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Invisible Threats, Shared Fates: Strengthening One Health Defenses against Environmental Toxins

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Abstract

Rationale: Environmental toxins constitute a pervasive and escalating challenge for public health and ecosystems worldwide. They arise from diverse sources, *i.e*, heavy metals, pesticides, pharmaceuticals, persistent organic pollutants (POPs), plastics, and emerging contaminants such as PFAS. While their toxicological mechanisms are well studied, their cumulative effects on human health, microbial systems, and ecological processes remain poorly integrated into mainstream policy and practice. The resulting "toxin conundrum" reflects the gap between scientific evidence and effective interventions, with vulnerable populations disproportionately affected. **Objective:** This paper seeks to critically examine the complex relationships between environmental toxins, human health, and ecosystems, viewed through the unique lens of a bacteriologist. Specifically, it assesses mechanisms of toxicity, human and ecological impacts, microbiome-mediated interactions, and the adequacy of current policy responses, while advancing practical pathways for prevention, remediation, and integrated governance. **Methods**: A narrative synthesis of peer-reviewed research, international policy reports, and global case studies (e.g., Minamata disease, DDT-induced avian declines, PFAS contamination, and antimicrobial resistance linked to pharmaceutical effluents) was conducted. Evidence was drawn from PubMed, PsycINFO, AJOL, Scopus, and complemented by grey literature and international treaties. Mechanistic, epidemiological, and ecological findings were systematically linked with policy frameworks to build a holistic account of the toxin-health-ecosystem nexus. **Results:** Findings demonstrate that toxins act through convergent mechanisms of oxidative stress, DNA damage, endocrine disruption, immune suppression, and microbiome perturbation. Health outcomes include neurodevelopmental disorders, cancers, infertility, and antimicrobial resistance. Ecosystem effects include biodiversity loss, bioaccumulation, and disruption of ecosystem services. Current policies show partial successes (e.g., Stockholm and Minamata Conventions) but remain fragmented, often addressing single chemicals rather than mixtures, with limited integration of microbial endpoints or equity concerns. Evidence supports upstream prevention, class-based regulation, enhanced surveillance (e.g., wastewater-based epidemiology), and validated remediation strategies as feasible interventions. **Conclusions**: The toxin conundrum highlights the interconnectedness of human and ecosystem health. Addressing it requires a shift from fragmented, reactive measures to integrated, preventive strategies that combine scientific innovation, policy reform, and community engagement. **Recommendations**: Adopt class-based restrictions, strengthen extended producer responsibility, scale up wastewater and exposome surveillance, integrate microbial endpoints into regulation, and prioritize equity for vulnerable populations. **Significant Health Statement:** Environmental toxins pose a silent but profound threat to global health. By bridging microbiology, toxicology, and policy, this work underscores that decisive, integrated action can reduce disease burdens, protect biodiversity, and promote health equity.

Keywords

Environmental Toxins, Human Health, Ecosystem Health, Microbiome, Antimicrobial Resistance, Policy, Remediation, One Health

1. Introduction

Environmental toxins such as fine-particulate air pollution, lead, "forever chemicals" (PFAS), and plastic fragments touch nearly every community on earth. Large syntheses already show the scale: pollution remains linked to roughly nine million premature deaths annually, with the heaviest burdens in low- and middle-income countries [1]-[10]. New exposure fronts keep emerging. Pharmaceuticals are now measurable in most of the world's rivers, signalling constant, low-dose ecosystem exposure [11]-[13]. Microplastics have been detected in human tissues, including surgically excised arterial plaques, where their presence correlated with a higher risk of cardiovascular events, an observation that is provocative even if causality still needs careful testing [14]-[20]. PFAS exposures, meanwhile, have been associated with immune effects and dyslipidemia in multiple human studies, underscoring why many countries are moving to phase them out [21] [22]. From an ecological lens, chemical pollution is increasingly recognized as an under-appreciated driver of biodiversity loss, acting alongside habitat change and climate stress [23]-[30]. And climate change itself is a threat multiplier, deepening exposure through wildfire smoke, heat-chemistry interactions, and extreme events that mobilize contaminants, an intersection tracked by the Lancet Countdown [28]-[42]. In short, we already know that toxins are widespread, that multiple exposure routes (air, water, food, dust, consumer products) operate at once, and that human health and ecosystem health are entangled [30]-[52]. Yet big uncertainties make this a "conundrum". First, people and wildlife are exposed to *mixtures* of chemicals, not one compound at a time; mixture effects can be additive or synergistic, but are rarely captured by single-chemical risk limits [49]-[52]. Second, some chemical classes (notably endocrine-disrupting chemicals) can show low-dose effects and non-monotonic dose-response curves, challenging traditional assumptions about thresholds [7]-[10]. Third, exposure science still struggles to see the full exposome (the totality of lifetime exposures): high-resolution mass spectrometry helps, but translating untargeted "feature lists" into actionable toxicology remains a work in progress [53] [54]. Fourth, new observational signals like microplastics detected in human arteries raise concern but also debate about confounding and temporality; cautious interpretation is essential until mechanistic links are nailed down [14] [55]. Finally, windows of heightened susceptibility (fetal life, infancy, puberty) complicate inference and suggest that "average adult" risk estimates can understate harm [56]. To organize evidence across scales, regulators are turning to adverse outcome pathways (AOPs) which refers to a structured, cause-and-effect model linking a molecular initiating event to a series of key biological responses that ultimately produce a measurable adverse outcome, mechanistic chains linking a molecular "trigger" to disease or ecological harm, but AOP coverage is still incomplete for many contaminants and species [57] [58]. In short, what remains unknown is how complex mixtures act across sensitive life stages and which mechanistic routes matter most; resolving this is the core scientific bottleneck. From a bacteriologist's perch, toxins do not just damage microbes; they can also re-engineer microbial communities in ways that rebound on human and ecosystem health. Heavy metals, disinfectants, and some pesticides co-select for antibiotic resistance by favoring genes and plasmids that carry both metal- and antibiotic-resistance traits [59]-[74]. Wastewater treatment plants, where antibiotics, metals, biocides, and human microbes mingle, are repeatedly flagged as hotspots for antibiotic-resistance genes and mobile genetic elements that can move into environmental and clinical settings [75]-[78]. Within biofilms, close cell-to-cell contact and stress gradients can accelerate horizontal gene transfer, although transfer dynamics vary across biofilm layers. At the same time, environmental chemicals, including plastic additives and PFAS, can shift the gut microbiome in animal models, with metabolic and inflammatory knock-ons that plausibly mediate disease risk in humans [79] [80]. This microbial lens links personal health to river catchments and soils: ecosystems shape microbiomes; microbiomes govern biodegradation, nutrient cycling, and, under selective pressure, the spread of resistance [34]. That makes One Health (connecting human, animal, and environmental health) more than a slogan; it becomes a practical framework for tracing how chemical stressors ripple through microbial networks to clinical outcomes. Accordingly, this paper argues for a practical, mixture-aware, microbially-literate roadmap for environmental toxicology. This study integrates three toolkits. First, exposomics, pairing targeted panels with non-targeted high-resolution mass spectrometry, can map hundreds to thousands of chemicals in people and environments, then link them to disease signals [53]-[55]. Second, wastewater-based epidemiology can track population exposure and identify upstream sources for intervention, complementing individual biomonitoring [81]-[104]. Third, adverse outcome pathways knit mechanisms across scales, helping translate exposomic hits into plausible causal chains for human and ecological outcomes [57] [58]. While keeping sight of ecosystem endpoints, this focus is timely because chemical production and diversity continue to rise, exposure monitoring is finally scalable, and policy windows (from PFAS restrictions to water-quality standards) are opening. Concretely, this study (i) charts the evidence connecting these toxin classes to human and ecosystem effects; (ii) identifies where mixture and life-stage uncertainties block decision-making; (iii) demonstrates a combined exposome wastewater, AOP workflow that a regional health system or water authority could implement; and (iv) prioritizes interventions that deliver health equity and biodiversity co-benefits.

2. Overview of Environmental Toxins

What we mean by "environmental toxins" refers to chemicals (natural or synthetic) that, at environmentally relevant doses, can harm humans, wildlife, or ecosystem functions. The big, well-studied families include heavy metals (e.g., lead, mercury, cadmium, arsenic), pesticides (from organophosphates to neonicotinoids and herbicides), and industrial chemicals (e.g., PCBs, dioxins, PAHs, solvents). We also include air pollutants such as fine particulate matter ($PM_{2.5}$) and ground-level ozone because they are chemically mediated exposures with clear toxic effects [1]-[10] [15]-[25]. Finally, a fast-growing set of "emerging contaminants" spans per- and polyfluoroalkyl substances (PFAS), micro- and nanoplastics, pharmaceuticals, and flame retardants. Each of these classes has distinct sources and behaviors. Metals persist and bioaccumulate, PFAS resist degradation ("forever chemicals"), and many pesticides are designed to be bioactive, yet their real-world risk often lies in mixtures and continuous low-dose exposures [82]-[95]. Recent syntheses estimate that pollution (across chemical and airborne exposures) remains linked to roughly nine million premature deaths annually, about one in six global deaths, underscoring that chemical exposures are not niche problems [82]-[104]. Meanwhile, WHO's latest fact sheet attributes more than 1.5 million deaths in 2021 to lead alone, highlighting the cardiovascular toll in adults and neurodevelopmental harm in children. Equally sobering, a 2023/2024 Lancet Planetary Health modeling analysis estimated ~765 million IQ points lost among children under five in 2019 due to lead, with the burden concentrated in low- and middle-income countries [105].

2.1. Where Toxins Come from and How Big the Problem Is

Heavy metals typically enter air, water, and soils via mining/smelting, informal battery recycling, lead-acid battery and e-waste handling, and legacy lead paint and piping [83]-[92]. Pesticides are applied intentionally to croplands, and global agricultural pesticide use reached ~3.70 million tonnes of active ingredients in 2022, nearly double 1990 levels, with use intensity up 94% per hectare, evidence that chemical pressure is still rising [59]-[70] [74]. Industrial chemicals and by-products (e.g., dioxins, PAHs, solvents) come from combustion, manufacturing, and waste mismanagement; airborne $PM_{2.5}$ remains a leading driver of cardiopulmonary mortality (short-term $PM_{2.5}$ alone is now linked to ~1 million premature deaths per year, on top of long-term effects) [7]-[10]. At the same time, pharmaceuticals are now detectable in rivers on every continent: a 258-river survey (1052 sites across 104 countries) found widely distributed active pharmaceutical ingredients, with many sites exceeding ecological-risk thresholds. "Emerging" contaminants keep shifting the frontier: PFAS have credible human evidence for immunotoxicity (e.g., reduced vaccine antibody response), prompting clinical guidance and regulatory action; and microplastics have been physically detected in human carotid plaques in a New England Journal of Medicine cohort, where their presence was associated with higher major adverse cardiovascular events strong signals that need further mechanistic work but cannot be ignored for surveillance.

2.2. Why a Bacteriologist Cares: Microbes Sit at the Crossroads

From a microbiology lens, environmental toxins do more than injure cells; they reshape microbial communities and select for traits that matter to people and ecosystems. Metals, biocides, and some pesticides can co-select antibiotic resistance, because resistance genes to metals and antibiotics often sit together on the same plasmids or integrons [6] [106]. Wastewater treatment plants (WWTPs), where antibiotics, metals, disinfectants, and human microbes mix, are repeatedly identified as hotspots for antibiotic-resistance genes (ARGs) and mobile genetic elements; even when treatment reduces viable bacteria, extracellular DNA and ARGs can persist and disseminate downstream [6] [75] [106]. This matters because resistant pathogens emerging from environmental reservoirs complicate clinical care, a classic One Health linkage. On the ecosystem side, trace pharmaceuticals at river concentrations can modulate fish behavior and migration: a 2025 Science study showed that benzodiazepine (clobazam) exposure altered Atlantic salmon migration behavior and dam passage dynamics in the wild, clear proof that neuroactive contaminants can change life-history traits with unknown long-term population effects [24] [107] [108]. Put simply, chemical pollution is not just toxicology; it is microbial ecology plus evolution, with feedback loops from sewers and soils to clinics and conservation [1] [26].

Table 1. Major toxin classes, sources, exposure routes, and sentinel effects.

Toxin class	Typical sources	Main exposure routes	Sentinel human effects	Microbial/ ecosystem signals	Example recent sources
Heavy metals (lead, mercury, cadmium, arsenic)	Mining/smelting, informal recycling, legacy paint/pipes, artisanal gold mining	Ingestion (water/food), inhalation (dust), dermal (limited)	Cardiovascular mortality (adults), neurodevelopmental IQ loss (children)	Co-selection of ARGs; altered microbial community structure in sediments	WHO 2023 lead fact sheet; Lancet Planetary Health 2023 modeling; reviews on metal-AMR co-selection; WWTP ARG hotspots [105]
Pesticides (insecticides, herbicides, fungicides)	Agricultural use (field/crop protection), storage/handling	Food residues, drift/inhalation, water runoff	Acute poisonings; endocrine and neurodevelopmental concerns (compound-specific)	Impacts on beneficial insects/aquatic invertebrates; shifts in microbial functions	FAO 2022/2023 pesticide use stats; WHO/FAO JMPR residue reports [59]-[70]
Industrial chemicals/ byproducts (PCBs, dioxins, PAHs, solvents)	Combustion, manufacturing, waste incineration, spills	Inhalation of $PM_{2.5}$/PAHs, food chain bioaccumulation	Cancer, cardiopulmonary disease, and developmental risks	Biodiversity pressure via chronic sublethal toxicity	$PM_{2.5}$ short-term mortality analysis 2024 [9] [10] [105]
PFAS ("forever chemicals")	Fluorinated coatings, firefighting foams, and industrial effluents	Drinking water, food packaging, and dust	Immunotoxicity (reduced vaccine response), dyslipidemia	Reduced microbial degradation capacity; potential food-web transfer	National Academies 2022 clinical guidance; commentary on PFAS immunotoxicity evidence [21]
Microplastics & Nanoplastics	Fragmented plastics from products, textiles, and tire wear	Ingestion (food/ water), inhalation (indoor/outdoor dust)	Under study; signals include systemic inflammation, possible CVD risk markers	Ingestion by zooplankton/fish; vectoring of chemicals/microbes	NEJM carotid-plaque finding (2024) and updates [18]-[20]
Pharmaceuticals (APIs in waters)	Human/animal use, excretion, improper disposal, WWTP effluents	Drinking/ recreational waters (trace); biota uptake	Ecological concern > direct human risk at typical levels; sentinel is behavior	Fish behavior/ migration changes; endocrine disruption in wildlife	Global rivers survey; Science 2025 salmon migration study [84]-[100]

Bringing these strands together: (1) use is rising or plateauing at high levels for many chemicals (e.g., 3.70 Mt pesticides in 2022), (2) health burdens are large and uneven (e.g., >1.5 million deaths from lead in 2021; ~9 million deaths annually from pollution broadly), and (3) microbial systems translate chemical pressure into clinical and ecological outcomes (AMR co-selection; WWTP hotspots; neuroactive drug effects in wild fish) [59]-[70] [74]. For a bacteriologist, that means decision-making should not treat chemicals as isolated hazards but as pressures acting on microbial networks that tie households, hospitals, and habitats together. Thus, Table 1 provides an overview for use in subsequent sections to (i) quantify mixture exposures, (ii) track microbial endpoints (resistance, biofilm function), and (iii) align interventions with One Health priorities.

3. Methods/Review Approach

Evidence Search and Selection

The literature search was conducted between January 2018 and March 2025 using PubMed, Scopus, Web of Science, and Google Scholar. Key search terms included combinations of "environmental contaminants", "microbial resistance", "toxicology", "ecosystem health", and "One Health". Both peer-reviewed studies and reputable policy documents (e.g., WHO, UNEP, and FAO reports) were considered. Inclusion criteria comprised studies providing empirical or mechanistic insights into contaminant-microbiome-health interactions or class-based regulation frameworks. Exclusion criteria included purely theoretical papers without biological relevance, duplicate records, and reports lacking methodological transparency. Titles and abstracts were screened for relevance, and the final synthesis emphasized diversity in contaminant classes and geographic coverage to ensure balanced representation.

4. Mechanisms of Toxicity

How Toxins Harm People and Ecosystems

Across chemical families, several recurring biological "entry points" explain why low, chronic exposures can still add up to disease. First is oxidative stress: many metals, pesticides, and industrial chemicals raise reactive oxygen species, tipping cells out of redox balance. Cells counter with the KEAP1-NRF2 defense pathway, which ramps up antioxidant and detox enzymes; when this response is overwhelmed or dysregulated, damage spreads to lipids, proteins, and mitochondria, amplifying injury across organs (liver, brain, lung) and species (from bacteria to fish to humans) (**Table 2**). This redox axis is now a backbone concept in toxicology and drug safety alike [59]-[70]. A second route is DNA damage and genome maintenance failure. Genotoxins can nick or crosslink DNA directly, or provoke indirect damage by ROS, blocking replication, mutating tumor suppressors, and triggering chromosomal aberrations; modern assays and computational pipelines now map these signatures with increasing precision [6] [106]. Parallel to sequence changes, epigenetic alterations, DNA methylation shifts, histone marks, and non-coding RNAs reprogram gene expression after exposure, helping to explain lasting effects from brief hits and even intergenerational echoes in experimental systems and human cohorts. Third, endocrine disruption remains central for many consumer and agricultural chemicals. For persistent pollutants such as PFAS, global monitoring shows PFOA and PFOS concentrations ranging from 5-200ng/L in drinking water systems (EPA, 2024). Epidemiological evidence links chronic exposure to a 10% - 20% higher risk of thyroid and immune dysfunction, even below current safety thresholds. Hormone-like compounds can act at very low doses, and their effects often follow non-monotonic curves, so high-dose tests may miss real-world hazards. The low-dose debate continues, but multiple evaluations conclude that non-monotonicity and sensitive windows (fetal life, puberty)

Table 2. Common environmental exposures, mechanisms, and their effects on fertility.

Environmental Exposure	Proposed Impact on Fertility	Mechanism of Action
Pesticides/Herbicides (Endocrine disruptors)		
DST, DBCP	Reduced spermatogenesis, sperm motility, estrogenic effects; altered hormone levels; decreased testicular and seminal vesicle volume	Androgen receptor blockade
Organophosphates	Reduced sperm count, motility, viability, concentration, and morphology; increased DNA damage; hormonal imbalances	Decreased antioxidant capacity; increased gonadotropin production; altered testosterone metabolism
Atrazine	Reduced sperm motility; altered Leydig and Sertoli cell function	
Plastics (Endocrine disruptors)		
Phthalates	Reduced sperm concentration and motility; increased DNA damage	Mimic endogenous hormones; hormone receptor binding/blocking; germ cell apoptosis
BPA	Reduced sperm counts, motility, and concentration; increased DNA damage	Weak estrogen agonist; decreased androgen receptor expression
Heavy Metals		
Cadmium	Reduced sperm count, concentration, motility, and morphology	Endocrine disruption; impaired Leydig cell function; Sertoli cell apoptosis
Lead	Reduced sperm concentration, motility, and viability	Testicular damage; Sertoli and germ cell cytotoxicity
Mercury	Reduced semen quality; increased DNA damage; increased spontaneous abortion	Endocrine disruption
Arsenic	Reduced semen quality	Oxidative stress; Sertoli cell apoptosis; decreased testosterone
Other Exposures		
Natural gas, oil	Reduced sperm motility; increased DNA damage	Undefined
Radiofrequency electromagnetic radiation	Reduced sperm viability and motility; increased DNA damage	Oxidative stress; germ cell apoptosis
Air pollution	Reduced sperm morphology, motility, and increased DNA damage; impaired spermatogenesis	Oxidative stress; epigenetic changes; germ cell apoptosis
Noise pollution	Decreased testosterone; germ cell maturation arrest	Endocrine disruption; activation of stress response
Hyperthermia	Increased DNA damage	Testicular hypofunction; oxidative stress
Lifestyle Factors		
Diet, obesity	Reduced sperm count; decreased testosterone	Endocrine disruption
Caffeine	Controversial	Undefined
Tobacco	Reduced sperm count, morphology, and motility	Oxidative stress; cytotoxicity
Alcohol	Decreased testosterone; impaired spermatogenesis	Endocrine disruption
Marijuana	Reduced sperm motility and viability; decreased testosterone	Endocrine disruption; activation of sperm cannabinoid receptors
Anabolic-androgenic steroids	Impaired spermatogenesis	Suppression of gonadotropins
Opioids	Reduced sperm motility and concentration; decreased testosterone	Suppression of gonadotropins; direct action on spermatozoa

deserve default attention in risk assessment [42] [71] [72]. Fourth, the immune system is a frequent target. For example, PFAS, the "forever chemicals", show consistent evidence for immunotoxicity in humans, including reduced antibody responses to vaccines; importantly, standard regulatory test batteries rarely include developmental immunotoxicity, so historical assessments likely underestimated risk [21] [109]-[129]. Finally, a bacteriologist's lens adds two mechanism layers that connect clinics to catchments. Environmental stressors (metals, biocides, some herbicides, and drugs) co-select antibiotic resistance because resistance traits travel together on plasmids and integrons; biofilm micro-gradients then accelerate horizontal gene transfer [111] [112]. At the same time, exposures remodel gut and environmental microbiomes, shifting metabolism of xenobiotics (which can detoxify or bioactivate chemicals) and altering host signaling, immunity, and barrier function. These microbial shifts help translate chemical pressure into patient-level outcomes and ecosystem change. Hence, recent global syntheses indicate that co-selection pressure from heavy metals can increase antibiotic resistance gene abundance by 2 - 10 fold in contaminated soils and sediments. Cadmium and lead concentrations above 0.5 mg/L in wastewater are associated with 35% - 60% higher multidrug-resistant bacterial counts compared with uncontaminated sites [6] [106].

5. Human Health Impacts

Air and chemical pollution remain among the world's largest preventable health risks. For cardiovascular disease, chronic exposure to $PM_{2.5}$ is causally linked to ischemic heart disease and stroke; global assessments attribute millions of deaths and over 100 million DALYs annually to fine particles, with risk persisting even below many legal limits. Mechanistically, particles drive oxidative stress, endothelial dysfunction, and systemic inflammation, exact pathways seen in experimental models. Lead contributes heavily to CVD too; a 2019 modeling analysis estimated ~5.5 million adult CVD deaths attributable to lead exposure, disproportionately in low and middle-income countries. Together, these burdens frame why cutting emissions and legacy exposures is one of the fastest ways to save lives [7]-[10]. Neurodevelopment and neurodegeneration remain priority concerns. Prenatal and early-life lead exposures are associated with large, population-level IQ losses, an estimated 765 million IQ points in 2019 among children under five, confirming decades of cohort evidence and reinforcing that there is no safe level. For adults, several lines of evidence connect pesticide exposure to Parkinson's disease risk and progression; recent longitudinal and population-wide studies strengthen associations, while animal and cellular models implicate mitochondrial stress and α-synuclein aggregation [59]-[70]. These findings argue for protecting farmworkers and rural communities and for tracking pesticide mixtures, not just single active ingredients [74]. Reproductive and endocrine outcomes show mixture-sensitive patterns. Meta-analyses and multi-city studies link ambient air pollution and phthalate biomarkers to reduced semen quality, hormonal shifts, and possible fertility impacts; while effect sizes vary and confounding must be handled carefully, signals are consistent enough to inform precautionary policy and targeted exposure reduction. Parallel literature ties certain pesticides and endocrine-active compounds to menstrual irregularities and adverse pregnancy outcomes, again with life-stage sensitivity (**Table 2**). Clinically, screening and exposure counseling can be folded into preconception and prenatal care in high-burden areas [111] [112]. Cancer and immunity round out the major endpoints. Benzene is a human carcinogen with strong evidence for leukemia (especially AML) and signals for lymphomas and myeloma; mechanistic work shows genotoxic and chromosomal effects even at relatively low occupational exposures, justifying stringent limits. For immunity, human studies and expert reviews converge on PFAS-related immunosuppression, notably blunted vaccine responses in children, and dyslipidemia as a metabolic co-outcome, evidence that is now shaping clinical guidance. Taken together, the epidemiology and the mechanistic threads above (redox injury, DNA damage, endocrine signaling, epigenetic memory, and microbiome-mediated modulation) explain why environmental toxins sit squarely in mainstream medicine and public health, not at the fringes [6] [106] [111] [112]. Recent evidence suggests that pharmaceutical residues, especially antibiotics, frequently exceed predicted no-effect concentrations (PNECs) in effluent waters. Median concentrations of ciprofloxacin (0.12 μg/L) and azithromycin (0.08 μg/L) have been reported in low- and middle-income country settings, levels sufficient to enrich resistance genes within 72 hours of exposure [58] [105].

5.1. Why the Bacteriologist's Angle Matters for These Outcomes

Across endpoints, microbes are not spectators: they help set dose at the target (by transforming chemicals in the gut and waters), control colonization resistance and immune tone, and under selective pressure, amplify antibiotic resistance that feeds back into hospitals and communities [71]-[73]. That is why monitoring needs to track mixtures plus microbiome together, and why interventions that cut chemical pressure at wastewater plants and in agricultural run-off can yield double dividends for infection control and chronic-disease prevention. These links also explain the study design choices in pairing exposomics with wastewater surveillance and microbial endpoints to find practical "pressure points" for action (**Figure 1**).

5.2. Ecosystem Consequences

Environmental toxins do not simply affect individual organisms; they erode ecosystem integrity on multiple levels (**Figure 2**). A classic example is Minamata disease, where mercury from industrial discharge bioaccumulated in fish, devastating human and wildlife neurology. Similarly, DDT remains a stark reminder of pesticide overuse: it biomagnifies through food chains, thinning eggshells and collapsing bird populations like the American bald eagle. More recently, microplastics and plasticizers have infiltrated seagrass meadows and sediments [59]-[74].

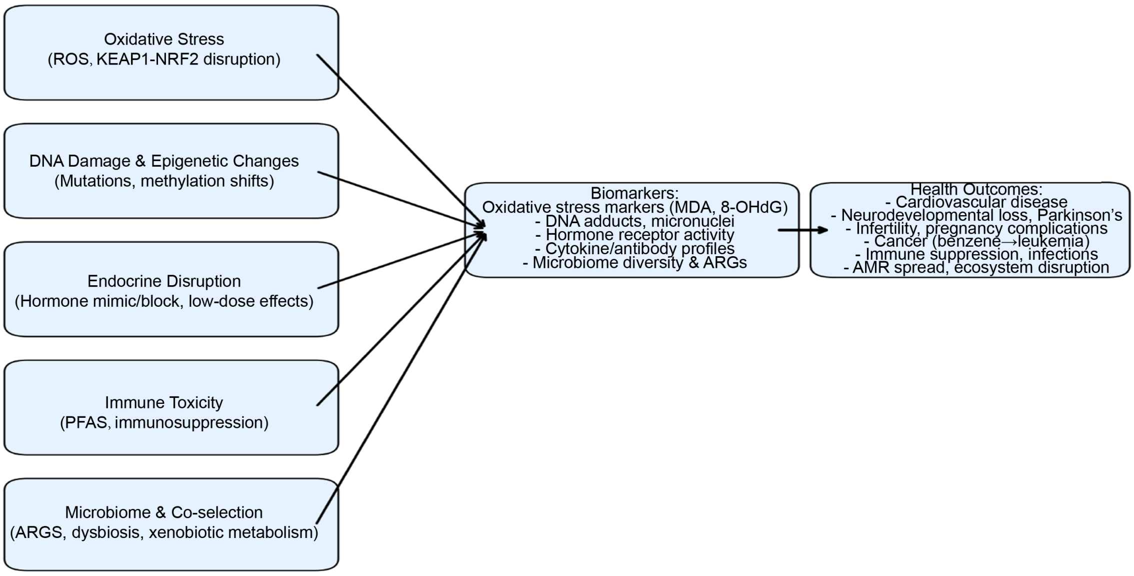

Figure 1. Shows a schematic diagram that illustrates the flow from mechanisms of toxicity → biomarkers → human health outcomes. Each mechanism (oxidative stress, DNA damage, endocrine disruption, immune toxicity, microbiome effects) connects to measurable biomarkers, which then link to specific health outcomes such as cardiovascular disease, cancer, infertility, immune suppression, and antimicrobial resistance spread.

Seagrasses, guardian habitats that store over $100 billion/year in ecosystem services in Southeast Asia are suffering stunted growth, reduced photosynthesis, and disrupted nutrient cycling due to microplastic interference and chemical leaching [34] [130]-[134]. These disruptions yield acute biodiversity loss and long-term degradation of ecosystem services such as water purification, carbon sequestration, and fisheries productivity [24] [34]-[41]. For example, impairment of nitrogen-fixing microbial communities in sediments due to microplastics complicates nutrient recycling and plant growth (**Figure 2**), thereby altering food webs [42]-[52]. Bioaccumulation of industrial chemicals like PCBs continues to magnify across trophic levels, posing chronic risks to predators at the top of food chains, including humans. In sum, environmental toxins reduce biodiversity, destabilize ecosystem functions, and carry far-reaching consequences for ecological resilience [34]-[40]. Quantitatively, studies show that in oil-impacted sediments of the Niger Delta, total petroleum hydrocarbon (TPH) levels exceeding 500 mg/kg have been associated with >70% loss of microbial diversity and over 50% decline in nitrogen-fixing bacteria, demonstrating the cascading ecological effects of hydrocarbon contamination [52].

6. Interplay between Human Health and Ecosystems

Human and ecosystem health are deeply intertwined: when toxins degrade ecosystems, they often rebound as heightened human exposures (**Figure 2**). Consider contaminated seagrass ecosystems: when microplastics and associated chemicals accumulate in foundational species, they ripple upward, depleting fisheries and damaging food security [135]-[138]. Similarly, loss of amphibians due to pollutants can alter

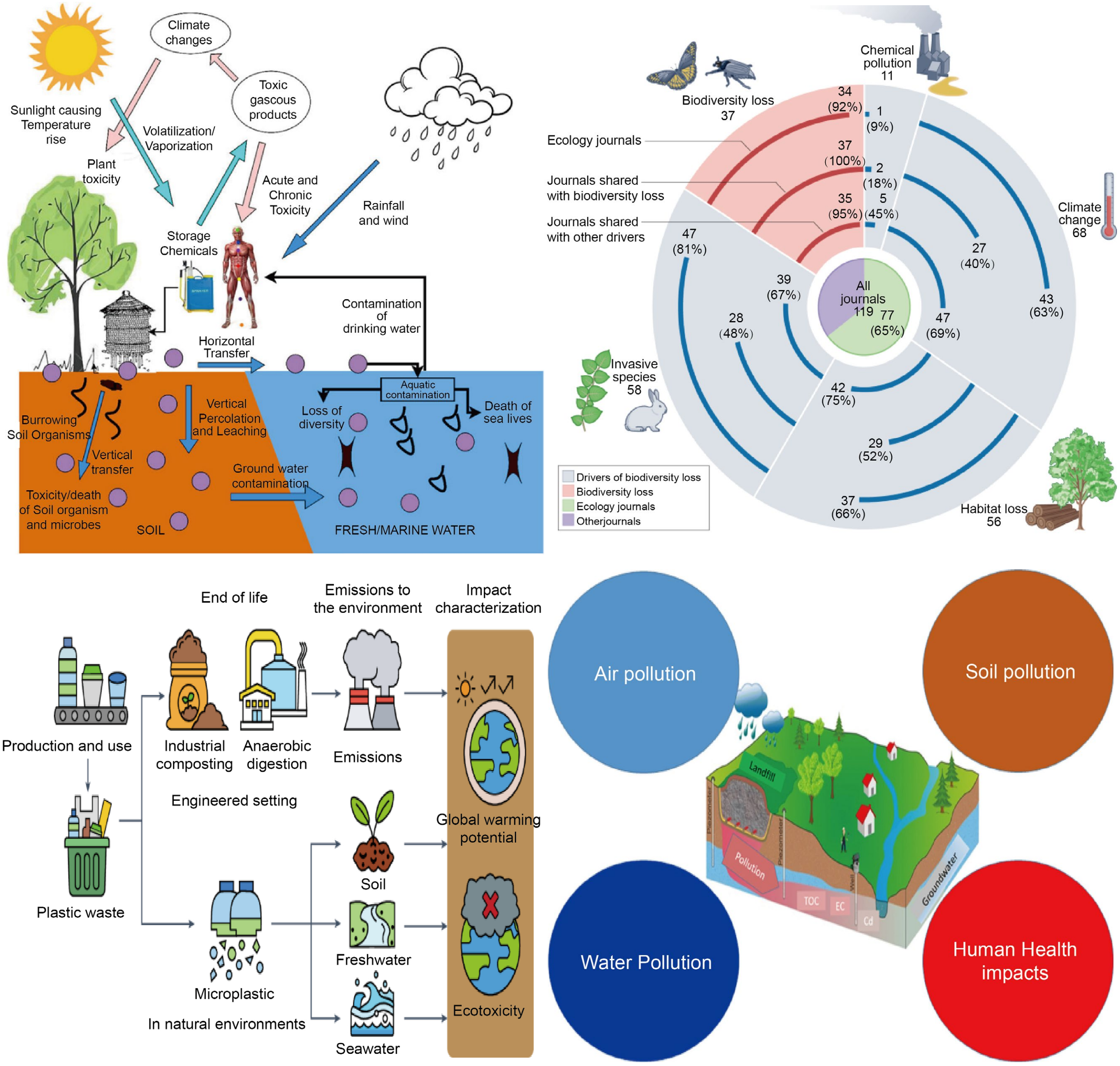

Figure 2. Illustrates the reinforcement of ecosystem-level impacts, from agrochemical pollution and biodiversity threats to plastic accumulation pathways.

disease-vector dynamics, potentially impacting human disease incidence. Mining, pesticide runoff, and industrial emissions, which disrupt water chemistry or biodiversity, ultimately compromise ecosystem services like clean drinking water, flood regulation, and crop pollination [59]-[70] [85]-[100]. Thus, toxic-loaded ecosystems increase vulnerability, particularly among communities reliant on natural resources. Restoring ecosystem functions, such as reestablishing seagrass beds or improving soil microbial health, thus represents both ecological and public-health strategies. This interplay underscores the necessity of integrated frameworks like One Health, which recognize the dual imperatives of protecting ecosystems and reducing human disease burdens [109].

Microbiome and New Frontiers

Expanding on the microbial lens, the human microbiome and broader environmental microbial communities are new frontiers in toxin research. Microbial systems both mediate and modulate chemical effects: environmental strains may transform xenobiotics into benign or toxic forms, while host microbiota can determine chemical absorption, metabolism, and immune responses. This intersection is the domain of pharmacomicrobiomics, which investigates how microbiome variation affects drug and toxin handling, defining toxicity in profoundly personalized ways. Technologically, multi-omics and metagenomic profiling now allow ecosystem-level environmental monitoring and human health linkage. Large-scale exposome studies are integrating DNA methylation, gene expression, proteomics, metabolomics, and metagenomics to trace how complex exposures manifest at molecular, microbial, and health levels [111] [112]. Genome-resolved metagenomics helps reconstruct functional microbial communities in environmental samples deeply impacted by toxins, without needing to cultivate them in labs. The convergence of these tools enables real-time, predictive insights into chemical exposures, microbial adaptation, and disease risk trajectories, opening space for precise, ecosystem-aware interventions.

7. Policy, Prevention & Remediation Strategies

7.1. Where Policy Has Worked and Where It Hasn’t

Global agreements such as the Stockholm Convention (persistent organic pollutants) and the Minamata Convention (mercury) show that international law can reduce production, use and releases of the most hazardous legacy chemicals; effectiveness evaluations report measurable progress but also persistent implementation gaps, particularly in waste management, substitution, and monitoring capacity in low and middle-income countries (LMICs). The same pattern appears with PFAS: policy momentum is rising (EU restriction proposals, US EPA actions, and national advisory reports), yet regulation is often piecemeal (single-compound limits or small subsets of PFAS) while emissions and product-phase-out policies lag behind scientific concerns about the entire class of compounds. These partial rules protect some communities but leave important exposure pathways open and create regulatory complexity for enforcement [138].

7.2. Policy Weaknesses That Matter for Public Health and Ecosystems

Major weaknesses include: (a) single-chemical risk frameworks that poorly capture mixtures and low-dose endocrine or developmental effects; (b) uneven monitoring capacity, with rich countries able to run exposomics (the comprehensive study of all environmental exposures an organism experiences throughout its lifetime and their corresponding biological effects, integrating chemical, physical, and social stressors with omics-based health data) and HRMS programs while many LMICs rely on infrequent spot tests; (c) limited integration of microbial endpoints (AMR, ARGs, microbiome shifts) into chemical regulation or water-quality standards; and (d) technology and scale gaps, for example, PFAS removal/destruction technologies (adsorption, ion-exchange, AOP, electrochemical and thermal destruction) show promise but remain costly, energy-intensive or insufficiently validated at full scale. These structural issues mean that policies sometimes reduce one hazard only to leave or create others (disposal, replacement chemicals, or transferred burdens).

7.3. Prevention-First: Upstream Policy Levers That Are Feasible and High-Impact

Preventing release is almost always a higher value than end-of-pipe fixes. Key feasible levers are: (a) class-based regulation (e.g., broad PFAS restrictions rather than one-by-one), (b) extended producer responsibility (EPR) and circular-economy mandates to reduce waste and design out persistent chemistries, (c) strict controls on primary sources (e.g., informal e-waste recycling, artisanal mining), and (d) green-chemistry incentives for benign alternatives. These approaches reduce future remediation costs and lower the chance of regrettable substitutions. The recent OSTP and EU assessments emphasize these prevention directions and investment needs.

7.4. Midstream: Surveillance, Treatment Upgrades, and Targeted Remediation

Where contamination already exists, a layered approach is pragmatic: (a) surveillance scale-up using wastewater-based epidemiology (WBE) and targeted exposomics to prioritize hotspots and measure intervention impact; (b) WWTP upgrades focused on pharmaceutically active compounds and ARGs, combining biological secondary treatment with tertiary steps (activated carbon/adsorption, membrane filtration, advanced oxidation) can cut many pharmaceutical residues and some PFAS precursors, though PFAS removal often requires additional ion-exchange or thermal destruction for ultimate disposal; (c) on-site hospital wastewater pre-treatment for high-risk streams reduces loads to municipal plants; and (d) phytoremediation and bioremediation for many heavy-metal and organic pollutants in soils and sediments offer low-cost, community-friendly options but require careful species selection and disposal of contaminated biomass. Technology reviews and pilot studies underline that matching the right tool to the contaminant matrix and local capacity is crucial, as no single silver-bullet technology exists.

7.5. Downstream: Destruction and Disposal for Legacy/High-Risk Wastes

For persistent contaminants (PFAS, some halogenated POPs), final-stage destruction is often required. Promising approaches include high-temperature thermal treatment (plasma/pyrolysis) and electrochemical/photochemical degradation under well-controlled conditions; these are advancing but need standardized performance metrics and lifecycle assessments to ensure they don't produce toxic by-products [109]. Pilot and early industrial reports stress the need for rigorous monitoring and validated destruction claims before scaling.

7.6. Social and Equity Aspects of Prevention and Remediation

Policies must prioritize communities with high cumulative exposures (informal recyclers, downstream communities, Indigenous peoples). Community engagement improves data quality and trust, and community-centred monitoring (citizen science paired with laboratory validation) can accelerate detection and politically sustain interventions. International funding and technical assistance should emphasize capacity building in LMICs for both monitoring and remediation [26] [29] [131] [139]-[143].

7.7. Concrete, Evidence-Based Policy Recommendations

i. Adopt class-based restrictions where toxicological plausibility supports it (PFAS-style).

ii. Require producer responsibility & disclosure of chemical constituents in products, to enable safer substitutions.

iii. National rollouts of sentinel wastewater surveillance + exposomics pilots to prioritize action and evaluate effectiveness.

iv. Invest in WWTP tertiary upgrades (targeted adsorption/AOP + ion-exchange where PFAS are present) and pilot validated PFAS destruction.

v. Integrate microbial metrics (ARGs, resistance plasmids, functional metagenomics) into chemical risk assessments and water-quality standards.

vi. Fund just transitions for exposed communities (health screening, remediation jobs, and safe recycling alternatives).

8. Outstanding Gaps and Future Directions

8.1. What Remains Unknown (Priority Knowledge Gaps)

i. Mixtures and cumulative risk: existing regulatory toxicology is dominated by single-chemical paradigms; we lack validated, scalable frameworks for assessing realistic environmental mixtures and their non-linear interactions across life stages.

ii. Mechanistic links via the microbiome: we need causal, longitudinal evidence showing when and how microbiome shifts mediate human disease after environmental exposures (and the conditions under which microbiomes detoxify versus bioactivate chemicals).

iii. Scalable PFAS destruction: many lab-scale methods exist, but validated, affordable, and energy-efficient full-scale destruction routes with clear by-product profiles are still scarce.

iv. Environmental AMR transmission dynamics: the pathways, rates, and realistic risks for ARG transfer from environmental reservoirs to clinical settings remain poorly quantified in many contexts.

v. Equitable monitoring and data sharing: LMICs often lack baseline data; global datasets are uneven, limiting risk prioritization and cross-border accountability.

8.2. High-Value Research & Technology Priorities

i. Integrative exposome cohorts that pair HRMS untargeted chemistry with longitudinal multi-omics (methylome, transcriptome, metabolome, metagenome) and health endpoints. These cohorts should include diverse geographies and high-exposure communities so findings are generalizable. Recent exposomics reviews outline the methods and informatics needs.

ii. Sentinel wastewater networks that combine chemical analytics, ARG surveillance, and pathogen genomics for near-real-time community exposure and AMR risk mapping. Wastewater epidemiology is mature enough to support large-scale deployment but needs funding and governance frameworks.

iii. Standardized adverse outcome pathway (AOP) libraries for mixture components and microbial interactions, to translate molecular signals into regulatory endpoints and to prioritize data-poor chemicals for testing.

iv. Scalable PFAS destruction pilots with transparent, independently validated performance metrics (destruction efficiency, by-products, lifecycle costs). Cross-sector testbeds (industry + academia + regulators) can fast-track validation.

v. AMR eco-epidemiology studies that quantify horizontal gene transfer rates in WWTPs, soils, and agricultural systems under chemical co-selection pressures, and test interventions that reduce co-selection (e.g., metal source control, reduced biocide use).

8.3. Interdisciplinary and Capacity-Building Needs

i. Cross-discipline centers (One Health hubs) that colocate chemists, microbiologists, exposure scientists, epidemiologists, and social scientists to run integrated studies and translate results to policy. The Lancet and others argue One Health is essential for AMR and chemical threats.

ii. Shared data platforms and open analytic pipelines for HRMS/exposomics, environmental metagenomics and PFAS treatment performance metrics, standardization accelerates uptake and ensures comparability.

iii. Technology transfer programs and financing to help LMICs acquire monitoring tools and pilot remediation (e.g., modular adsorption units, community phytoremediation projects) while building local lab capacity. International conventions already emphasize technical support; targeted funding modalities should follow.

8.4. Regulatory Innovation and Ethical Guardrails

i. Require mixture testing and cumulative risk frameworks where evidence suggests overlapping exposure pathways and shared modes of action (e.g., neurotoxicants in early life).

ii. Implement transparent reporting and right-to-know policies for community exposure data, coupled with support for appropriate clinical follow-up to avoid harm from uninformed testing. Recent national PFAS reports illustrate tensions in testing policy and risk communication.

Thus, policymakers, scientists, and communities face a twofold challenge: (1) prevent future toxic legacies through upstream policy (class-based bans, producer responsibility, green chemistry), and (2) manage existing contamination with a realistic toolbox (surveillance, WWTP upgrades, validated destruction where needed, and community remediation). Closing the key knowledge gaps, mixtures, microbiome mediation, PFAS destruction, and AMR transmission will require sustained investment in integrative exposomics, wastewater sentinel systems, One Health research hubs, and equitable technology transfer. When combined, this policy, technical, and research pathways provide a feasible roadmap for moving from reactive cleanup to preventive stewardship of chemical and microbial risks.

9. Implications for Policy and Interventions

The evidence reviewed highlights that environmental toxins cannot be effectively managed through fragmented or chemical-by-chemical approaches. Instead, regulatory frameworks must evolve toward class-based restrictions (e.g., addressing PFAS as a group), comprehensive mixture risk assessments, and cumulative exposure evaluations. Policymakers should also strengthen upstream controls by embedding extended producer responsibility (EPR), incentivizing green chemistry, and investing in alternative non-toxic materials. Importantly, interventions must not be confined to high-income nations: capacity building for low- and middle-income countries (LMICs) remains crucial to ensure equitable protection. Surveillance systems such as wastewater-based epidemiology (WBE) and environmental exposomics should be mainstreamed to detect early signals of toxic exposures and antimicrobial resistance hotspots, thus linking ecological monitoring with public health decision-making. Moreover, integrating microbial endpoints, including microbiome alterations and antimicrobial resistance gene dynamics, into risk assessment frameworks will align chemical policies with One Health objectives. Globally, concentrations have been reported across contaminant classes. Cumulative exposure to polluted environments contributes to an estimated 9 million premature deaths annually, or roughly 16% of global mortality, underscoring the urgent need for integrated pollution control and One-Health policies [12]. Taken together, the interventions point to a dual strategy of prevention first, complemented by targeted remediation where legacy contamination persists.

10. Limitations of This Review

While this narrative synthesis integrates diverse strands of evidence linking environmental contaminants, microbial ecology, and health outcomes, it is subject to certain limitations. The narrative design inherently carries selection and interpretation bias, as evidence inclusion was guided by thematic relevance rather than exhaustive systematic screening. Additionally, publication bias may have influenced the available literature, since studies reporting positive or significant associations are more likely to be published. To capture emerging data, this review also incorporated preprints and grey literature, which, although valuable for timeliness, may not have undergone full peer review and should therefore be interpreted with caution. Future research should complement this narrative overview with systematic reviews or meta-analyses that quantitatively assess effect sizes, confidence intervals, and evidence quality across contaminant classes.

11. Conclusion

The toxin conundrum reveals itself as both a scientific and societal challenge: toxins infiltrate ecosystems, accumulate through food chains, disrupt microbiomes, and ultimately undermine human health. The evidence demonstrates that mechanistic pathways (oxidative stress, DNA damage, endocrine disruption, and microbiome-mediated toxicity) translate into profound health burdens, including neurodevelopmental disorders, cancers, reproductive impairments, and immune suppression. Equally, toxins destabilize ecosystems, eroding biodiversity, impairing nutrient cycling, and diminishing the ecosystem services upon which human societies depend. This multidimensional problem demands integrated action. Moving forward, success will require aligning science, governance, and community engagement into coherent prevention and intervention frameworks.

12. Summary of the Findings

i. **Environmental toxins are pervasive**, spanning heavy metals, pesticides, pharmaceuticals, PFAS, plastics, and emerging contaminants.

ii. **Mechanisms of toxicity are well-characterized**, with oxidative stress, DNA damage, endocrine disruption, immune toxicity, and microbiome perturbation as central pathways.

iii. **Human health impacts are wide-ranging**, encompassing acute (respiratory illnesses, poisoning events) and chronic (cancers, infertility, neurodevelopmental delays) outcomes.

iv. **Ecosystem-level impacts** include biodiversity collapse (e.g., DDT and avian declines, mercury in aquatic food webs), bioaccumulation, and disruption of carbon/nutrient cycling.

v. **Human-ecosystem interdependence** creates feedback loops: degraded ecosystems amplify human exposure risks, weakening resilience.

vi. **Microbiome research is reshaping understanding**, with microbes acting both as mediators and modulators of toxic exposures, advancing the frontier of exposome science.

vii. **Policy strengths and weaknesses are uneven**: international treaties (Stockholm, Minamata) show progress, but major gaps persist in regulating mixtures, PFAS, pharmaceuticals, and antimicrobial co-selection pressures.

13. Recommendations

i. Adopt class-based regulation for persistent and bioaccumulative chemicals to avoid piecemeal bans.

ii. Strengthen prevention-first strategies, including green chemistry incentives, safer substitutions, and circular economy approaches.

iii. Invest in surveillance systems, especially wastewater epidemiology and exposomics, to provide early warnings of population-level exposures.

iv. Integrate microbial endpoints (AMR, microbiome shifts) into environmental and health risk assessments.

v. Scale up remediation technologies (adsorption, advanced oxidation, ion-exchange, bioremediation, PFAS destruction pilots), matched to local context and capacity.

vi. Prioritize equity by directing resources to vulnerable and disproportionately exposed communities.

vii. Foster interdisciplinary collaborations, linking bacteriologists, toxicologists, epidemiologists, policymakers, and social scientists through One Health hubs.

14. Health Significance

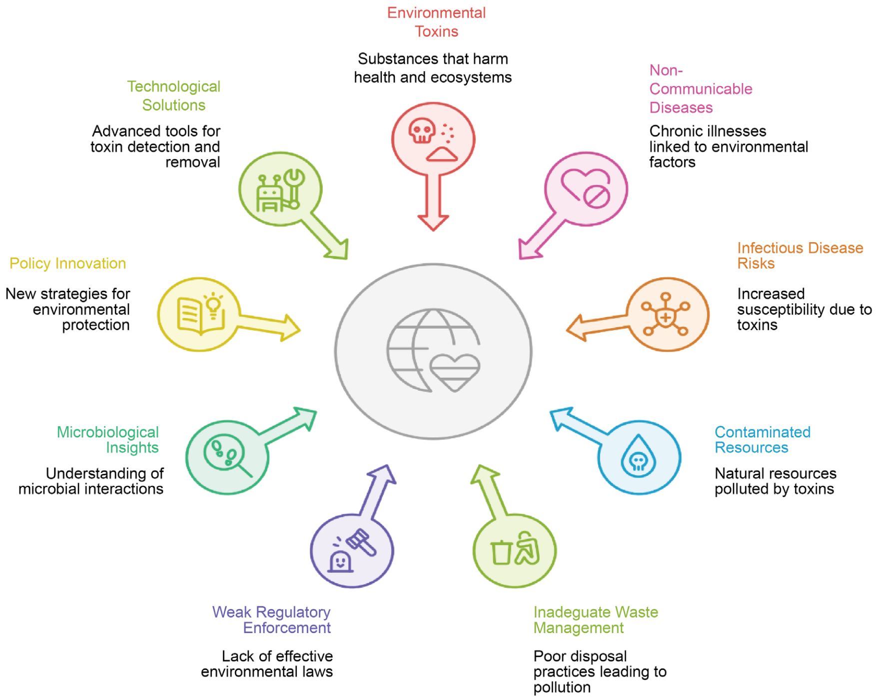

Figure 3. Addressing Environmental Toxins for Global Health. Source: Author design.

From a public health perspective, environmental toxins represent a silent but escalating global health crisis. They not only increase the burden of non-communicable diseases but also compound infectious disease risks through immunotoxicity and antimicrobial resistance co-selection. For communities in LMICs, the health significance is amplified by reliance on contaminated natural resources, inadequate waste management, and weak regulatory enforcement. Addressing this conundrum is therefore not only a matter of environmental protection but also of disease prevention, health equity, and sustainable development. By bridging microbiological insights with policy innovation and technological solutions, this work underscores the urgency of integrated action. The next steps must be decisive: moving from fragmented, reactive responses toward coordinated, preventive strategies that safeguard ecosystem resilience and human health alike. Thus, graphically it is represented (**Figure 3**) as.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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