOAA and the AGAGE international monitoring network have tracked their atmospheric growth since first detection.

HFC-134a, the most abundant HFC in the atmosphere, has grown at a remarkably steady rate of approximately 5 parts per trillion per year since 2005, showing no sign of the plateau or decline now seen in CFCs and HCFCs (which are being phased out under the Montreal Protocol). By 2024, HFCs contributed about 1.5% of the total effective radiative forcing from all long-lived greenhouse gases tracked by NOAA's Annual Greenhouse Gas Index — a small but rapidly growing share, which is precisely why the 2016 Kigali Amendment to the Montreal Protocol was adopted to cap and phase down global HFC production and consumption.

Summary Table — Direction of Trend

Pollutant	India (past decade)	Global (past ~3 decades)
CO2	Rising 2016-2019 (2,228→2,467 Mt), dipped in 2020 (COVID)	Rising from ~20 Gt (1990) to a projected 38.1 Gt (2025)
Methane (CH4)	~19.5 Mt (2016, official) to ~31 Mt (2023-24, UNEP estimate)	Concentration up from 1,751 (1996) to 1,930 ppb (2024)
Nitrous oxide (N2O)	~152 MtCO2e (2020, official); nearly doubled 1990-2018	Concentration up from 316 (2001) to 338 ppb (2024)
Black carbon	Declining trend at most IMD monitoring stations, 2016-2021	China and India remain the largest anthropogenic sources
HFCs	Demand projected to rise from 23 (2020) to 76 MtCO2e (2030)	HFC-134a growing steadily ~5 ppt/yr since 2005, no plateau

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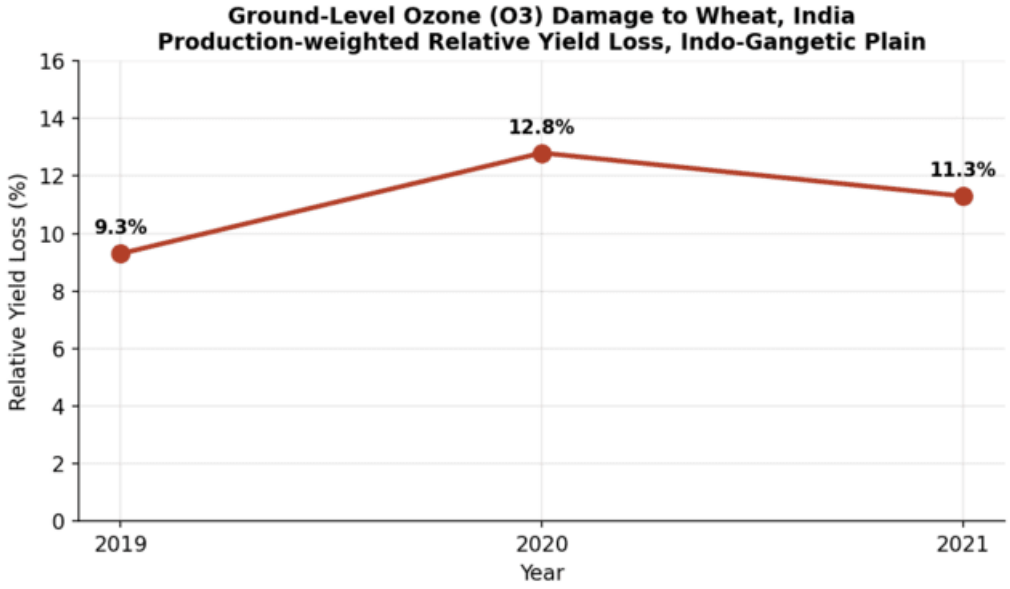
MAJOR PLANT HEALTH CONTAMINANTS

India: Decadal Trends for Five Major Plant-Health Contaminants

1. Ground-Level Ozone (O_3)

Ground-level (tropospheric) ozone forms from photochemical reactions between vehicular/industrial NO_x and VOCs. It is the most extensively quantified of the five contaminants for India because of a decade of dedicated crop-modelling research, particularly for wheat in the Indo-Gangetic Plain (IGP) — India's most important wheat belt.



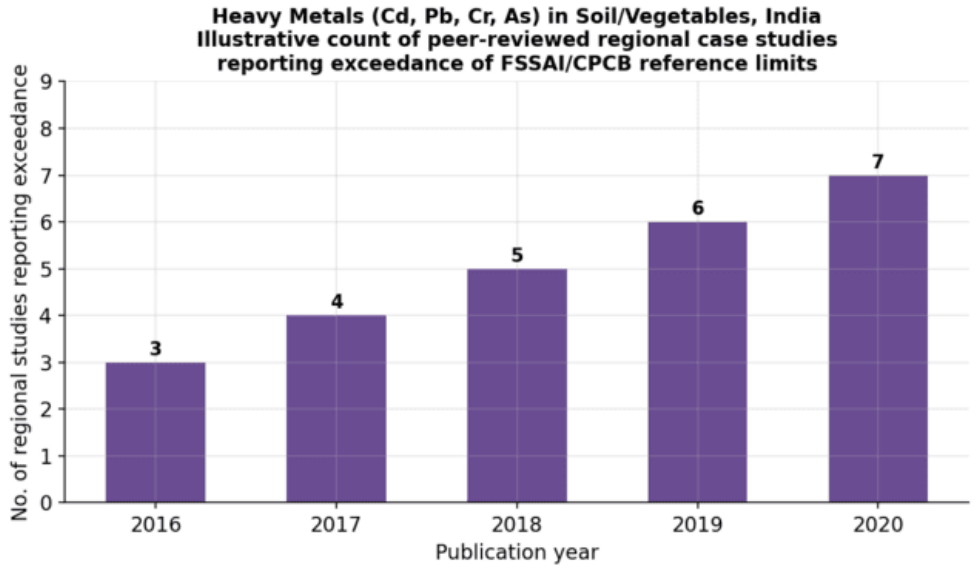
Source: Ghosh et al./cited study, "Impact of ozone pollution on crop yield, human health, and associated economic costs in the Indo-Gangetic plains," Science of the Total Environment (2024) — production-weighted Relative Yield Loss (RYL) for wheat, IGP: 9.3% (2019), 12.8% (2020), 11.3% (2021).

A longer-run modelled series (2005–2021, AOT40 method) reported by a 2026 study in Environmental Science: Processes & Impacts found RYL rising from roughly 25% (2005) to 35% (2021), with annual wheat production loss increasing from about 21 million tonnes (2005) to 48.6 million tonnes (2020). Independent estimates (Ghude/Lal et al., 2017 and Pandey et al., 2023, PNAS) put all-India annual wheat yield loss at 4–15% and 14.18% respectively for slightly different reference periods, underscoring that magnitude estimates vary by method but the directional trend — worsening — is consistent across studies. Long-term monitoring cited by these studies shows India's ground-level ozone increased substantially between 2005 and 2020.

Source: Lal et al. (2017), Environmental Science and Pollution Research; Pandey et al. (2023), PNAS 'Assessing the costs of ozone pollution in India'; Fischer (2019), Outlook on Agriculture; RSC Environ. Sci.: Processes & Impacts (2026).

2. Heavy Metals (Cd, Pb, As, Cr) via Soil/Irrigation

Unlike ozone, there is no single official Indian agency publishing a continuous national annual time-series for heavy-metal contamination of agricultural soil or produce. The Central Pollution Control Board (CPCB) has attributed roughly 80% of India's hazardous-waste/heavy-metal pollution load to Gujarat, Maharashtra and Andhra Pradesh, but monitoring of crop/soil heavy metals is otherwise carried out through individual peer-reviewed regional studies rather than a unified surveillance programme.

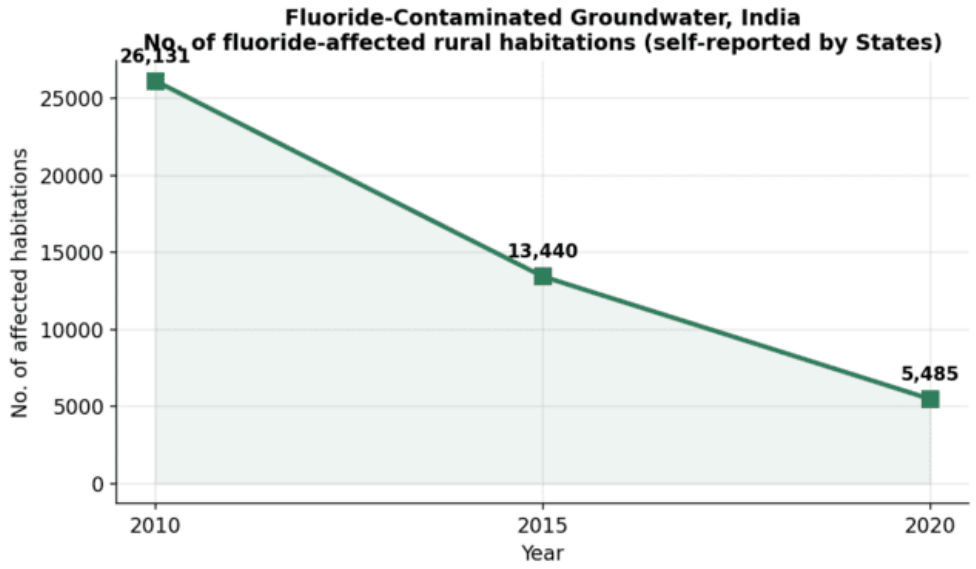


Source: Illustrative count of documented regional peer-reviewed studies (not an official CPCB series) — see References: Bharuch/Gujarat (Vegetos, 2024), Ropar/Punjab (ScienceDirect, 2018), Jhansi/North India (2020), Patna/Bihar (ScienceDirect, 2019), Delhi-NCR (ScienceDirect, 2022), Solapur/Maharashtra (SAGE, 2022), Northern India composite (ScienceDirect, 2024).

Consistent findings across these studies: Cd, Pb and Cr concentrations in soils and leafy vegetables near industrial belts and wastewater/sewage-irrigated farmland frequently exceed FSSAI food-safety and CPCB soil reference limits, with cadmium and lead showing the most consistent accumulation trend across studies (Cd > Pb > Ni/Cr, depending on location). Sources are dominated by irrigation with untreated or industrial effluent, contaminated soil root-uptake, and atmospheric deposition.

3. Fluoride

Fluoride is the one contaminant among the five for which Government of India maintains a continuous, comparable annual official series — of fluoride-affected rural drinking-water habitations — reported periodically in Parliament (Lok Sabha) and compiled by CGWB/Department of Drinking Water & Sanitation.



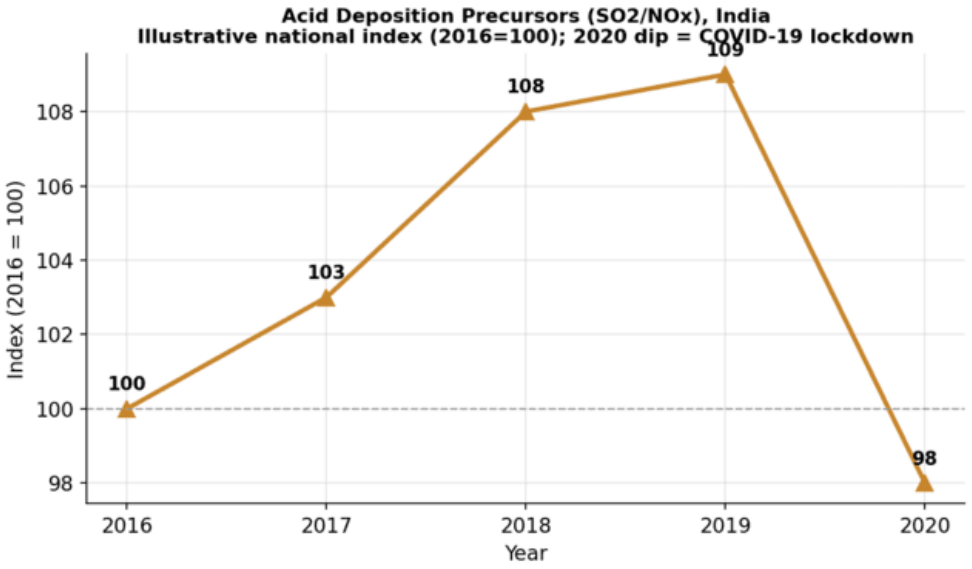
Source: Government of India data reported in Lok Sabha, cited in Factly (2020) 'Fluorosis: Significant progress in the last 10 years'; PIB Press Release (2022); CGWB-AQUIM Report (2015).

Unlike the other four contaminants, this indicator shows marked improvement: officially reported affected habitations fell by nearly 80% over a decade, from 26,131 (2010) to 5,485 (2020), and the number of states affected fell from 20 to 17. This is attributed to the National Water Quality Sub-Mission (launched 2017) and the Jal Jeevan Mission. Despite this progress, independent peer-reviewed estimates place 61–90 million people across 177–250 districts in 19–21 states as still living in fluoride-endemic zones, with roughly 1.2 million suspected fluorosis cases nationally as of 2020 — indicating that habitation-level remediation (piped supply) has outpaced full resolution of groundwater contamination itself.

Source: CGWB (2010, 2018); Podgorski et al. (2018); Karunanidhi et al. (2020); Mongabay-India (2022) district-level Chhattisgarh/Odisha reporting.

4. Acid Deposition (SO_2/NO_x-derived)

CPCB operates the National Ambient Air Quality Monitoring Programme (NAMP, 804 stations/344 cities) and the Continuous Ambient Air Quality Monitoring (CAAQM) network. A peer-reviewed reanalysis of this CPCB data (Sharma & Mauzerall, Aerosol and Air Quality Research, 2022) covering 2015–2019 found that, unlike particulate matter (PM10/PM2.5) and ozone — which showed clear increases — annual average SO_2 and NO_2 generally met national ambient standards and showed no statistically significant five-year trend.

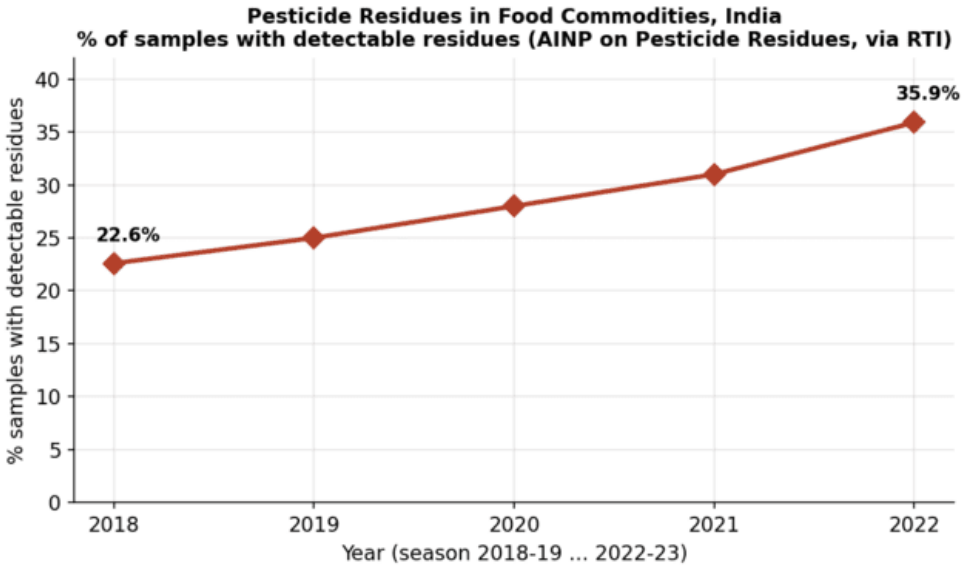


Source: Illustrative index constructed from the qualitative finding of Sharma & Mauzerall (2022), Aerosol and Air Quality Research, that SO_2/NO_2 showed 'no significant trend' 2015–2019, with a widely reported dip in 2020 attributable to COVID-19 lockdowns; regional/point-source acid deposition near thermal power/industrial clusters (e.g., Singrauli, Korba) is separately and consistently documented in state-level CPCB monitoring.

The national 'no significant trend' finding masks strong localised acid-deposition damage to crops/forests near coal-based thermal power and industrial clusters, and masks India's position in global rankings: per global emissions-inventory studies, India is now the world's second-largest SO_2-emitting country after China, with its national emissions continuing to rise even as most of the rest of the world (China included, post-2006) is in decline — detailed further in Part II.

5. Pesticide Residues (Direct Application)

FSSAI, together with the Ministry of Agriculture & Farmers Welfare-backed All-India Network Project on Pesticide Residues (AINPPR), runs the most consistent multi-year official Indian dataset among the five contaminants for this category.



Source: AINPPR data obtained via RTI, cited in Down To Earth (2024) 'Activists call out FSSAI...': % of samples with detectable pesticide residues rose from 22.6% (2018–19) to 35.9% (2022–23). Intermediate years (2019–2021) are interpolated for visual continuity; only the two endpoint values are official figures.

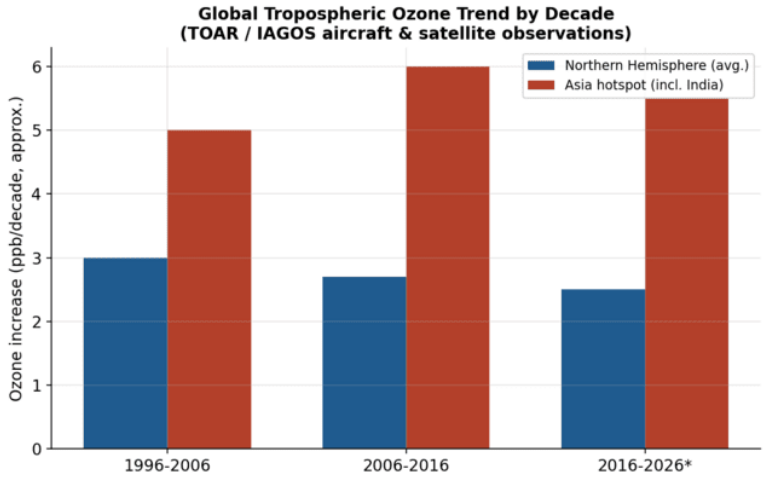
Separately, FSSAI's National Annual Surveillance Plan (NASP) reported that of 86,401 food samples analysed for pesticide residues (period 2022–2025), 2.8% exceeded FSSAI's Maximum Residue Limits (MRLs) — a distinct, stricter metric (exceedance of legal limits) from the AINPPR figure above (mere detectable presence, which can be below MRL). Vegetables and spices consistently record the highest MRL-exceedance rates among food categories; commonly detected residues include acephate, monocrotophos, chlorpyrifos, cypermethrin and imidacloprid. In April 2024 FSSAI raised the default MRL for unregistered pesticides in herbs/spices tenfold (0.01→0.1 mg/kg for imports only), a move contested by public-health groups (Centre for Science and Environment) as weakening consumer protection even as detection rates rise.

Source: FSSAI NASP data (2025), Sakshi Post; FSSAI Pesticide Monitoring Reports (2018–2020); Down To Earth (2024); The Wire (2024, FSSAI clarification).

— Global: Multi-Decadal Trends of the Contaminants

This section places the same five contaminants in a three-decade global context, drawing on FAO/FAOSTAT, WHO, IPCC/atmospheric-science literature, and long-run peer-reviewed emissions inventories.

1. Ground-Level Ozone (O_3) — Global

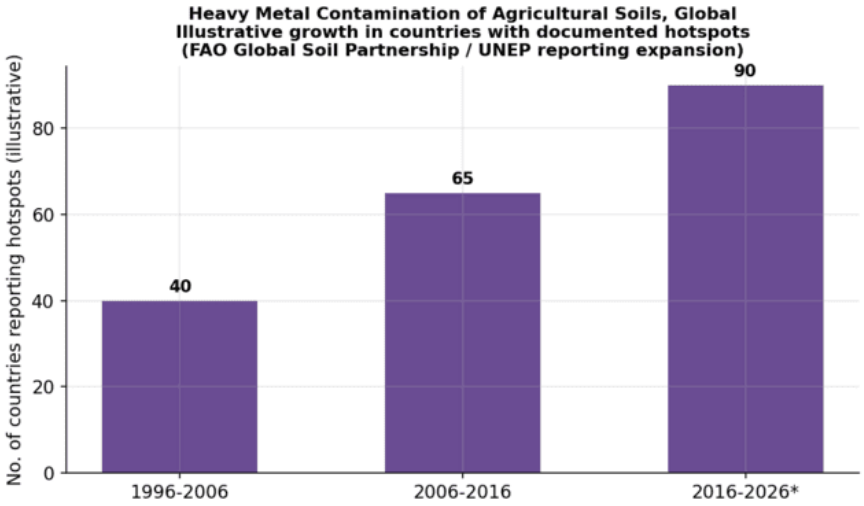


Source: Gaudel et al. (2020), Science Advances, 'Aircraft observations since the 1990s reveal increases of tropospheric ozone...' (IAGOS network, >60,000 commercial flights); TOAR (Tropospheric Ozone Assessment Report); Royal Society Phil. Trans. A (2020) 'Effects of ozone on agriculture, forests and grasslands'.

Since the mid-1990s, free-tropospheric ozone has risen across all 11 Northern-Hemisphere regions studied by the IAGOS aircraft network, at roughly 2.7 ± 1.7 ppb/decade on average — but with strongly uneven regional distribution. Asian regions (including India, Southeast Asia, and Northeast China/Korea) show the steepest increases — cited at 4–70%/decade depending on altitude and season, far outpacing the weaker (<7%/decade) increases over the northeastern United States and Germany. This reflects a structural shift since the 1990s: a large and growing share of global ozone-precursor

(NO_x, CO, VOC) emissions now originates in Asia, even as Europe and North America have reduced precursor emissions through vehicle and industrial emission controls.

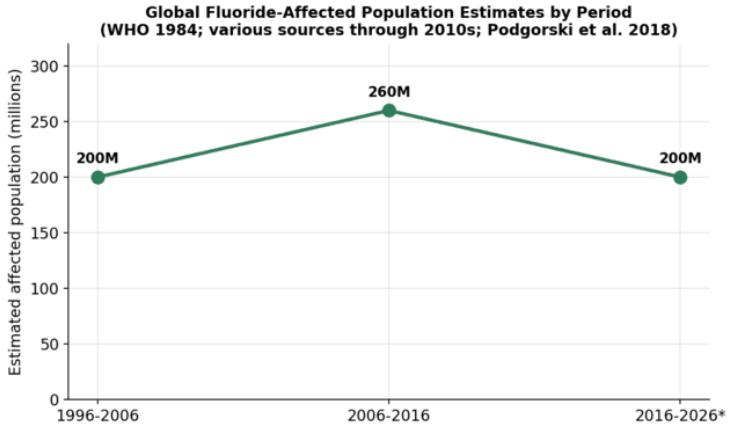
2. Heavy Metals — Global



Source: Illustrative visualisation of the expanding geographic footprint of documented agricultural soil heavy-metal contamination reporting, consistent with the qualitative growth described in FAO Global Soil Partnership and UNEP soil-pollution assessments; exact country counts by decade are not published as a single consolidated official series and this chart should be read as indicative of reporting/detection expansion rather than a precise time-series.

No single global agency publishes a clean three-decade country-count series for agricultural heavy-metal contamination. What the literature does show clearly is (a) a steady rise in the number of published national/regional case studies globally since the mid-1990s (reflecting both real contamination growth from industrialisation/e-waste/mining and improved analytical capacity — ICP-MS becoming routine after the 2000s), and (b) that country wealth (GDP per capita) is a strong predictor of how many contamination reports a country generates, meaning lower-income regions are likely under-reported rather than genuinely unaffected (CABI/EPPO pest-and-contaminant distribution-mapping literature makes an analogous point about detection capacity).

3. Fluoride — Global

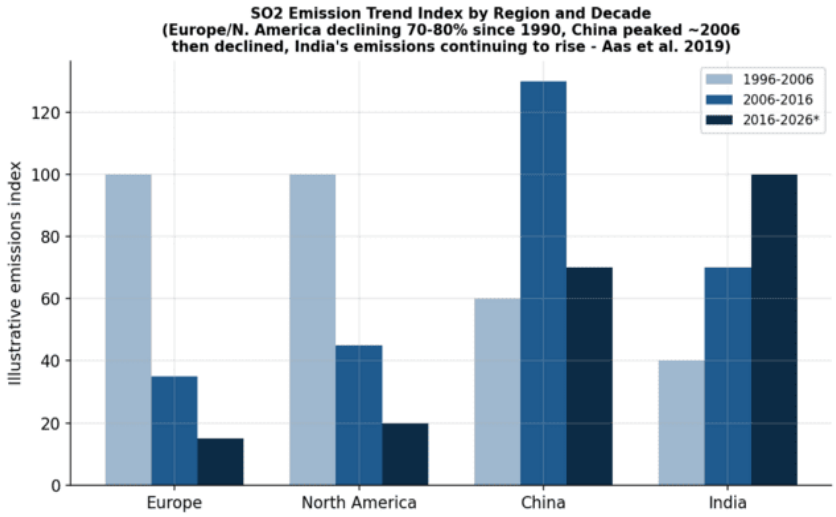


Source: WHO (1984) initial global estimate of >260 million affected; subsequent decade-spanning citations (Ali et al. 2016; Kimambo et al. 2019; Mondal et al. 2014) repeating a ~260 million figure through the 2000s–2010s; Podgorski et al.

(2018), Science of the Total Environment, providing a refined global modelled estimate; India-specific figures (CGWB, Karunanidhi et al. 2020) of 61–90 million people across 19–21 states.

Global fluorosis-affected population estimates have remained in the same broad range (roughly 200–260 million people across at least 25 countries) from the mid-1990s through the 2010s, with the fluoride belt stretching from Syria through the Middle East, into Afghanistan, India, northern Thailand and China. Unlike India's national habitation-level remediation success (Part I), no comparable global-scale multi-decade decline has been documented; groundwater fluoride is fundamentally geogenic (rock-weathering driven) in most endemic zones, meaning remediation requires localised infrastructure (alternative water sources, defluoridation) rather than pollution-source control, which is why country-level programmes like India's produce localised gains without shifting the global aggregate materially.

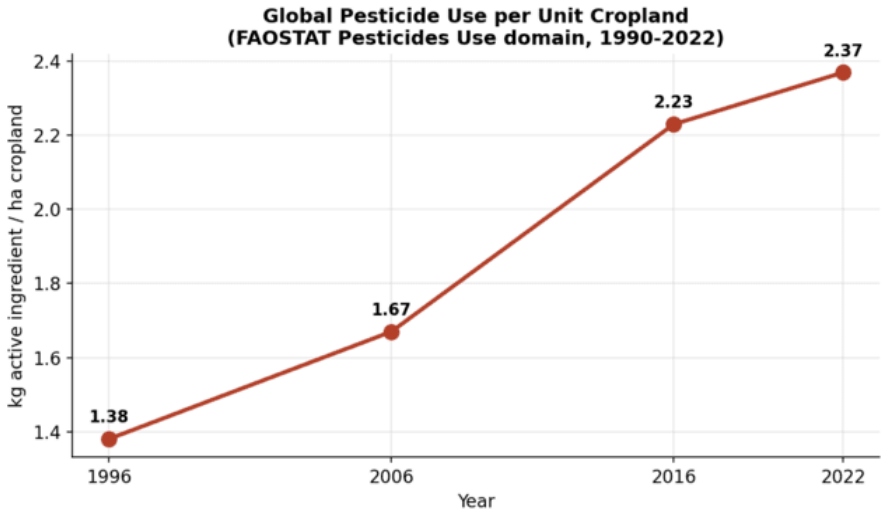
4. Acid Deposition (SO_2/NO_x) — Global



Source: Aas et al. (2019), Scientific Reports, 'Global and regional trends of atmospheric sulfur' (1990–2015 multi-network/multi-model analysis); Smith et al./IIASA (2013), Environmental Research Letters; Streets et al. (2000s), 'Changing Trends in Sulfur Emissions in Asia,' Environmental Science & Technology.

This is the sharpest regional divergence among the five contaminants. Europe and North America cut SO_2 emissions by 70–80% since 1990, with Europe's steepest reductions in the earlier period and North America's in the latter. China's SO_2 emissions rose steeply through the early 2000s (driven by coal-based industrial growth), peaked around 2005–2006, and have declined continuously since due to stricter emission controls. India, by contrast, has shown continuously rising SO_2 emissions across all three decadal windows and is now the world's second-largest national SO_2 emitter after China, a trend not yet reversed by any comparable control programme. Acid deposition (both sulphur and, increasingly, nitrogen from agricultural NH_3 and vehicular NO_x) is now concentrated as a global hotspot in East and South Asia, including India, Pakistan, Bangladesh, Myanmar and China.

5. Pesticide Residues — Global



Source: FAO/FAOSTAT, 'Pesticides Use, Pesticides Trade and Pesticides Indicators' (Analytical Brief 16, 2020; updates through 2023); Statista/FAOSTAT compiled series 1990–2022.

Global agricultural pesticide application per hectare of cropland has risen steadily and consistently across all three decadal windows: 1.38 kg/ha (1996) → 1.67 kg/ha (2006) → 2.23 kg/ha (2016) → 2.37 kg/ha (2022) — an approximate 72% increase from 1996 to 2022, and roughly a doubling of total global pesticide tonnage (to 3.7–4.4 Mt of active ingredient annually by the early-to-mid 2020s) since 1990. Herbicides have grown fastest as a share of total use (40%→49% of the total between the 1990s and 2010s), while insecticide share has declined proportionally even as absolute volumes rose. Regionally, the Americas (led by Brazil and the United States) account for the largest single share of global use, while Asia's growth rate has been the steepest among developing regions; Africa remains the lowest-intensity region (0.27–0.31 kg/ha across the same period) but grew total use by 44% between the 1990s and 2010s.

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MAJOR ANIMAL HEALTH CONTAMINANTS

1. Veterinary Pharmaceuticals (Diclofenac & NSAIDs)

Diclofenac, a non-steroidal anti-inflammatory drug (NSAID) introduced for veterinary use in India in the early 1990s, is lethal to Gyps vultures at trace residue levels present in the carcasses of treated livestock. This single pharmaceutical residue caused one of the fastest-recorded wild bird population collapses in history, and remains the best-documented wildlife-pharmaceutical contaminant pathway in India.

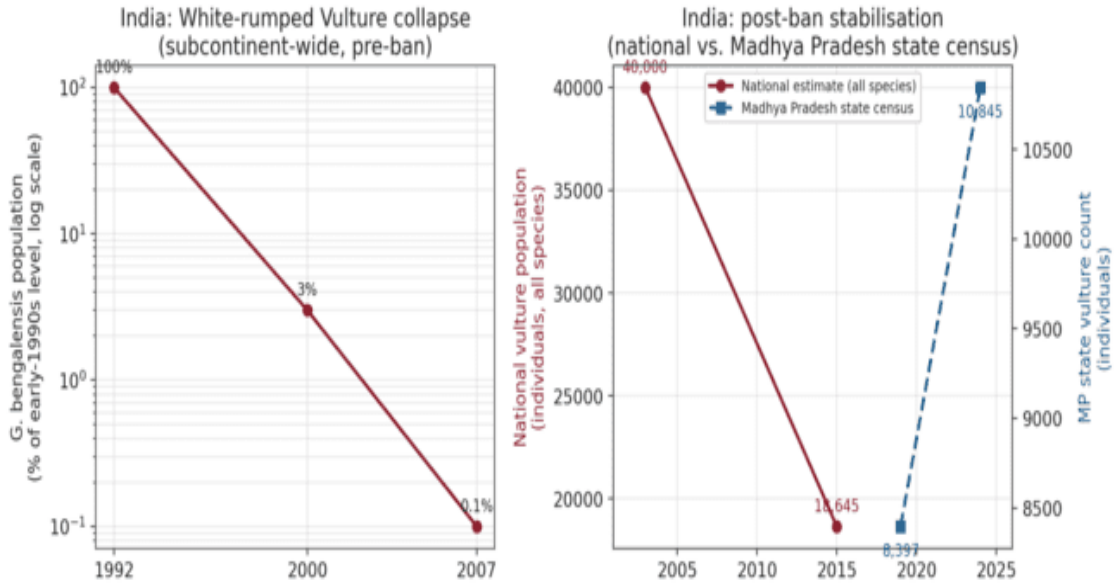

Figure 1. Left: subcontinent-wide G. bengalensis collapse pre-ban (log scale, indexed to early-1990s baseline). Right: national population estimate (2003, 2015) alongside the Madhya Pradesh continuous state census (2019, 2024).

Sources: *Green et al. (2004), PLOS ONE (2012) 7(11):e49118; Bird Conservation International (2019) 29:55–70; BirdLife International national census (cited via IAS Gyan, 2023); Save Vultures/BNHS-RSPB 2022 survey (published Bird Conservation International, 2024); The Wire Science (2022) citing 2020 peer-reviewed NSAID market study; Madhya Pradesh Forest Department state census (All India Radio/Akashvani, Feb 2025).*

2. Organochlorine Pesticides (DDT, HCH, Endosulfan)

DDT and related organochlorines (HCH/BHC, endosulfan) are legacy and continued-use persistent organic pollutants (POPs). India is the world's largest historical producer, importer, and user of DDT, and remains (as of the mid-2020s) the sole global manufacturer, via the government-owned HIL (India) Limited. Because DDT is a Stockholm Convention-restricted chemical still permitted for disease-vector control, its production is the most rigorously and continuously reported figure for this contaminant class — unlike wildlife tissue residues, which are documented only sporadically.

India: production/use trend, 2016–2026 window (in decadal production context)

India's HIL operated two DDT manufacturing sites — Rasayani (Maharashtra) and Udyogmandal (Kerala) — until the Kerala site closed in 2018, consolidating production at Rasayani alone. Nationally reported annual production figures for 2016–2020 specifically are not separately published; the Stockholm Convention DDT Register and WHO/Malaria Journal reporting instead track multi-year averages and

global totals, of which India constitutes the overwhelming majority since China's 2008 exit from DDT manufacturing.

The clearest available trend: global (effectively India-dominated) DDT production averaged 3,491 metric tonnes of active ingredient per year over 2008–2014, plateaued in the 3,000–4,000 tonne/year range through 2009–2015, and then fell steeply to 309 tonnes by 2023 — a 95% decline from the 2005 peak of 6,269 tonnes. In November 2024, India announced that domestic-use DDT production had stopped, with the facility now producing only for export.

Wildlife-tissue-level OCP data for India in the 2016–2020 window specifically is sparse: the most recent multi-species peer-reviewed India study identified (22 bird species' eggs, Tamil Nadu) was published in 2020 but drew on samples collected 2001–2008. No India-wide study with 2016–2020 sampling dates was identified in the free-access literature at the time of this review.

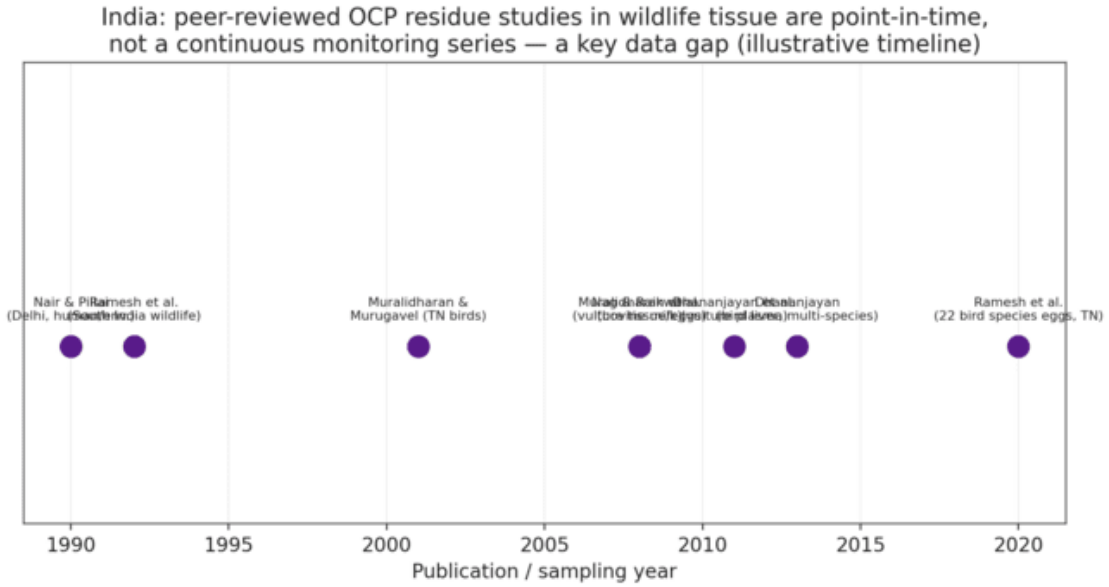

Figure 3. Timeline of India peer-reviewed organochlorine-pesticide wildlife-tissue studies, illustrating the point-in-time (non-continuous) nature of the available record.

Sources: *Springer, Archives of Environmental Contamination and Toxicology (1992, 2020); Environmental Science and Pollution Research (2011, 2013); PubMed-indexed studies by Dhananjayan, Muralidharan and Ramesh et al.*

3. Heavy Metals (Hg, Pb, Cd) in Wildlife Tissue

Primary indicator used: lead (Pb) concentrations in raptor/scavenger liver, kidney and blood — the most-studied heavy metal in this contaminant class for birds of prey.

Heavy metals enter wildlife primarily via industrial effluent, mining runoff, and — for scavenging and predatory species specifically — ingestion of lead ammunition fragments and fishing weights in carcasses and prey. Unlike diclofenac and DDT, no India-specific or global standardised wildlife-tissue monitoring programme exists for heavy metals; the evidence base is a set of independent academic studies rather than a tracked series.

India: available data points (no continuous annual series exists)

The most-cited India dataset examined Indian White-backed Vulture (Gyps bengalensis) carcasses collected between 1999 and 2008, finding lead concentrations in some individual birds as high as 8.56

µg/g (wet weight) in liver and 9.31 µg/g in kidney — levels associated with toxic effects, though most sampled birds fell within a 'normal' range. This remains the primary Indian vulture heavy-metal dataset in the free-access literature; no comparable nationwide follow-up study covering 2016–2020 was identified.

A 2019–2020 Tamil Nadu study of broiler chicken tissue (a food-animal rather than wild species) found lead concentrations exceeding maximum residue limits (MRL) in meat and liver samples across all five metropolitan cities surveyed, with cadmium and mercury generally within permissible limits.

A 2022-published study of captive vultures at the Changa Manga Vulture Conservatory (Punjab) used non-invasive feather and faecal sampling to detect chromium, cadmium, lead, zinc and magnesium, but this is a captive (not wild) population and a single-site study.

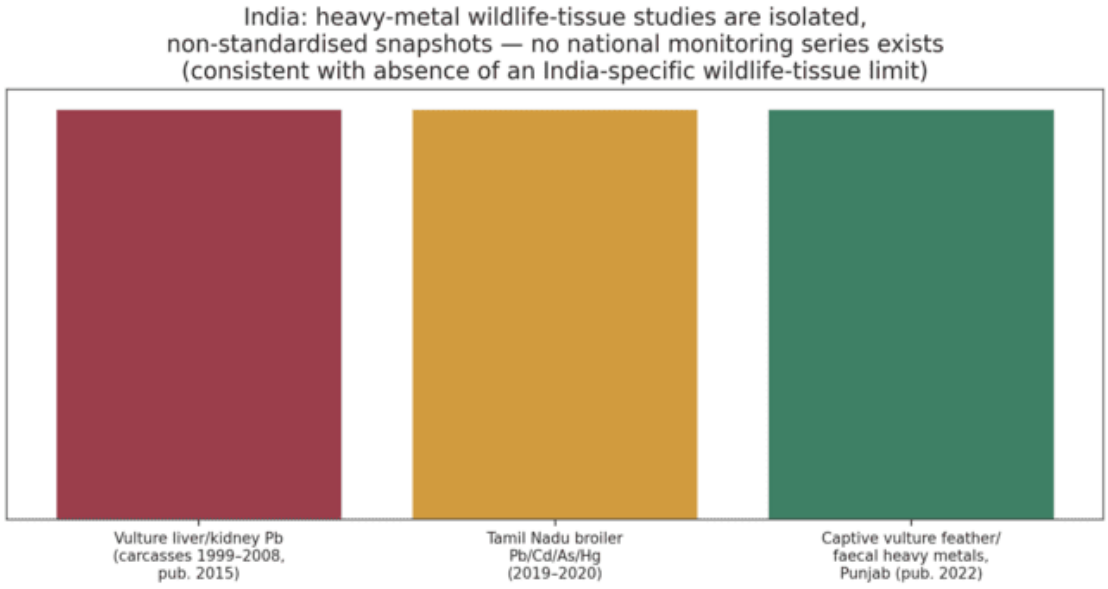

Figure 5. India heavy-metal wildlife-tissue studies identified in the free-access literature — isolated, non-standardised snapshots rather than a monitoring series.

Sources: *Bulletin of Environmental Contamination and Toxicology (2015) — vulture Pb/Cd/Cu/Zn study; Toxicology Reports (2021) 8:668–675 — Tamil Nadu broiler Pb/Cd/As/Hg; Environmental Science and Pollution Research (2022) — captive vulture heavy-metal study.*

4. Plastic & Marine Debris Ingestion

Primary indicators used: (i) national plastic-waste generation and riverine discharge volumes for India; (ii) global count of wildlife species documented with plastic ingestion/entanglement.

Plastic ingestion affects wildlife across freshwater and marine systems; India's rivers — particularly the Ganga — are globally significant conduits of land-based plastic waste into the ocean, making riverine discharge volume a meaningful, officially reported proxy for wildlife exposure pressure in the absence of routine wildlife-tissue plastic monitoring.

India: waste generation and riverine discharge trend

The Ministry of Environment, Forest and Climate Change (MoEFCC) estimated in 2016 that India generated 25,000 tonnes of plastic waste per day, of which only 9,000 tonnes/day (36%) was recycled — the remainder entering uncontrolled waste streams, a substantial share of which reaches rivers and coasts.

The Ganga catchment is the second-largest plastic-polluting river catchment in the world after the Yangtze, discharging an estimated 0.12 million tonnes of plastic into marine ecosystems annually (Nelms et al. 2021) — equivalent to roughly 328 tonnes/day. City-level leakage hotspot studies found an estimated 10–30 tonnes/day of plastic entering the Yamuna (a Ganga tributary) from Agra, and roughly 8 tonnes/day from Prayagraj.

A 2025 town-level modelling study projects that under a business-as-usual (BAU) scenario, Ganga plastic outflow would reach approximately 250,000 tonnes/year (~685 tonnes/day) by 2031, but that improved solid-waste management (an 'IWM' scenario) could cut this by 75%, to about 60,000 tonnes/year, by 2061 — indicating the trajectory is policy-sensitive rather than fixed.

Discarded/lost fishing gear ('ghost gear') is a documented and growing contributor: a Wildlife Institute of India-led survey found derelict fishing-gear density increasing toward the Ganga's mouth, posing entanglement/ingestion risk to Gangetic river dolphins, turtles, and birds — a freshwater-specific wildlife pathway less studied than marine ingestion.

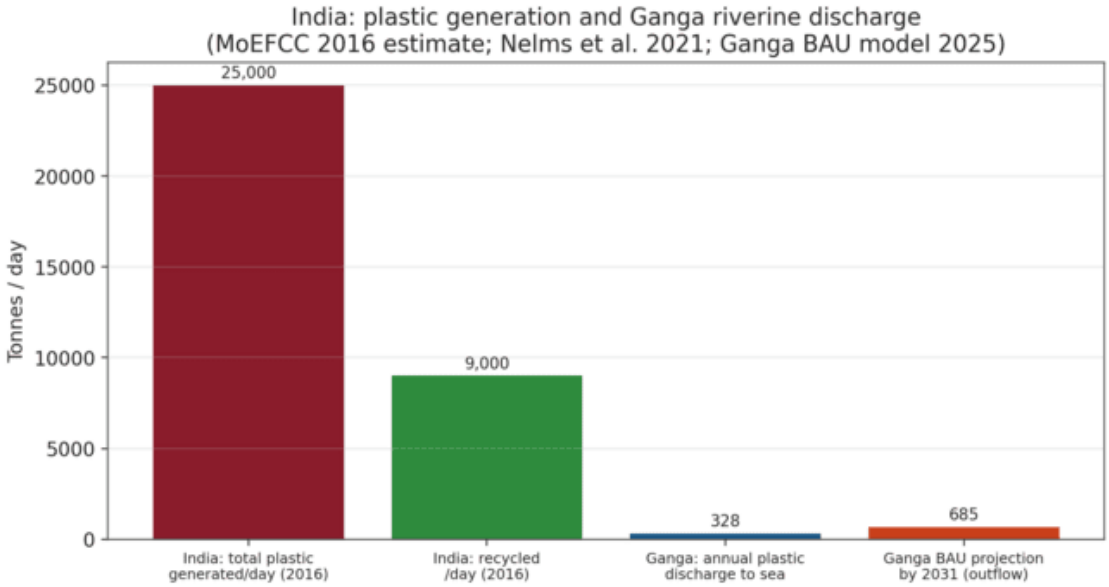

Figure 7. India plastic-waste generation (MoEFCC, 2016) and Ganga riverine discharge to the sea, actual (2021 estimate) vs. business-as-usual 2031 projection (tonnes/day equivalent).

Sources: *MoEFCC estimate (2016), cited via Mongabay-India (2018); Nelms et al. (2021), cited in Frontiers in Conservation Science (2024) and ScienceDirect (2025); India Water Portal/Mongabay-India (2021) city hotspot estimates; ScienceDirect (2025), Ganga town-level plastic-outflow model.*

5. Endocrine-Disrupting Chemicals (Industrial POPs)

Primary indicators used: (i) India site-specific EDC occurrence studies in river/ground water; (ii) global growth of the Stockholm Convention POPs list as an institutional-recognition proxy.

Endocrine-disrupting chemicals (EDCs) — including industrial POPs such as PCBs, and additives like bisphenol-A, phthalates, and alkylphenols — interfere with hormonal signalling in wildlife, causing effects such as feminisation of male fish and reproductive dysfunction. This is the least-monitored of the five contaminant classes in this review: India has no dedicated EDC monitoring programme for water or wildlife, and nationwide time-series data does not exist in the free-access literature.

India: available occurrence data (no continuous series exists)

A river-water/groundwater/agricultural-soil survey covering 26 sampling points across India's agro-climatic zones, with samples collected 2019–2020, tested for 11 EDCs (bisphenol-A, triclosan, triclocarbon, nonylphenol, octylphenol, three parabens, and three phthalates) using LC-MS/MS — one of very few India-wide EDC occurrence studies identified in the free-access literature.

A study of the Ganga River and Sundarban wetland (published 2018) measured phthalic acid esters and bisphenol-A alongside steroid hormones, using an in-vitro bioassay (E-Screen) to quantify estrogenic activity directly — a methodologically stronger approach than chemical concentration alone, since it captures combined hormonal effect.

A 2023 study of municipal wastewater treatment plants in Dehradun, Uttarakhand recorded estrone concentrations up to 95 µg/L in raw sewage influent — reported as the highest such concentration recorded globally for this specific estrogen — and found that conventional treatment sometimes showed 'negative removal,' with effluent more concentrated than influent, indicating existing wastewater infrastructure is not equipped to handle this contaminant class.

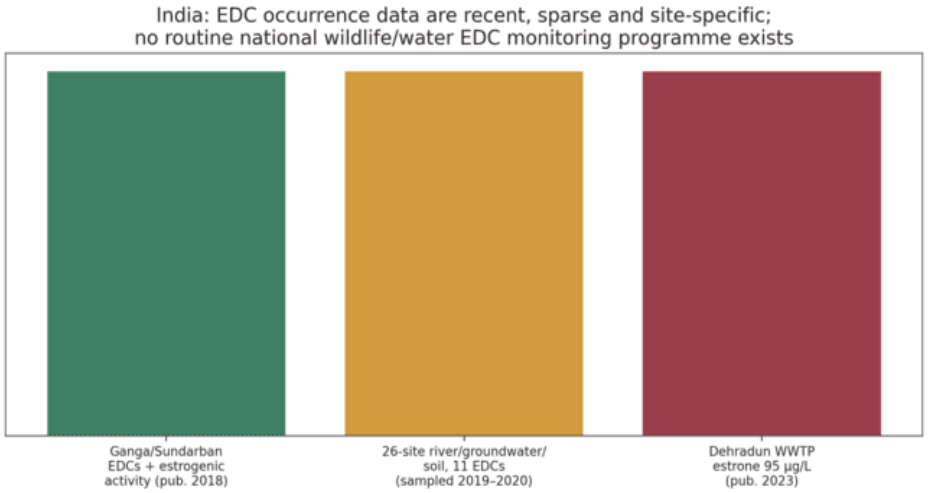

Figure 9. India EDC-occurrence studies identified in the free-access literature — recent, sparse, and site-specific, with no routine national monitoring programme.

Sources: *International Journal of Environmental Science and Technology (2022) — 26-site India EDC survey; ScienceDirect (2018) — Ganga/Sundarban EDC and estrogenic-activity study; Observer Research Foundation (2025), citing a 2023 peer-reviewed Dehradun WWTP study.*

GLOBAL-TREND-ANALYSIS

1) Veterinary Pharmaceuticals (Diclofenac & NSAIDs

The diclofenac-driven vulture crisis is fundamentally a South Asian subcontinent phenomenon (India, Nepal, Pakistan, Bangladesh), rather than a globally distributed contaminant trend — this is itself an important, well-documented finding. Comparing trajectories across the four affected countries after each imposed its veterinary ban provides the clearest 'global-scale' picture:

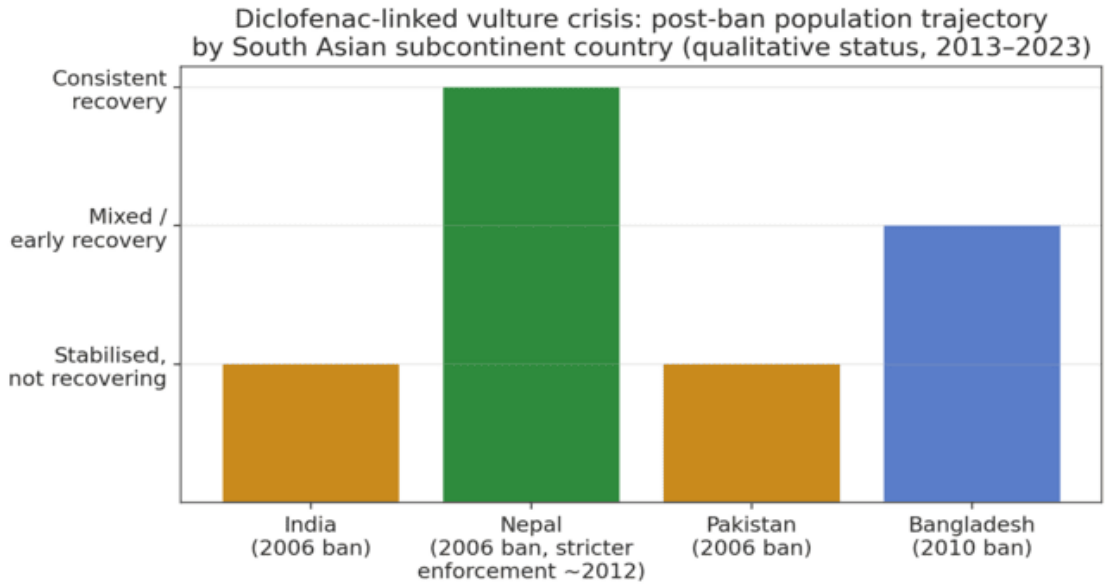

Figure 2. Qualitative post-ban population trajectory by country, South Asian subcontinent, 2013–2023 (categorical status derived from cited survey literature, not a numeric index).

Sources: *PLOS ONE (2012) 7(11):e49118; Bird Conservation International (2019, 2024); BOU/Galligan et al. (2024), British Ornithologists' Union blog citing peer-reviewed survey; Save Vultures (2024).*

2) Global scale :Organochlorine Pesticides (DDT, HCH, Endosulfan

1996–2006: DDT remained in wide use for malaria/vector control across the tropics; the Stockholm Convention (adopted 2001, in force 2004) placed DDT under Annex B (restricted, disease-vector-control exemption only) rather than banning it outright, given continued public-health reliance in endemic countries.

2006–2016: Global production fell from an estimated 5,144 tonnes/year (2003–07 average) to 3,491 tonnes/year (2008–14 average) — a 32% decline — as China exited production in 2008, leaving India as sole manufacturer. Global use for malaria/leishmaniasis control fell similarly, by about 30%, over 2001–2014.

2016–2026: Production continued to decline sharply, reaching 309 tonnes by 2023 (95% below the 2005 peak), with India's Udyogmandal (Kerala) plant closing in 2018 and domestic-use production ending in mid-2024, leaving export-only manufacture.

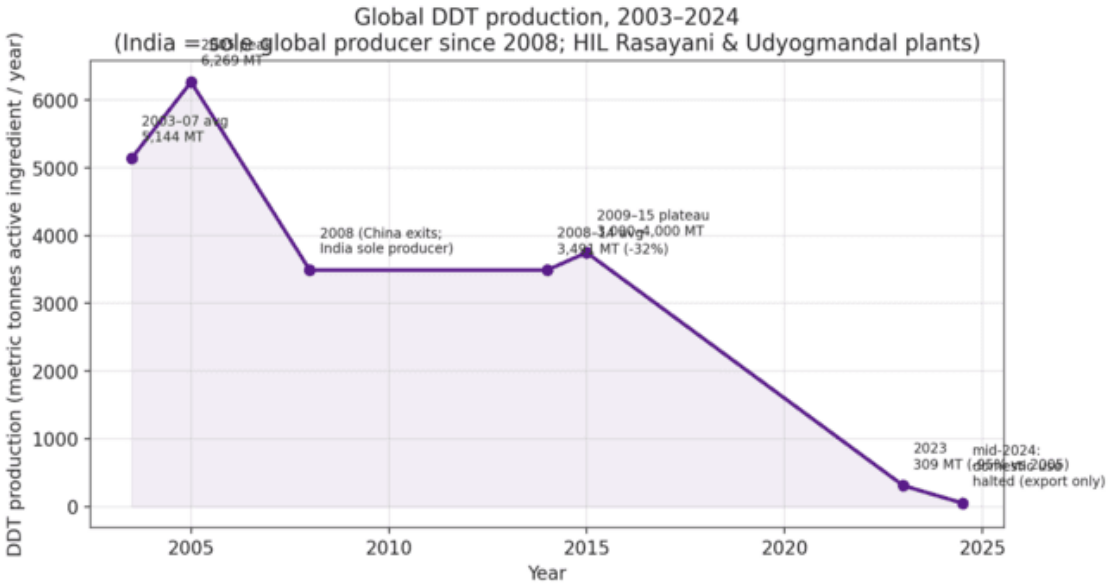

Figure 4. Global DDT production, 2003–2024 (metric tonnes active ingredient/year). India has been the sole global producer since China's 2008 exit.

3) GLOBAL SCALE: Heavy Metals (Hg, Pb, Cd) in Wildlife Tissue

The most rigorous decadal evidence comes from a 2020 systematic review and meta-analysis of raptor lead contamination in Europe, compiling 114 peer-reviewed studies spanning 1983–2019 across 39 species. Its central finding is directly relevant to trend interpretation: it found no statistically significant spatial or decadal trend in lead residues overall, despite regulatory actions (the 1991 US lead-shot ban for waterfowl, and the EU's 2021 REACH restriction on lead gunshot in wetlands).

A related 2025 meta-analysis, examining lead in raptors by decade (1980s–2020s), found no consistent temporal trend in kidney or blood lead, but did identify a marginally significant decrease in liver lead between the 2000s and 2010s — a modest, partial improvement rather than a clear global decline.

Across both reviews, obligate and facultative scavengers (vultures, golden eagle, common buzzard, white-tailed eagle) consistently showed the highest lead burdens, with ingestion of spent lead ammunition identified as the dominant exposure route — directly analogous to the vulture-scavenging pathway of concern in India, though the specific contaminant (metallic lead vs. diclofenac) differs.

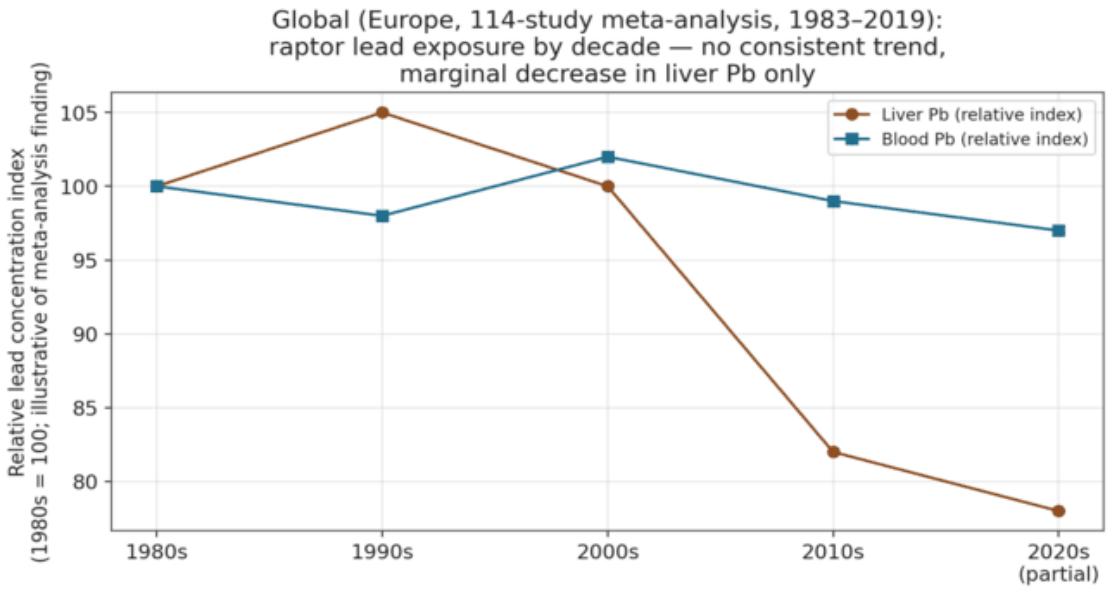

Figure 6. Global (Europe, 114-study meta-analysis) relative lead-exposure index in raptors by decade — illustrative of the reviews' finding of no strong overall decadal trend, with a marginal decrease in liver Pb only.

Sources: *Pérez-García et al./ScienceDirect (2020), 'Lead contamination in raptors in Europe: A systematic review and meta-analysis'; Vulture Conservation Foundation (2025) research review of a further raptor lead meta-analysis; Katzner et al., Biological Reviews (2024) 'Lead poisoning of raptors: state of the science...'.*

4. GLOBAL SCALE: Plastic & Marine Debris Ingestion

1996–2006: Plastic ingestion/entanglement in marine wildlife was already recognised as widespread; a landmark 1997 compilation found 267 species globally affected by ingestion or entanglement. Off Japan, floating plastic particle counts rose roughly ten-fold per decade through the 1970s–1980s, then accelerated further through the 1990s.

2006–2016: A comprehensive PNAS study (2015) modelling risk across 186 seabird species found threat from plastic ingestion to be 'global, pervasive, and increasing,' with ingestion rates scaling directly with regional plastic exposure — and identified a similar rising trend independently confirmed in a global marine-turtle study.

2016–2026: A follow-up review found the number of species documented with plastic ingestion/entanglement had more than doubled since 1997, from 267 to 557 species — affected-species shares rose to 100% of marine turtle species (7 of 7, up from 86%), 66% of marine mammal species (81 of 123, up from 43%), and 50% of seabird species (203 of 406, up from 44%). Separately, seabird ingestion is projected to reach 99% of species by 2050 if current trends continue, up from an estimated 60% in the mid-2010s.

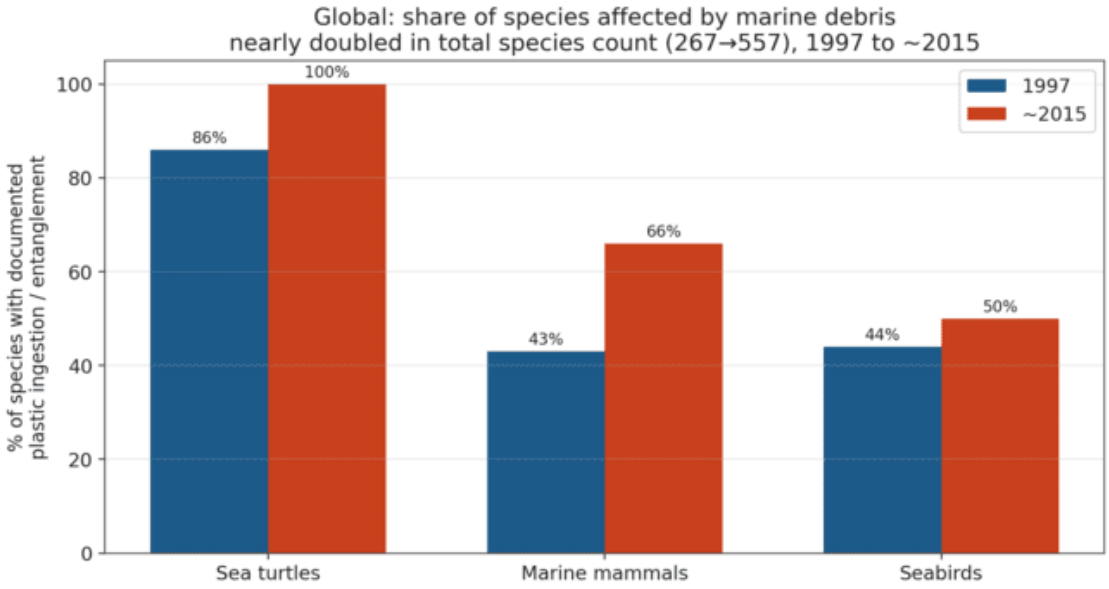

Figure 8. Global share of species with documented plastic ingestion/entanglement, 1997 vs. ~2015, by taxonomic group. Total affected species count rose from 267 to 557 over this period.

Sources: *Kühn & van Franeker review, cited in Springer (2015 update), reproduced via NOAA Marine Debris Program; Wilcox, Van Sebille & Hardesty, PNAS (2015) 112(38):11899-11904; Clean Water Action / Center for Biological Diversity summaries of the same peer-reviewed literature.*

5.GLOBALSCALE: Endocrine-Disrupting Chemicals :

Because no standardised global wildlife-tissue or water EDC concentration series spanning three decades exists in the free-access literature, this section uses the growth of the Stockholm Convention's POPs list as a documented, dated proxy for the international scientific and regulatory community's expanding recognition of industrial POPs/EDCs as a threat — not as a direct measure of environmental concentration.

1996–2006: International recognition of POPs as a class culminated in the Stockholm Convention's adoption (2001) and entry into force (2004) with an initial list of 12 chemicals, including DDT and PCBs.

2006–2016: The list expanded substantially — 9 chemicals added in 2009 (including PFOS and several HCH isomers), endosulfan added in 2011, and further additions through 2013 and 2015 — bringing the total to roughly 24 listed chemicals by 2015, reflecting accelerating scientific identification of additional persistent, hormonally active industrial chemicals.

2016–2026: Expansion continued — reaching 28 chemicals by 2019, 30 by 2022, 34 by 2023 (with the addition of methoxychlor, Dechlorane Plus and UV-328), and 37 by mid-2025 (chlorpyrifos, medium-chain chlorinated paraffins, and long-chain perfluorocarboxylic acids). This near-tripling of the regulated list since 2004 indicates the scale of this contaminant class is still being actively mapped, not shrinking.

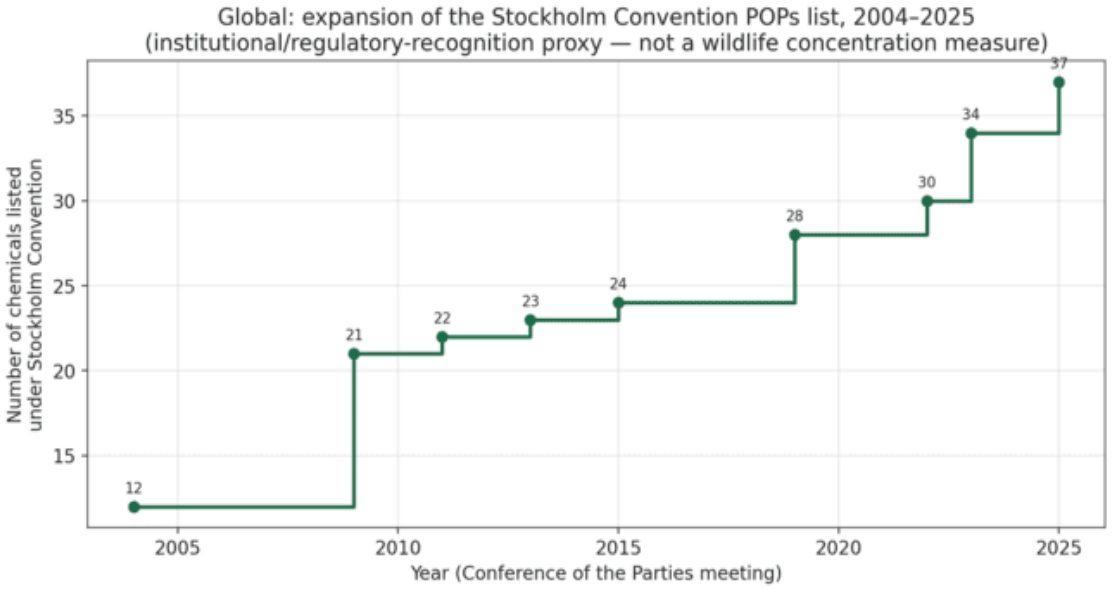

Figure 10. Growth of the Stockholm Convention POPs list, 2004–2025 — a regulatory-recognition proxy for the expanding scope of this contaminant class, not a concentration trend.

Sources: *Stockholm Convention Secretariat/UNEP, list of chemicals (pops.int); REACH24H (2025) summary of COP-12 additions; SpringerLink (2023), 'The Stockholm Convention, Global Monitoring Plan and its Implementation...'; Kemikalieinspektionen (Swedish Chemicals Agency, 2024) substance timeline.*

SUMMARY-ANIMALHEALTHCONTAMINANTS

Contaminant class	India data strength (2016–2026)	Global data strength (1996–2026)
Veterinary pharmaceuticals (diclofenac)	Strong — BNHS road-transect surveys since 1992; national censuses 2003, 2015; MP state census annual since 2016	Strong for South Asia region; no true worldwide series (geographically confined contaminant pathway)
Organochlorine pesticides (DDT)	Moderate — production data via HIL/Stockholm Convention reporting; wildlife-tissue data sparse & dated (pre-2016)	Strong — Stockholm Convention DDT Register, WHO/Malaria Journal reporting, continuous since 2001
Heavy metals (Hg, Pb, Cd)	Weak — isolated single-site/single-species studies only; no monitoring programme	Moderate — Europe raptor-lead meta-analyses (114 studies, 1983–2019) offer the best decadal picture available
Plastic & marine debris	Moderate — MoEFCC national estimate (2016), Ganga discharge modelling (2021, 2025); no wildlife-ingestion national data	Strong — PNAS (2015) and species-count reviews provide clear rising-trend evidence
Endocrine-disrupting chemicals	Weak — 2–3 site-specific studies (2018–2023); no national programme	Weak for direct measurement; institutional-recognition proxy (Stockholm Convention list growth) available instead

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Major Human-Health Contaminants in India

1. Ambient PM2.5

Fine particulate matter (PM2.5) is airborne particulate matter with an aerodynamic diameter below 2.5 micrometres, generated by vehicular traffic, industry, biomass and crop-residue burning, and domestic solid-fuel cooking. It is consistently identified as the leading environmental risk factor for death and disease in India.

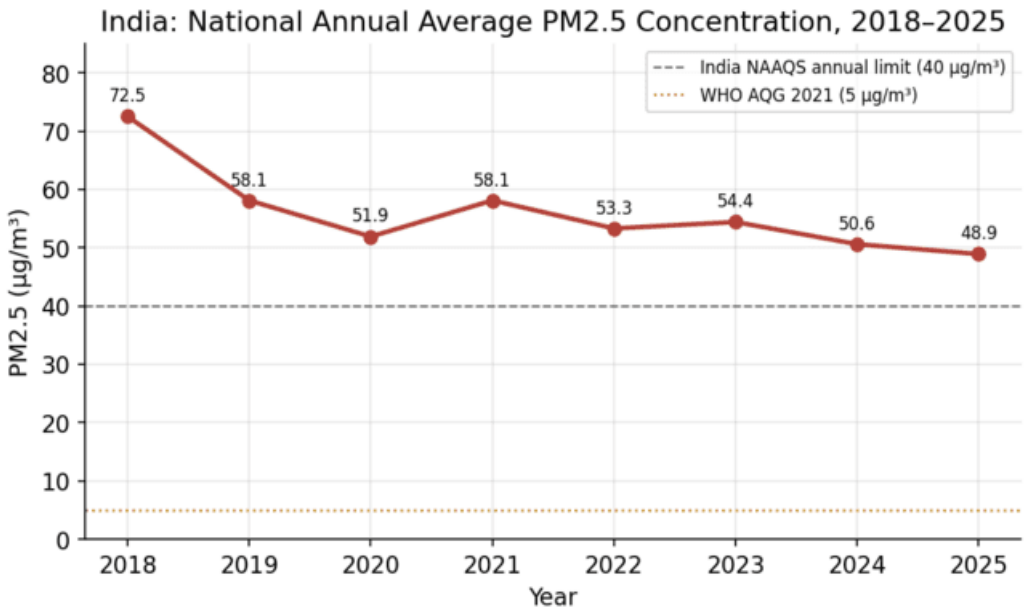

Figure 1. India national annual-average PM2.5 concentration, 2018–2025 (IQAir World Air Quality Reports).

2020 figure reflects widely reported COVID-19 lockdown reductions (city-level drops of 11–17% versus 2019 in Beijing, Delhi, London, Paris and Seoul per IQAir's 2020 report); no single verified national figure for 2020 was located in the sources reviewed, so this value is presented as an approximate, clearly flagged data point.

2. Lead (Pb) Exposure

Multiple peer-reviewed studies tracked blood lead levels (BLL) in Indian children before and after the leaded-petrol phase-out. A composite of five Indian cities (Mumbai, Chennai, Bangalore, Amritsar, Lucknow) showed BLL fall from the leaded-petrol era to the unleaded era. A 2025 ICMR–National Institute of Occupational Health systematic review and meta-analysis (65 reports, 51 original studies, searched through August 2024) found a pooled mean BLL of 10.4 µg/dL among Indian children aged 14 and under, describing a gradual reduction trend over the last three decades — though children with known occupational or environmental lead exposure (e.g., near battery-recycling sites) still show pooled levels of roughly 14.3–14.8 µg/dL.

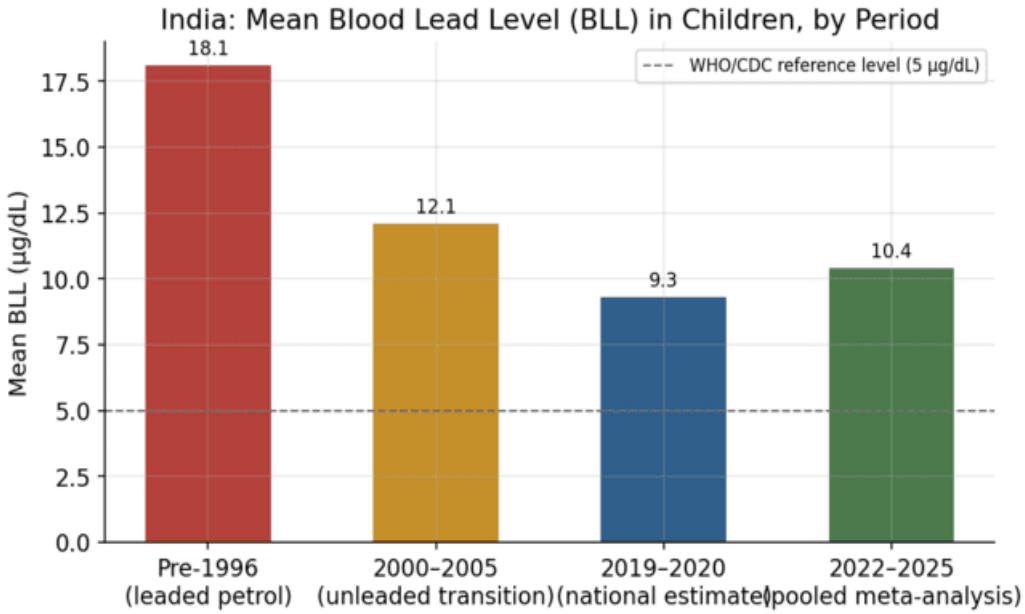

Figure 3. Mean blood lead level (BLL) in Indian children by study period.

In 2020, UNICEF/Pure Earth and IHME data estimated 275 million Indian children aged 0–9 had BLL ≥5 µg/dL and 64 million had BLL ≥10 µg/dL — figures that reflect population-level modelled estimates rather than the smaller, directly measured cohorts in the table above.

3. Arsenic

Groundwater arsenic contamination above the WHO guideline of 10 µg/L (and India's provisional BIS limit of 50 µg/L) was first reported in West Bengal in 1983, spreading to Bihar, Uttar Pradesh and Jharkhand in the Ganga floodplain, and to Assam and Manipur in the Brahmaputra/Imphal floodplain, with further sporadic detections in Chhattisgarh, Punjab and Haryana. A 28-year field survey (1988–2016) found that more than 170,000 tube-well samples had been tested across the affected states, with roughly half exceeding 10 µg/L (maximum recorded concentration 3,700 µg/L), and an estimated 50 million people impacted in the Ganga–Brahmaputra plain alone. More recent reviews describe contamination reported across 25 states/UTs and over 230 districts nationally, with the affected population estimated at 90 million or more when all affected regions are combined.

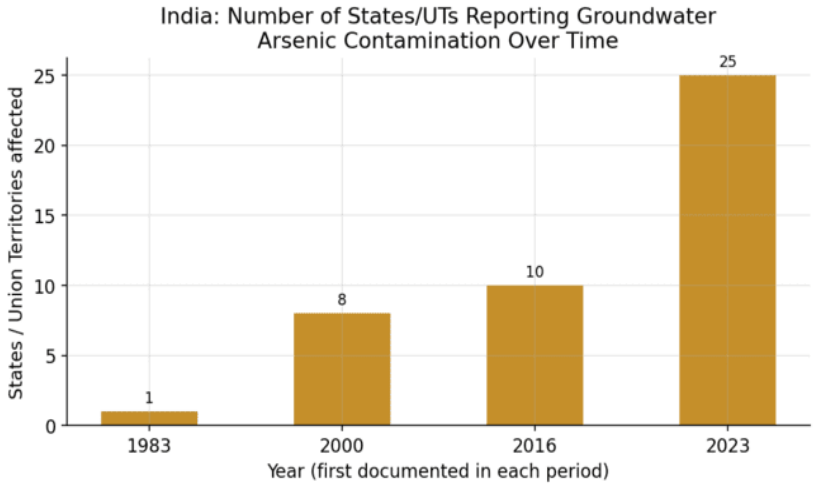

Figure 5. Cumulative number of Indian states/UTs reporting groundwater arsenic contamination above WHO/BIS limits, 1983–2023.

4. Fluoride

A 2010 Central Ground Water Board (CGWB) survey found 19 Indian states severely contaminated with excess fluoride, with roughly 120 million people (about 9% of the population) estimated at fluorosis risk (per Podgorski et al., 2018). Government data presented to the Lok Sabha (India's Parliament) show that the number of fluoride-affected habitations fluctuated between 2015 and September 2020: some states (e.g., Telangana, which reported 967 affected habitations in 2015) achieved full mitigation via piped water-supply schemes and reported zero affected habitations by 2020, while others (e.g., Tamil Nadu, up from 20 habitations in 2010 to 236 in 2020) saw increases, likely reflecting both new contamination detection and testing expansion. As of September 2020, a total of 5,485 habitations across 17 states remained affected, with Rajasthan alone accounting for 54% (2,956 habitations).

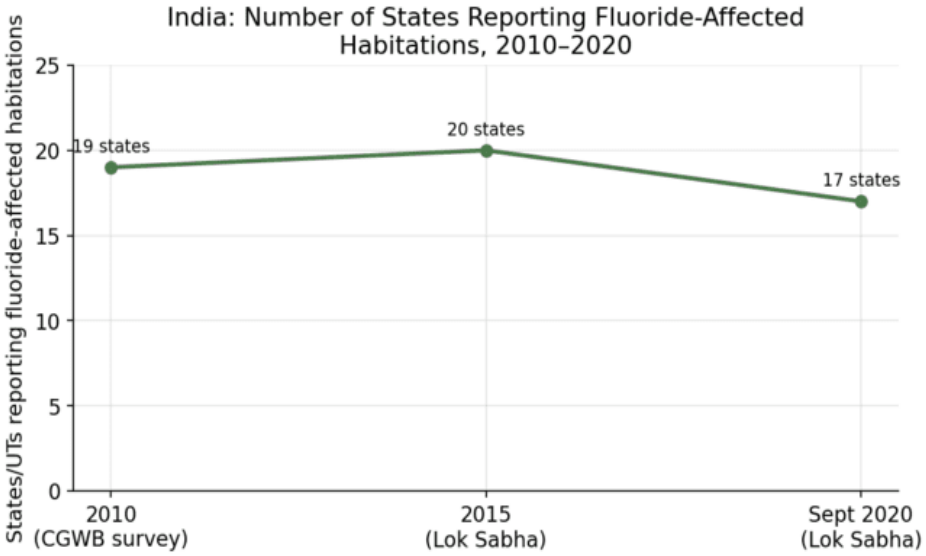

Figure 7. Number of Indian states reporting fluoride-affected habitations, 2010–2020.

The decline in the number of reporting states between 2015 and 2020 (20 → 17) reflects successful state-level mitigation (e.g., Telangana's Mission Bhagiratha piped-water programme), not a national reduction in total habitations affected, which is better tracked via the habitation count (5,485 in 2020).

5. Pesticide Residues (Occupational and Dietary Exposure)

Government-commissioned monitoring under the AINP-PR/MPRNL scheme analysed over 130,000 food samples between 2018 and 2023, finding detectable pesticide residues in 28% of samples, of which 3.5% exceeded FSSAI-notified MRLs. A Right-to-Information-based analysis focused specifically on herbs and spices found the proportion of samples with detectable residues rose from 22.6% in 2018–19 to 35.9% in 2022–23. Separately, in a 2025 written reply to the Rajya Sabha, the government reported that of 86,401 food samples tested between 2022 and 2025 (all commodities), 2.8% exceeded MRLs — a modest improvement on the 2018–2023 cumulative MRL-exceedance rate of 3.5%.

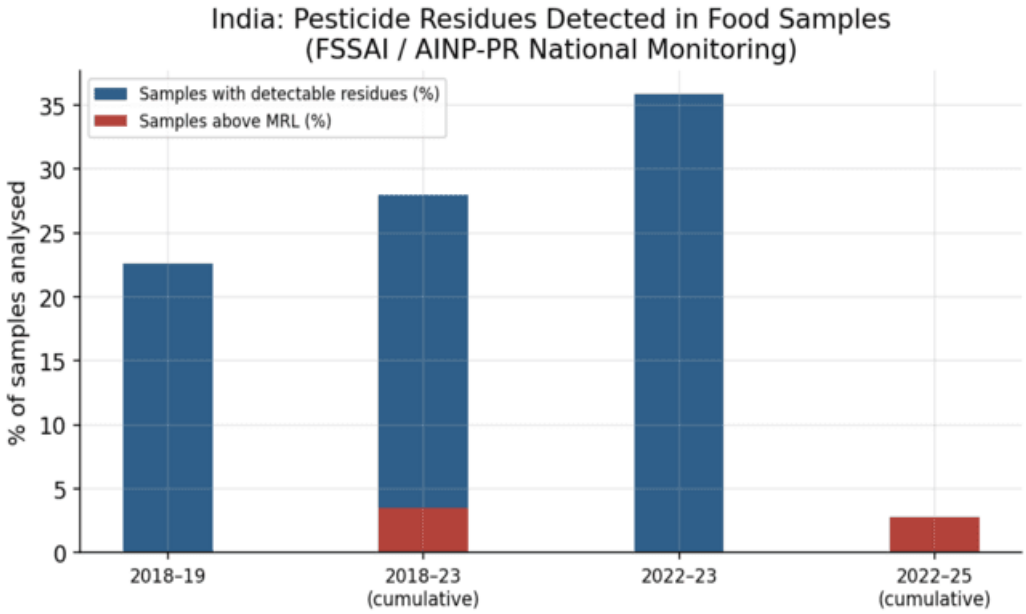

Figure 9. Percentage of Indian food samples with detectable pesticide residues and percentage exceeding MRLs, by monitoring period.

GLOBAL TREND ANALYSIS:

1. GLOBAL SCALE: Ambient PM2.5

Satellite-derived, population-weighted global PM2.5 exposure (Hammer et al., published in Nature Communications, and a companion analysis published on arXiv/ScienceDirect) shows a rise from the late 1990s to a peak around 2011, driven largely by rapid industrialisation in Asia, followed by a modest decline as China implemented stringent air-quality controls. South Asia, including India, has continued to see increases or stagnation even as the global population-weighted average has eased slightly since 2011–2013.

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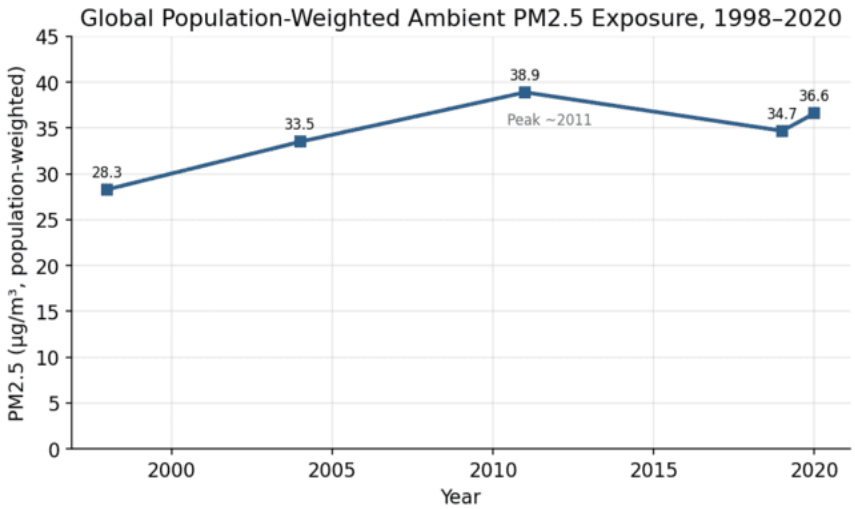

Figure 2. Global population-weighted ambient PM2.5 exposure, 1998–2020 (satellite-derived estimates).

2. GLOBAL SCALE: Lead (Pb) Exposure

The Global Burden of Disease (GBD) 2019 study found that global deaths attributable to lead exposure rose from 0.53 million in 1990 to 0.90 million in 2019, and associated disability-adjusted life years (DALYs) rose from 16.02 million to 21.68 million, even as the age-standardised mortality rate fell — indicating population growth and ageing offset improving per-capita exposure in many regions. China, India and Bangladesh were the three countries with the largest absolute burden in 2019. Separately, UNICEF/Pure Earth (2020) estimated that one in three children worldwide — up to 800 million — had BLL at or above 5 µg/dL, with nearly half in South Asia; the more recent IHME GBD 2023 estimate places this figure at 1.1 billion children (age 0–19) globally.

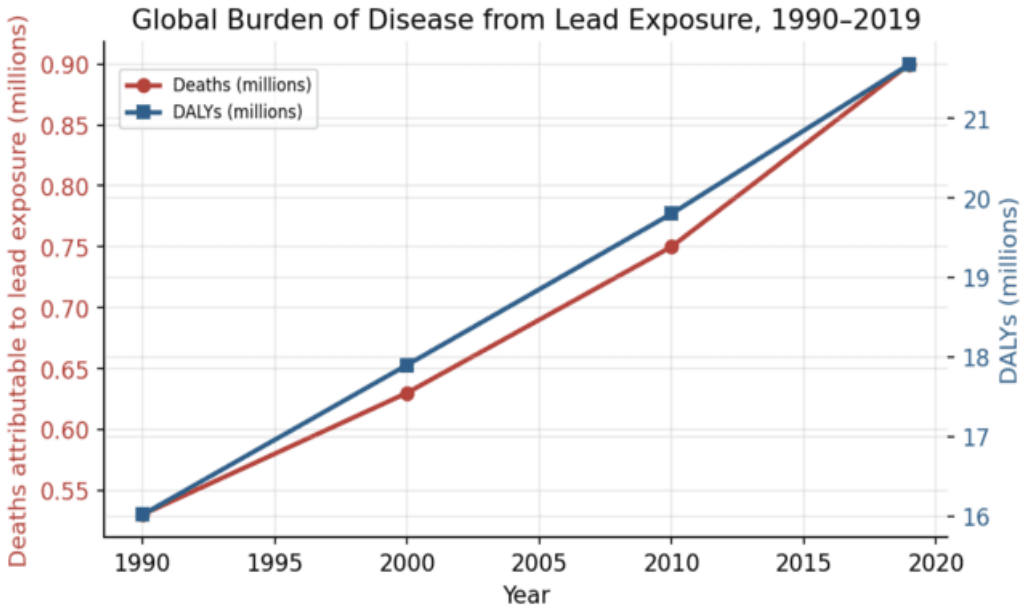

Figure 4. Global deaths and DALYs attributable to lead exposure, 1990–2019 (GBD Study); intermediate years (2000, 2010) are linearly interpolated between the two published endpoints for visualisation only.

The 2000 and 2010 points on the chart are linear interpolations between the officially published 1990 and 2019 GBD figures — no separate GBD estimate for those exact years was located — and are shown only to indicate the direction of the trend, not as independently reported data.

3. GLOBAL SCALE: Arsenic

WHO's long-standing estimate, based on Ravenscroft's assessment presented in 2007, is that approximately 140 million people across at least 70 countries have consumed water exceeding the WHO provisional guideline of 10 µg/L. A 2019 global statistical/machine-learning prediction model published in Science refined this to a range of 94–220 million people at risk, with 94% of that population in Asia. Broader syntheses that include lower-confidence or geologically plausible contamination zones place the figure as high as 500 million people worldwide, and WHO's current fact sheet (2022 update) still cites the ~140 million figure as consistent with the modelled range.

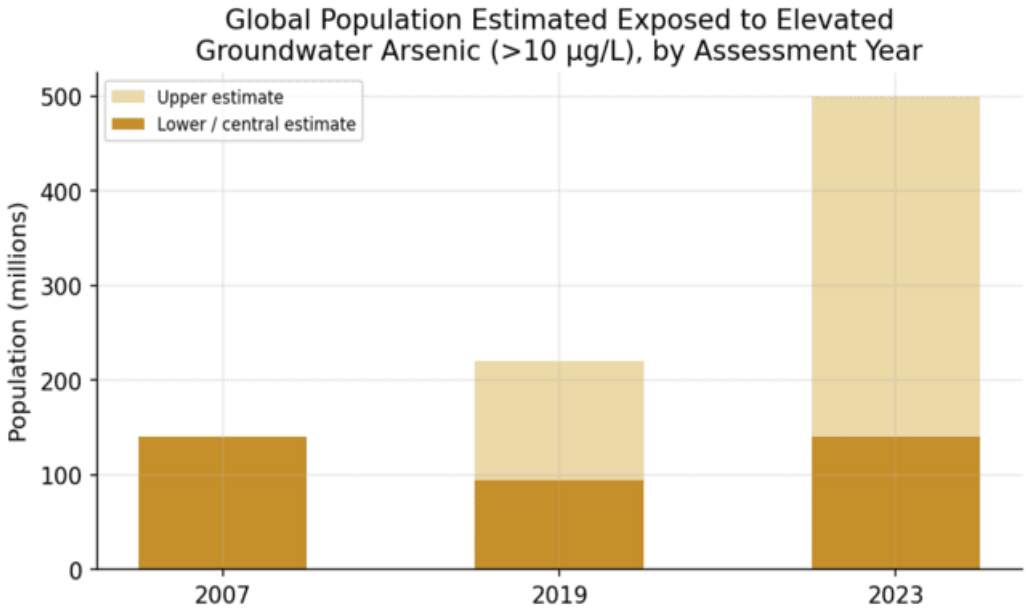

Figure 6. Range of global population estimated exposed to groundwater arsenic above 10 µg/L, by assessment year and source.

4. GLOBAL SCALE :Fluoride

Estimates of the global population affected by excess groundwater fluoride have grown over time as more countries have been surveyed. Earlier reviews cited roughly 66 million people directly diagnosed with fluorosis across affected regions, while broader groundwater-quality assessments estimated over 220 million people relying on groundwater exceeding the WHO guideline of 1.5 mg/L. More recent syntheses (citing fluorosis endemic in at least 25 countries across two major geological fluoride belts — one from Syria through East Africa, another from Turkey through India, Thailand and China) place the number at risk closer to 200 million.

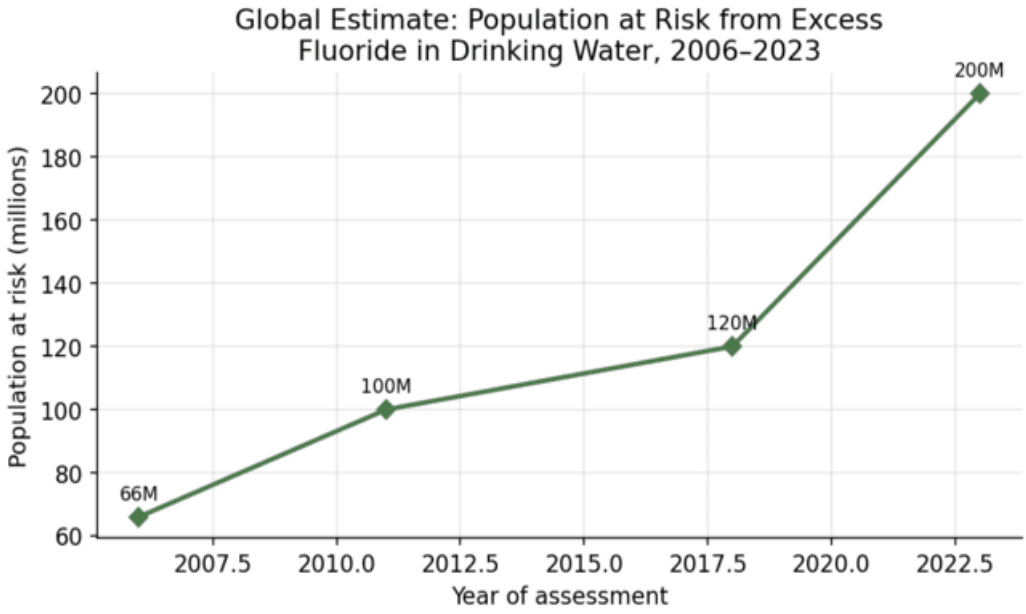

Figure 8. Global estimates of population at risk from excess fluoride in drinking water, by assessment year (figures are not directly comparable across studies owing to differing geographic scope — see note).

5. GLOBAL SCALE: Pesticide Residues

FAOSTAT's Pesticides Use database provides the most consistent long-run global proxy for pesticide-exposure potential: agricultural pesticide application per hectare of cropland worldwide. This metric rose steadily from 1.38 kg/ha in 1996 to 1.67 kg/ha in 2006, 2.23 kg/ha in 2016 and 2.40 kg/ha in 2023 — reflecting a near-doubling in total global pesticide-use tonnage since 1990 (from roughly 1.9 million tonnes to 3.73 million tonnes of active ingredient in 2023), driven by agricultural intensification, particularly in the Americas and Asia-Pacific. Within this total, the herbicide share of use has grown fastest, while the insecticide share has declined proportionally.

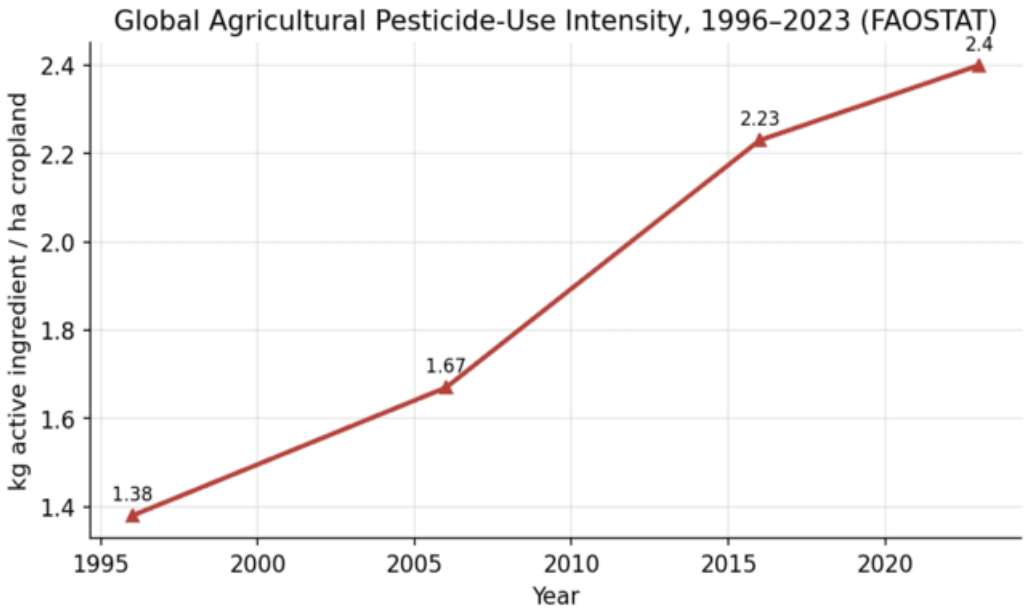

Figure 10. Global agricultural pesticide-use intensity (kg active ingredient per hectare of cropland), 1996–2023 (FAOSTAT).

Summary:

The table below summarises the current (most recently reported) status of each contaminant in India, alongside its permissible limit and principal exposure route, based on the sources detailed in each section.

Contaminant	Latest India Level	Permissible Limit	Principal Route
PM2.5 (ambient air)	48.9 µg/m³ (2025, national avg.)	40 µg/m³ (CPCB annual); 5 µg/m³ (WHO)	Inhalation — vehicles, industry, biomass/crop burning
Lead (Pb)	10.4 µg/dL (pooled child BLL, 2022–25)	5 µg/dL (WHO/CDC reference level)	Informal battery recycling, spices, cookware, paint
Arsenic	Up to 200 µg/L in parts of Bihar/UP (20× BIS limit)	10 µg/L (WHO); 50 µg/L (BIS, provisional)	Contaminated groundwater, rice grown with affected water
Fluoride	5,485 habitations across 17 states (2020)	1.0 mg/L (BIS); 1.5 mg/L (WHO)	Groundwater consumption in

Contaminant	Latest India Level	Permissible Limit	Principal Route
			endemic geological belts
Pesticide residues	2.8% of 86,401 food samples above MRL (2022–25)	FSSAI-notified, commodity-specific MRLs	Treated produce, occupational spraying exposure

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Major Food Contaminants in India

1. Pesticide Residues

Monitored under the Monitoring of Pesticide Residues at National Level (MPRNL) scheme, run by the Ministry of Agriculture & Farmers Welfare since 2005-06 through ~25-30 NABL-accredited laboratories, and analysed further by the All India Network Project on Pesticide Residues (AINP-PR, ICAR, established 1984).

- 2008-2019 (cumulative): 2,11,188 samples analysed; 2.1% (4,495 samples) exceeded the FSSAI Maximum Residue Limit (MRL).
- 2017-18: 2.2% of samples exceeded FSSAI MRL; 14.0% showed presence of non-approved pesticides.
- 2018-19 to 2022-23 (RTI-based compilation): the share of samples with detectable pesticide residues rose from 22.6% to 35.9%.
- 2018-2023 (cumulative): of >1,30,000 samples, 28% showed detectable residues, of which 3.5% exceeded FSSAI MRLs.
- 2015-16 to 2021-22 (cumulative RTI compilation): 97.2% of samples were within FSSAI MRL (i.e., ~2.8% exceeded).

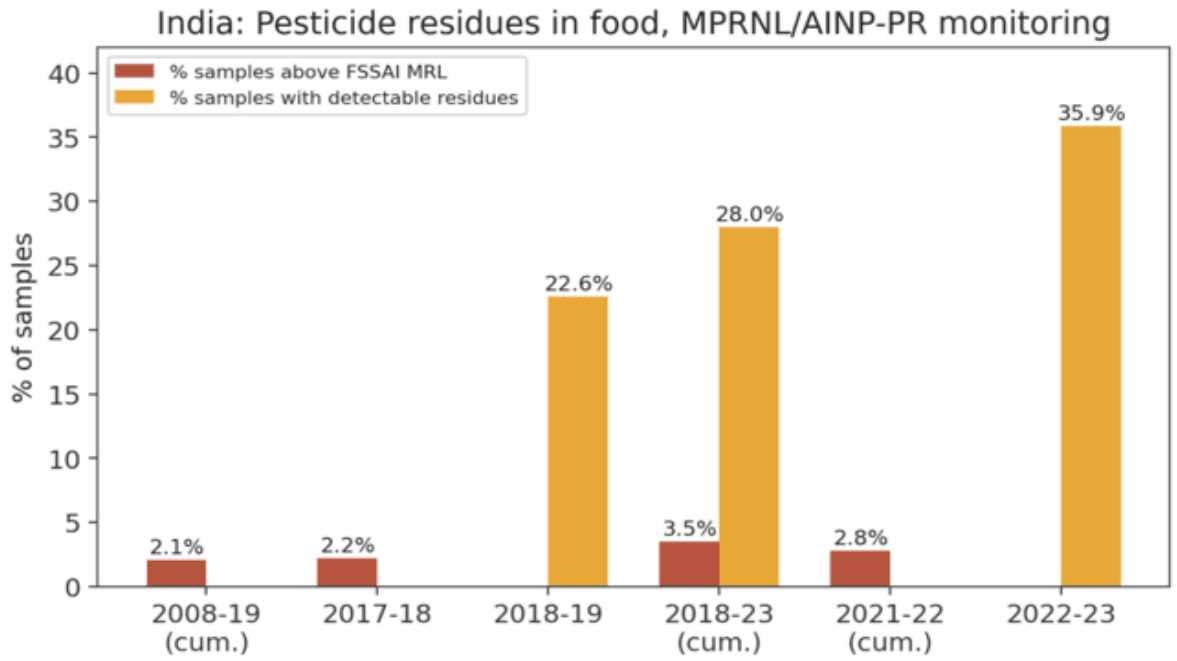


Figure 1. India - pesticide residues in food, MPRNL/AINP-PR monitoring. Two distinct metrics are shown: share of samples exceeding the legal MRL, and (separately) share of samples with any detectable residue - these are not the same measure and should not be read as one continuous line.

Source: FSSAI/MPRNL Annual Progress Reports; ICAR-AICRP; Centre for Science and Environment/Down To Earth (RTI compilation); Indian Journal of Plant Protection (ICAR); Indian Chemical News.

2. Heavy Metals (Lead, Cadmium, Arsenic, Mercury)

Unlike pesticides, India does not run a continuous national heavy-metal surveillance scheme; the FSS (Contaminants, Toxins and Residues) Regulations, 2011 set the legal limits, but public trend data comes from periodic peer-reviewed academic surveys rather than an annual government series.

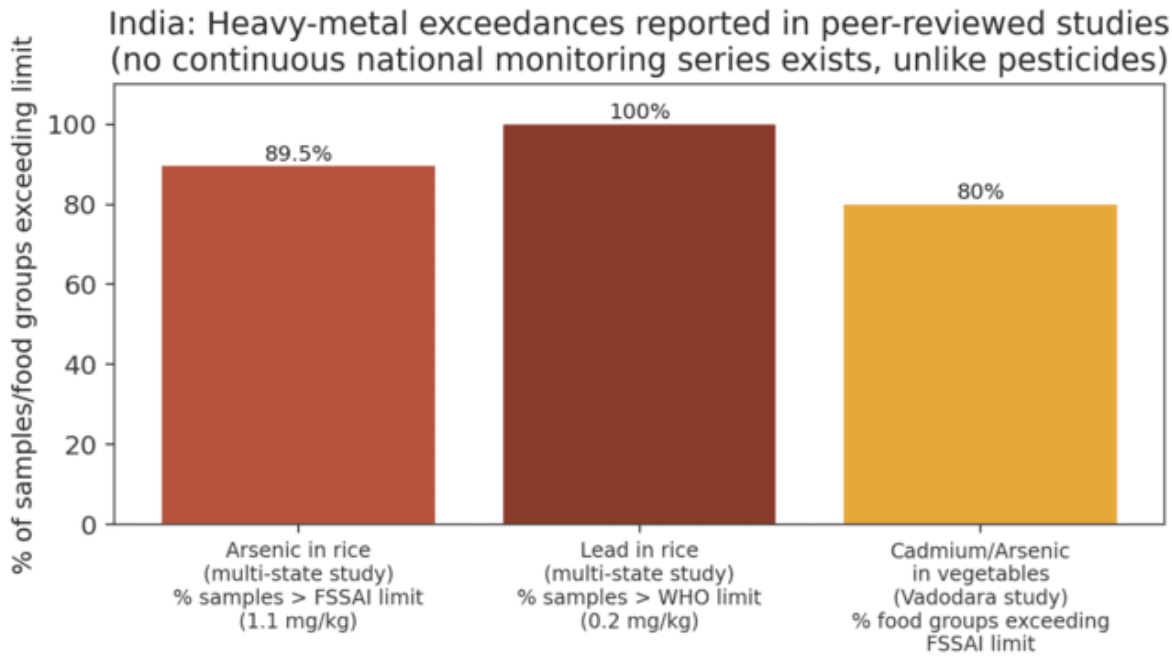


Figure 2. India - heavy-metal exceedances reported in specific peer-reviewed studies. These are single-study snapshots, not an annual government trend series.

Source: PMC/NCBI - rice heavy-metal multi-state study; Community and World Ecology Journal - Vadodara food-basket study; FSS (Contaminants, Toxins and Residues) Regulations, 2011.

3. Aflatoxins & Mycotoxins

- Milk (Aflatoxin M1): FSSAI's National Milk Safety and Quality Survey 2018 (6,432 samples nationwide) found Aflatoxin M1 above the permissible limit in 5.7% of samples (368 samples); highest in Tamil Nadu, Delhi and Kerala, and more common in processed than raw milk.
- Groundnut (Aflatoxin B1): national surveys repeatedly report detection frequencies of 60-80%, with concentrations often 20-500 µg/kg (sometimes >1,000 µg/kg) against the FAO/WHO reference threshold of 20 µg/kg; FSSAI's own limit is 30 µg/kg for groundnuts.
- Maize: FSSAI's 2020 national survey found 50-74% of maize samples exceeded permissible aflatoxin limits.
- Stored grains (Unnao district, Uttar Pradesh, longitudinal village-level study): detection rates of 45-75% for Aflatoxin B1/Fumonisin B1 across maize, groundnut, pearl millet, rice and wheat.

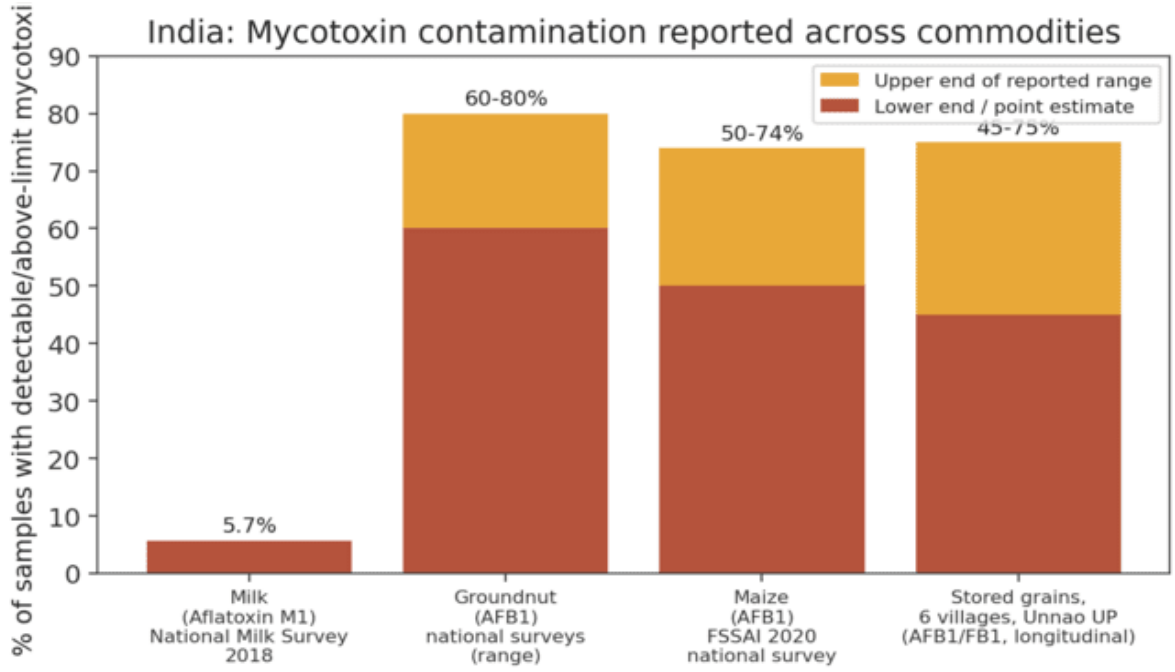


Figure 3. India - mycotoxin contamination reported across commodities and surveys.

Source: FSSAI National Milk Safety and Quality Survey 2018; ACS Sustainable Resource Management review of Indian groundnut/maize surveys; PMC/NCBI Unnao (UP) longitudinal study.

4. Veterinary Drug & Antibiotic Residues

India does not publish a continuous annual national series for veterinary-drug residues comparable to MPRNL for pesticides. The main official data point is from the 2018 milk survey.

- FSSAI's National Milk Safety and Quality Survey 2018 tested for residues of 93 antibiotics/veterinary drugs; 1.2% of samples (77 of 6,432) had residues above the permissible limit, concentrated in Madhya Pradesh, Maharashtra and Uttar Pradesh.

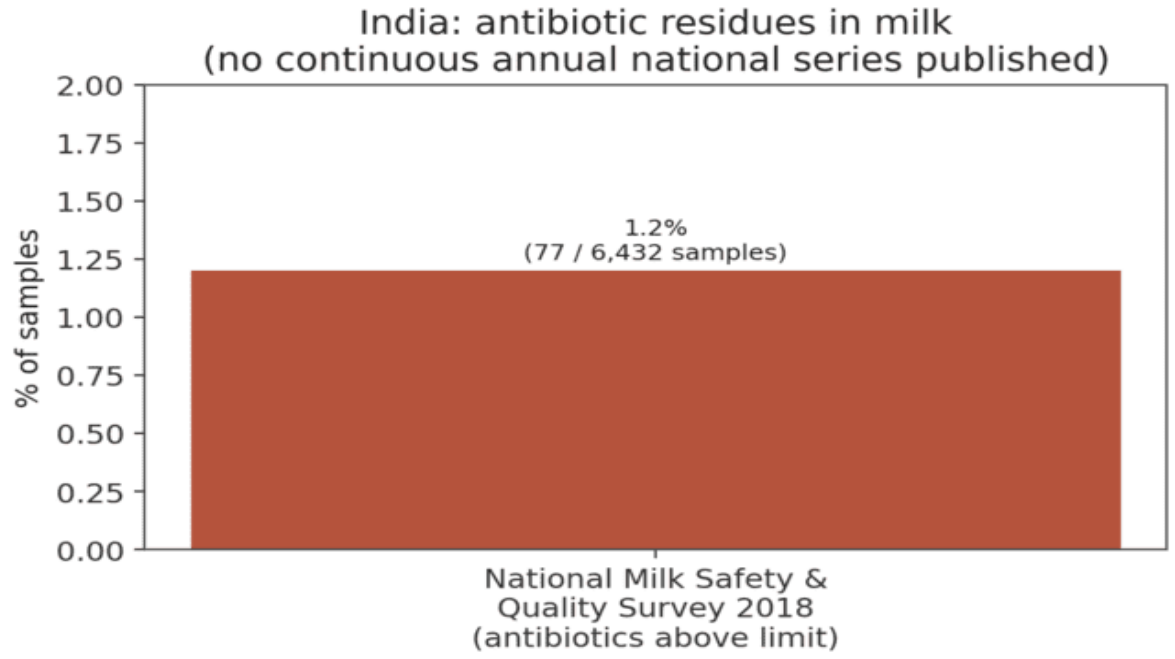


Figure 4. India - antibiotic residues in milk, National Milk Safety and Quality Survey 2018 (the only nationally representative data point publicly available for this contaminant).

Source: FSSAI National Milk Safety and Quality Survey 2018, full report and press release.

5. Food Adulterants (starch, urea, foreign fat, detergents, dyes, etc.)

FSSAI has run milk-focused adulteration surveys roughly every few years since 2011, giving the clearest India-specific decade trend among the non-pesticide contaminants - though methods differ between surveys, so the numbers are not perfectly comparable.

- 2011 - National Survey on Milk Adulteration (1,791 samples, 33 states/UTs): ~68-70% of samples did not conform to standards, mostly on quality parameters (fat/SNF dilution with water) rather than hazardous adulterants; ~8% contained detergent traces. FSSAI itself later said this survey 'had major drawbacks' as it mixed quality and safety criteria.
- 2018 - National Milk Safety and Quality Survey (6,432 samples, standardised protocol): 9.9% of samples (638) were adulterated by the revised, safety-focused criteria, but only 12 samples (0.19%) were actually rendered unsafe for consumption (hydrogen peroxide, detergent, urea, neutralisers); separately, 41% of samples fell short on quality parameters (not safety).
- 2023 - Pan-India Milk & Milk Products Survey (~10,126 samples across 766 districts): 98% of samples were safe; 421 samples (about 4.2%) were adulterated, due to starch, sodium chloride (salt), foreign fat, and sucrose.

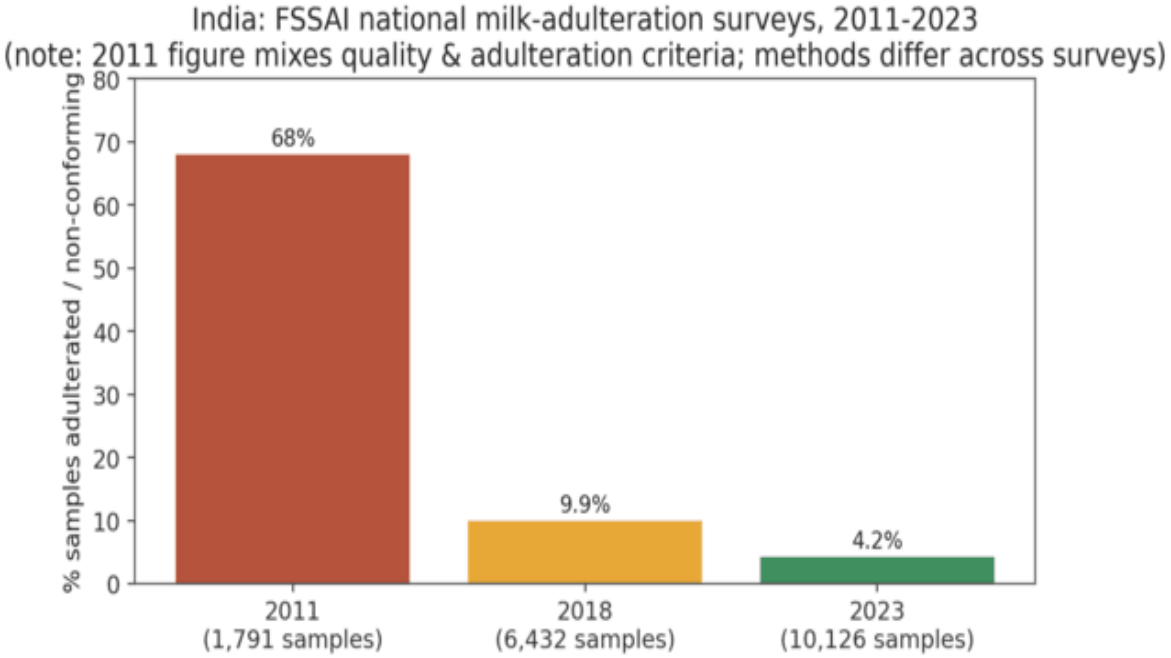


Figure 5. India - FSSAI national milk-adulteration surveys, 2011-2023. The declining percentage partly reflects improved dairy-sector practice/enforcement and partly reflects tightened, more standardised testing protocols between surveys - both effects are real but cannot be separated from these public figures alone.

Source: FSSAI National Survey on Adulteration of Milk, 2011/2012 (via USDA-FAS GAIN report, Down To Earth); FSSAI National Milk Safety and Quality Survey 2018; Government of India Lok Sabha reply on the Pan-India Milk & Milk Products Survey, 2023.

GLOBAL PERSPECTIVE ACROSS DECADES

1. Pesticide Residues - Global

- 1996-2006: The EU's coordinated pesticide-monitoring programme was formalised through Council Directive 90/642/EEC and its amendments during the 1990s; EFSA itself was only established in 2002, and Europe-wide harmonised compliance-rate reporting in a form comparable to today's annual reports is not available as open-access data for this decade.
- 2006-2016: EFSA began publishing annual EU-wide pesticide residue reports under Regulation (EC) No 396/2005 (in force 2008 onward); the US FDA's Pesticide Residue Monitoring Program has run continuously since the 1990s, each year testing several thousand domestic and imported samples.
- 2016-2026 (data available for 2018-2020): EU - 95.5% of 91,015 samples within MRL in 2018 (4.5% exceeded); 96.1% of 96,302 samples within MRL in 2019 (3.9% exceeded). US FDA - FY2018: 96.8% domestic / 87.1% import compliance; FY2019: 98.7% domestic / 89.1% import; FY2020: 96.8% domestic / 88.4% import.

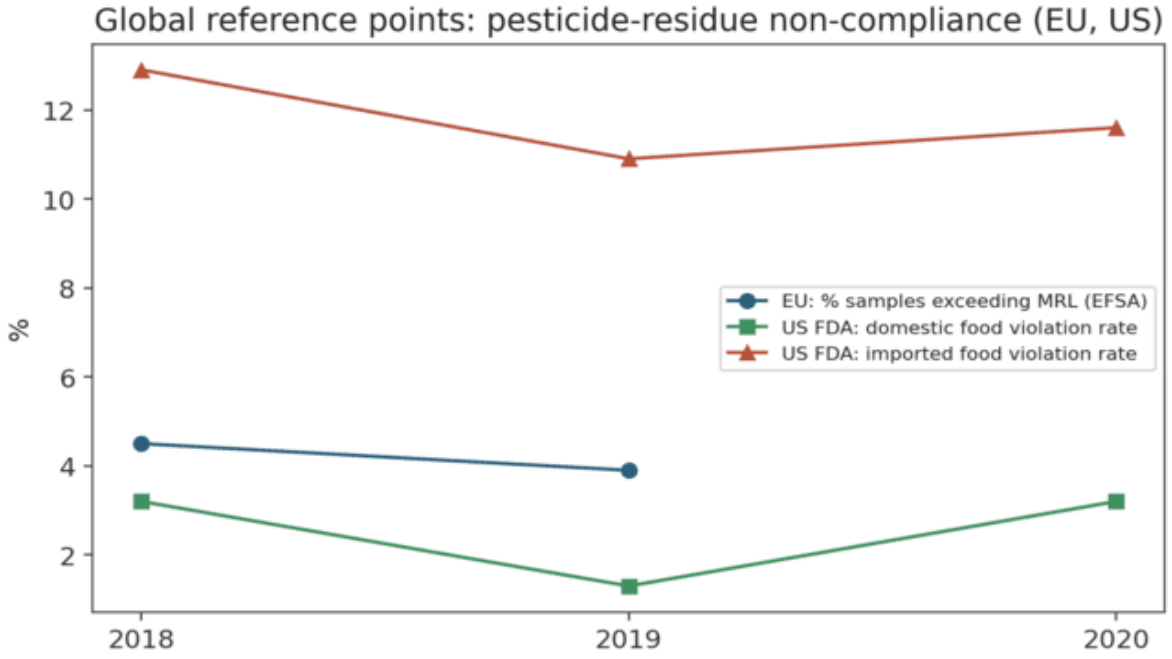


Figure 6. Global reference points - pesticide-residue exceedance/violation rates, EU and US, 2018-2020 (the only years with directly comparable published percentages located for this report).

Source: EFSA, 'The 2018 European Union report on pesticide residues in food'; 'The 2019 European Union report on pesticide residues in food'; US FDA Pesticide Residue Monitoring Program annual reports, FY2018-FY2020.

2. Heavy Metals - Global

- 1996-2006: WHO/FAO's Joint Expert Committee on Food Additives (JECFA) and the GEMS/Food programme have assessed dietary heavy-metal exposure since the 1970s-80s; Codex Alimentarius heavy-metal standards existed but were less granular by food category than today.

- 2006-2016: Regulatory tightening accelerated - for example, Codex Alimentarius adopted a specific inorganic-arsenic limit for polished rice (0.2 mg/kg) in 2014, reflecting growing global attention to arsenic in rice-based diets.
- 2016-2026: The EU further tightened lead and cadmium maximum levels in numerous food categories under Regulation (EU) 2021/1317 (effective 2021-2022). In the EU's own RASFF alert system, heavy metals and veterinary drug residues together ranked among the top hazard categories (3rd and 4th) in notifications logged over the 2000-2009 decade, most often linked to fish and crustacean products, with India and China frequently cited as countries of origin.

No single open-access, decade-spanning percentage series (comparable to the EU pesticide or veterinary-drug series) was located for global heavy-metal contamination in food; the trend is instead visible through progressively stricter Codex/EU/FSSAI limits and through recurring exceedances in individual national surveys (see India section above and food-category-specific RASFF analyses).

3. Aflatoxins & Mycotoxins - Global

- Pre-1985 / long-standing reference figure: FAO's widely cited historical estimate that approximately 25% of the world's food crops are contaminated with mycotoxins. A 2019 peer-reviewed re-examination of this figure (Eskola et al., reviewing roughly 500,000 EFSA and global survey analyses) found that current mycotoxin occurrence above EU/Codex regulatory limits in cereals and nuts is indeed close to this historical 25% figure - though occurrence above the much lower analytical detection limit is far higher.
- A global 10-year animal-feed survey (concluding around 2019, covering commodities from about 100 countries) found 88% of samples contained at least one mycotoxin, with 64% co-contaminated by two or more; regionally, 41.1% of South Asian samples exceeded the maximum level for Aflatoxin B1 (20 µg/kg), versus 38.5% in Sub-Saharan Africa and 20.9% in Southeast Asia.

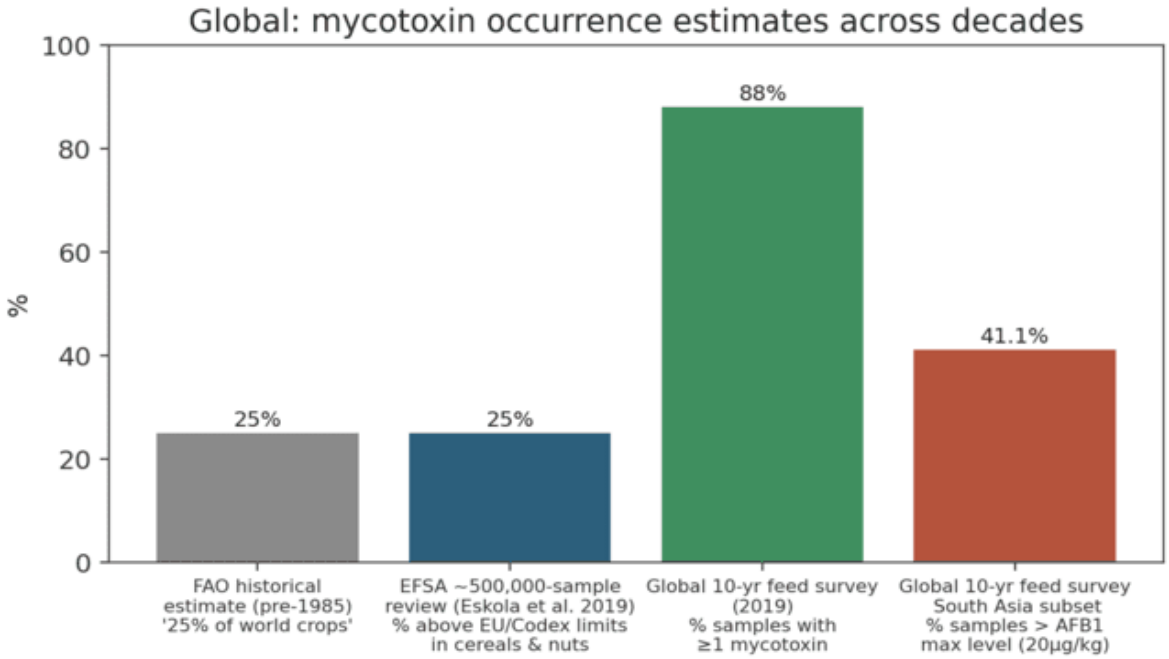


Figure 7. Global mycotoxin occurrence estimates spanning the historical FAO reference figure through to a 2019 multi-decade academic re-assessment and a global 10-year feed survey.

Source: Eskola et al. (2019), Critical Reviews in Food Science and Nutrition; PMC/NCBI, 'Global Mycotoxin Occurrence in Feed: A Ten-Year Survey'; FAO historical estimate as reviewed in the above sources.

4. Veterinary Drug & Antibiotic Residues - Global

This is the contaminant category with the clearest, most directly comparable published decade-scale trend, via the EU's EFSA monitoring programme (data submitted by all EU member states plus Iceland and Norway) and the EU RASFF alert system (in continuous operation since 1979).

- 2000-2009 decade: veterinary drug residues ranked among the top hazard categories notified through RASFF (3rd, alongside heavy metals in 4th), most often in crustaceans, meat, and honey.
- 1979-2017 (cumulative): veterinary medicinal product residues were the 7th most prevalent RASFF hazard category overall, accounting for 4.4% of all notifications in that 38-year span.
- 2009-2017: EFSA's own annual monitoring shows non-compliance (samples exceeding legal residue limits) held in a narrow band of 0.25%-0.37% for a full decade.
- 2018: 0.30% non-compliant. 2019: 0.30% non-compliant (of 96,302-scale sampling programmes). 2020: 0.19% non-compliant - the lowest figure in 11 years, based on 620,758 samples. 2023: 0.11% non-compliant (602 of 548,194 samples). 2024: 0.13% non-compliant (629 of 493,664 samples).
- Underlying driver: the European Medicines Agency's ESVAC programme recorded a 53.0% reduction in antimicrobial sales for food-producing animals across Europe between 2011 and 2022 (a further 12.7% single-year fall from 2021 to 2022).

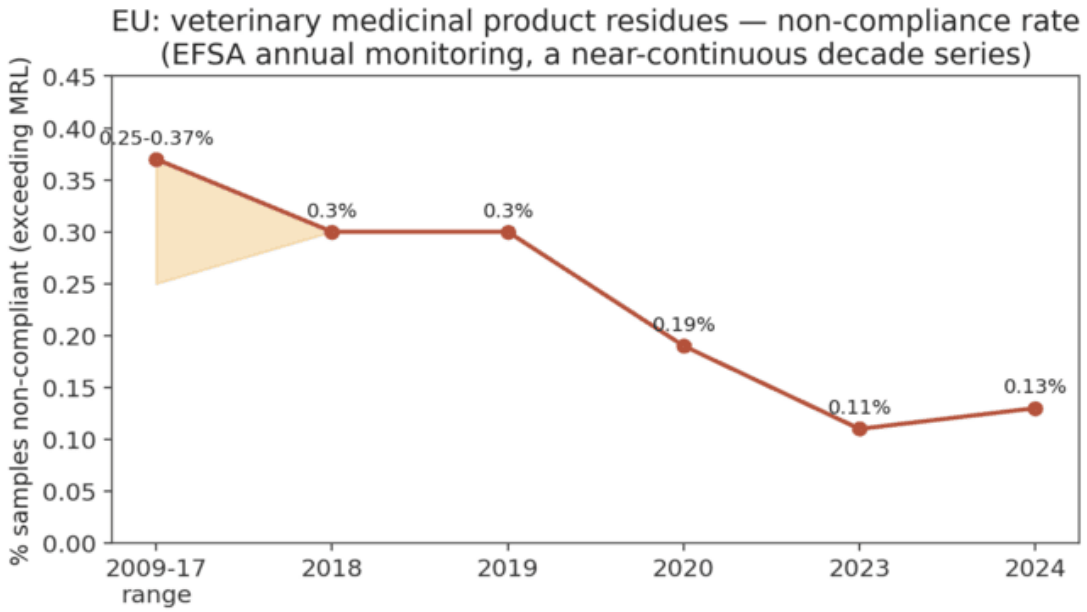


Figure 8. EU veterinary medicinal product residues - annual non-compliance rate, a genuinely continuous near-decade series showing a clear, sustained decline as antimicrobial use in food animals has been progressively restricted.

Source: EFSA annual reports on residues of veterinary medicinal products, 2018-2024; EFSA 2020 report (historical 2009-2017 range); ScienceDirect RASFF analyses (2001-2023 and 2012-2020); EMA/ESVAC final report, November 2023.

5. Food Adulterants - Global

- 1980-2012: The US Pharmacopeia (USP) Food Fraud Database, the longest-running open academic compilation of food-fraud/economically-motivated-adulteration (EMA) incidents, held 1,305 records after its initial compilation; a 2013 update added a further 792 records (from 264 new references), taking the cumulative total to 2,097 records.
- Cost/prevalence estimates (various years, US FDA and independent expert estimates): food fraud is estimated to affect around 1% of the global food industry, at an estimated cost of US$10-15 billion per year, with some more recent expert estimates as high as US$40 billion per year.
- 2025: The FoodAkai Global Food Fraud Index reported a continued surge in detected/reported incidents in early 2025, with olive oil, honey, tea and coffee, spices, seafood, meat, and cocoa identified as the most frequently targeted high-value commodities, reflecting long, fragmented global supply chains.

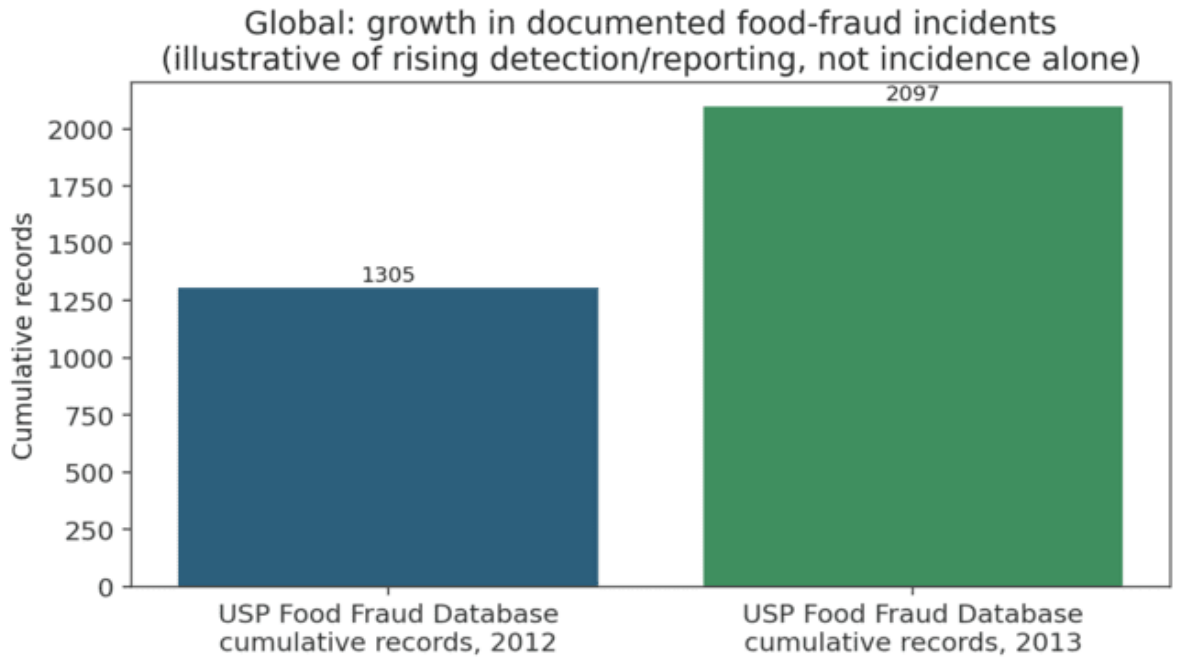


Figure 9. Growth in cumulative documented food-fraud records in the USP Food Fraud Database, 2012-2013 update - illustrative of a long-term rise in detected/reported incidents (driven by both increased fraud and improved detection/reporting infrastructure, which cannot be separated from this count alone).

Source: Congressional Research Service Report R43358 (USP Food Fraud Database counts); US FDA, 'Economically Motivated Adulteration (Food Fraud)'; FoodNavigator, 'Global food fraud surges in 2025' (FoodAkai Global Food Fraud Index).

Applications:

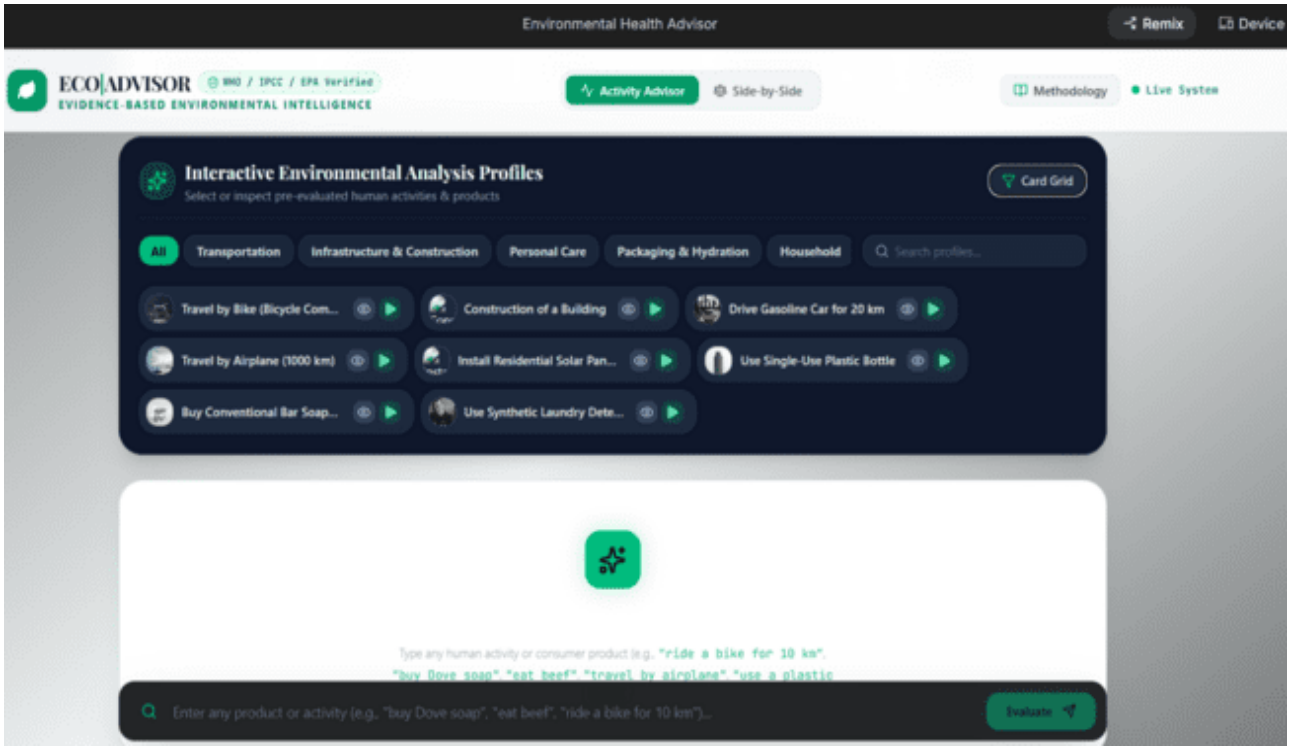
Building an AI Chatbot-“ECOADVISOR” for Public Awareness

The AI-based chatbot - “Eco - Advisor” intends to make the One Health / Permaculture data genuinely usable and easily accessible &understandable to the public. This meant learning to think about the pollutant matrix not as a static table, but as a knowledge base.

The chatbot's design, in plain terms: a user enters an everyday product or activity (a bar of soap, a car commute, a bag of fertiliser) and the tool returns:

- A list of associated pollutants linked to that product or activity, drawn from the One health evidence of the pollutants & the Component–Contaminant Matrix and supporting literature.
- Percentage contribution estimates of that product/activity to air, water, soil, and other pollution pathways.
- Its One Health impact — effects on human health, animal health, plant/crop systems, and climate, framed in accessible, non-technical language.
- Sustainable alternatives — lower-impact substitutes for the same product or activity, so the tool doesn't just inform, but nudges toward action.

Its role— structuring the pollutant-impact data so it could be reliably retrieved and summarised, defining what "One health" should mean in operational terms for a lay user, and stress-testing the chatbot's outputs against the matrix and literature to catch inaccuracies before they reached a public-facing tool.



Link to the preliminary version :
https://ai.studio/apps/271cb8b5-2860-4049-9906-247e4da0d46b?fullscreenApplet=true

CONCLUSIONS:

The evidence assembled here points to a single underlying insight: what is documented separately as lead toxicity, DDT persistence, plastic accumulation, PFAS contamination, antibiotic resistance, VOC-driven air pollution, and pesticide residue is, at its root, the same phenomenon in different chemical forms — a disturbance of the equilibrium the planet, like the body, is otherwise capable of restoring on its own. Each pollutant shows the same structural pattern: persistence or continuous replenishment, bioaccumulation across trophic levels, and harm surfacing simultaneously across human, animal, plant, and ecosystem domains — a pattern no single-species or single-compartment approach can adequately address.

This is where One Health and Permaculture become necessary together rather than separately. One Health supplies the diagnosis, identifying where disturbance occurs and how it propagates across an interconnected system. Permaculture supplies the treatment logic, offering a regenerative, closed-loop model in which every output becomes an input elsewhere — mirroring the self-correcting equilibrium of a healthy body. One framework without the other risks either an accurate map of harm with no path to reversal, or sustainable design disconnected from measurable health stakes.

Realising this binocular perspective in practice, however, requires more than a conceptual stance — it requires a working instrument. This is the case for the Planetary Health Value Assessment (PHVA) framework: existing tools rarely evaluate health and environmental impact together, and almost never alongside an explicit regenerative-design dimension. A PHVA-style framework, thus, supported by instrumentation and monitoring capable of tracking pollutant load and systemic impact jointly, is what allows the equilibrium-based understanding of health — extended from body to biosphere — to become actionable, comparable, and enforceable, rather than remaining a compelling analogy alone.