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The double-edged nanoparticle: remediation benefits *vs.* mechanistic toxicity risks in aquatic systems

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Engineered nanomaterials (ENMs) offer a double-edged sword for aquatic remediation: while serving as powerful agents for pollutant removal, their inherent reactivity creates significant ecotoxicological risks. This critical review deconstructs this duality by providing an integrated analysis of remediation benefits *versus* mechanistic hazards. It is argued that the physicochemical properties driving remedial function—such as high surface reactivity and redox potential—are the shared origin of the molecular initiating events of toxicity. For instance, while photocatalytic ENMs can achieve $>90\%$ degradation of recalcitrant organics, this same non-selective reactivity can trigger a 1.5–2-fold increase in intracellular ROS in non-target aquatic organisms. The analysis reveals how this relationship is dynamically modulated by environmental transformations (*e.g.*, eco-corona formation, aggregation), creating profound challenges for conventional risk assessment. Consequently, a paradigm shift from a reactive, *post hoc* evaluation to a proactive safe-and-sustainable-by-design (SSbD) framework is advocated. This approach, which embeds mechanistic toxicology as an *a priori* design tool, is presented as the critical pathway to rationally decouple efficacy from hazard. Only through this integrated perspective can the transformative potential of nanoremediation for ensuring global water security be realised through sustainable design.

Environmental significance

The sustainable deployment of nanoremediation is hindered by a fundamental paradox: the physicochemical properties driving pollutant removal are also the primary drivers of ecotoxicity. This review critically dissects this benefit–risk nexus, revealing how environmental transformations dynamically modulate the hazard profile of nanomaterials. By synthesising the shared mechanistic origins of function and harm, this work highlights the limitations of conventional, *post hoc* risk assessment. It establishes the urgent need for a paradigm shift towards a proactive safe-and-sustainable-by-design (SSbD) approach, where mechanistic toxicology informs the *a priori* design of nanomaterials to decouple efficacy from hazard.

1. Introduction

The global water cycle faces unprecedented anthropogenic stress, with aquatic ecosystems increasingly serving as the ultimate sinks for a vast array of contaminants originating from the technosphere.[1–4] The relentless expansion of industrialisation and intensive agriculture discharges a complex mixture of chemical stressors—ranging from heavy metal cations (*e.g.*, Cd^{2+}, Hg^{2+}, Pb^{2+}) to bioactive micropollutants—posing a profound threat to ecological stability.[5–7] Legacy water treatment infrastructures are fundamentally ill-equipped to eliminate these recalcitrant contaminants, which often persist at trace concentrations (ng L^{-1} to μg L^{-1}) yet exert potent biological effects. For instance, endocrine-disrupting compounds can induce reproductive failure in aquatic vertebrates at concentrations below 1 ng L^{-1}, while persistent organic pollutants (POPs) bioaccumulate to toxic levels in higher trophic organisms.[2,4,8–10]

The pseudo-persistence and bioaccumulation potential of these substances demand a paradigm shift towards advanced, robust, and highly efficient remediation technologies. In this context, nanotechnology offers a potent, and potentially disruptive, toolkit for environmental remediation.[11–14] Engineered nanomaterials (ENMs) exhibit extraordinary physicochemical properties that are not apparent in their bulk-phase counterparts, originating from their vast specific surface areas, high densities of coordinatively unsaturated surface sites, and quantum confinement effects.[15,16] These attributes enable ENMs to function as powerful, nanoscale chemical reactors.

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They offer exceptionally high adsorption capacities, potent catalytic and photocatalytic activity for pollutant mineralisation, and unique redox functionalities. Indeed, ENMs based on metal oxides (*e.g.*, TiO_2, CeO_2), carbon allotropes (*e.g.*, graphene oxide, CNTs), and zero-valent metals (*e.g.*, nanoscale zero-valent iron, nZVI) have demonstrated remarkable efficacy in sequestering or degrading some of the most intractable aquatic pollutants.[17–21]

However, this high efficacy presents a fundamental mechanistic paradox. The very properties that confer upon ENMs their remediation prowess are inextricably linked to the mechanisms that drive their ecotoxicological hazard. The high surface reactivity, engineered to break down robust chemical bonds in a pollutant, can similarly disrupt the delicate lipoprotein structures of a cell membrane.[22,23] The capacity of a semiconductor nanoparticle like TiO_2 to generate highly oxidising hydroxyl radicals ($^{\bullet}OH$) for mineralising a dye molecule is the same process that induces oxidative stress, lipid peroxidation, and genotoxicity in aquatic organisms.[22] The strong affinity of a surface-functionalised ENM for capturing a heavy metal ion is governed by the same chemical principles that might allow it to strip essential metallic co-factors from vital enzymes.

This "double-edged sword" is not merely an analogy but a reflection of a shared mechanistic origin, creating an inherent tension between engineered function and environmental safety. Consequently, the intentional deployment of these materials for *in situ* remediation necessitates a rigorous and predictive understanding of their potential for adverse ecological outcomes.[24–27] This is particularly critical given the evolving regulatory landscape; frameworks such as EU REACH (specifically Annex XIII regarding persistence and bioaccumulation) and standardised testing protocols like OECD TG 318 (dispersion stability) and TG 319 (bioaccumulation) increasingly demand a granular understanding of nanomaterial behaviour in complex aquatic matrices.[28,29] Much of the existing literature has addressed the efficacy and the ecotoxicology of remediation ENMs as separate, often disconnected, fields of inquiry.[24,26,27] Efficacy studies are typically performed under idealised laboratory conditions optimised for performance, while toxicity assessments are conducted using standardised protocols that may not reflect realistic exposure scenarios.

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Unlike prior reviews that have catalogued remediation efficiency and ecotoxicity as separate domains, this critical review synthesises them as mechanistically coupled phenomena. The central thesis of this work is that a truly predictive assessment of nanoremediation requires moving beyond a parallel evaluation of 'benefit' and 'risk' towards an integrated analysis of the shared physicochemical phenomena that govern both. This review critically interrogates the mechanistic interface between remediation and toxicity. It moves beyond a reliance on apical toxicity endpoints (*e.g.*, mortality, LC_{50}), which, while vital for regulation, offer little insight into causative pathways. Instead, the focus is placed squarely on **mechanistic nanotoxicity**—the molecular and cellular-level perturbations that initiate adverse outcomes—and how these mechanisms correlate with the chemical processes engineered for remediation. To this end, this review addresses several critical questions:

1. To what extent are the surface and solution-phase reactions underpinning pollutant remediation **mechanistically coupled** to the pathways of cellular and molecular toxicity?
2. How do dynamic, environmentally relevant transformations (*e.g.*, aggregation, dissolution, eco-corona formation) simultaneously modulate both the remediation efficiency and the toxicological hazard profile of an ENM post-application?
3. Can the principles of mechanistic toxicology be leveraged to inform the *a priori* '**Safe-and-Sustainable-by-Design**' (**SSbD**) of next-generation ENMs, enabling the decoupling of efficacy from toxicity?

To address these questions, this review first critically evaluates the performance of principal remediation ENM classes (the "Benefit" edge). It then synthesises the current understanding of their environmental fate and transformation, the critical bridge between application and exposure. Subsequently, it provides a critical synthesis of the core mechanistic toxicity pathways in aquatic organisms (the "Risk" edge). Finally, it seeks to unify these threads at the "Nexus", critically analysing the benefit–risk balance and charting a course for future research to foster the development of genuinely sustainable and effective nanoremediation strategies.

2. The "Benefit" edge: nanomaterial-enabled aquatic remediation

The application of engineered nanomaterials (ENMs) to aquatic remediation leverages their distinct capacity to interact with and neutralise contaminants at efficiencies that substantially surpass

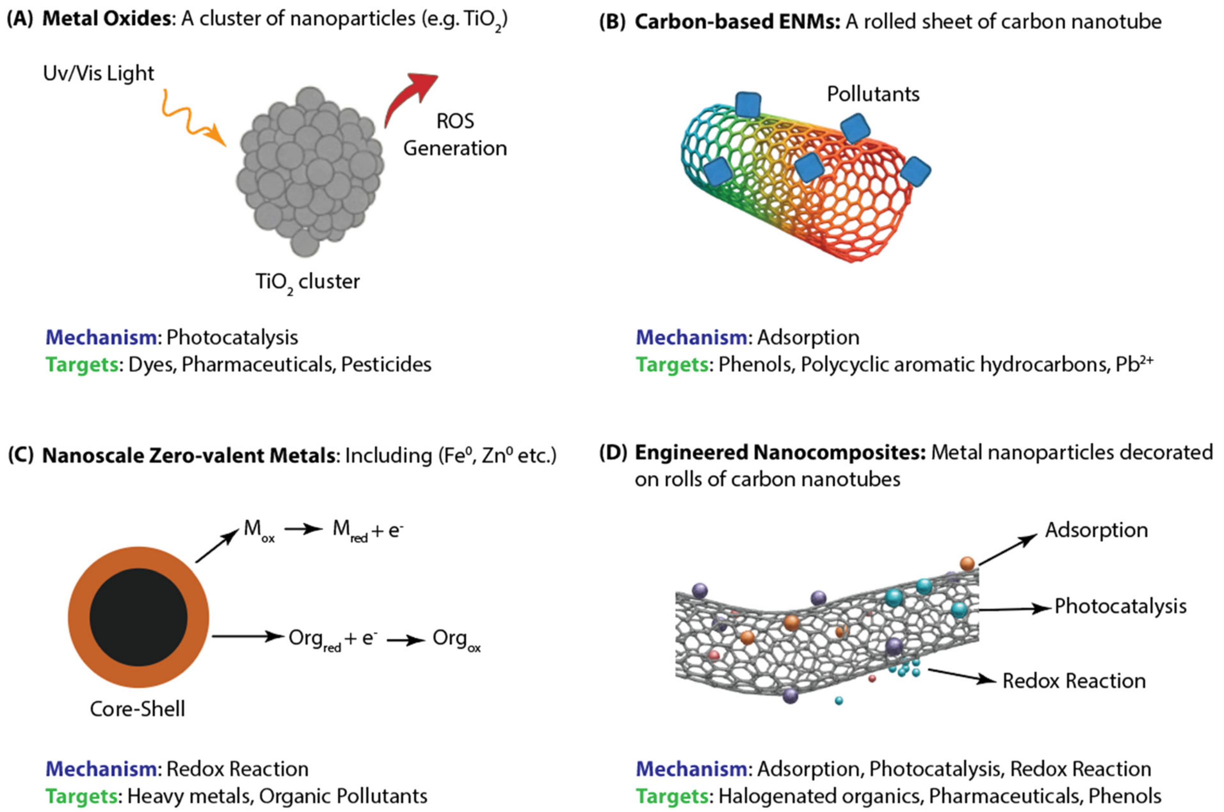

Fig. 1 Schematic overview of the four principal classes of engineered nanomaterials (ENMs) used for aquatic remediation. For each class—(A) **metal oxides**, (B) **carbon-based ENMs**, (C) **zero-valent metals**, and (D) **nanocomposites**—the diagram illustrates a representative core structure, the primary chemical or physical remediation mechanism (*e.g.*, photocatalysis, adsorption, redox reaction), and the respective classes of target aquatic pollutants (*e.g.*, dyes, heavy metal ions, persistent organic pollutants).

those of conventional bulk materials. This superior performance is a direct manifestation of their nanoscale dimensions, which give rise to expansive specific surface areas and unique electronic and quantum properties. These characteristics facilitate a spectrum of potent removal mechanisms, which can be broadly categorised into three mechanistic pillars: (i) high-capacity sorption of metals and organic molecules; (ii) advanced oxidation and reduction processes for contaminant transformation; and (iii) synergistic multifunctional systems that integrate multiple functionalities. To systematically evaluate this landscape, remediation ENMs are classified based on their dominant physicochemical mode of action: semiconductor photocatalysts (metal oxides), high-surface-area adsorbents (carbonaceous ENMs), and redox-active agents (zero-valent metals), followed by emerging composite architectures. This section critically deconstructs their operational principles and performance limits. The primary material classes, their target pollutants, and core mechanisms are conceptualised in Fig. 1 and representative examples are detailed in Table 1.

2.1. Key classes of remediation ENMs

- **Metal oxides: photocatalysts and selective adsorbents**

This class represents one of the most mature and extensively investigated groups of ENMs, valued for their chemical stability, low production cost, and versatile functionality.[30,31] Semiconductor metal oxides, with titanium dioxide (TiO_2) and zinc oxide (ZnO) as the archetypes, are pre-eminent photocatalysts. Upon irradiation with photons whose energy ($h\nu$) exceeds the material's band-gap energy (E_g), an electron (e^-) is promoted from the valence band (VB) to the conduction band (CB), generating a highly oxidising hole (h^+) in the VB.

These charge carriers migrate to the nanoparticle surface and initiate a cascade of reactions with adsorbed water and oxygen to produce non-selective, powerfully oxidising reactive oxygen species (ROS), principally the hydroxyl radical ($^{\bullet}OH$).[32]

$$\text{Metal Oxide} + h\nu \rightarrow e^-_{cb} + h^+_{vb}$$

$$h^+_{vb} + H_2O \rightarrow OH^{\bullet} + H^+$$

$$e^-_{cb} + O_2 \rightarrow O_2^{\bullet -}$$

These radicals can subsequently mineralise a vast array of recalcitrant organic pollutants into benign end-products such as CO_2 and H_2O.[33–35] Recent studies from 2022–2024 have highlighted the dual nature of this mechanism; for example, while enhanced charge separation in heterojunction

Table 1 Overview of remediation ENMs, their key physicochemical properties and major limitations

ENM class	Example ENM	Key properties	Remediation mechanism(s)	Target pollutants	Typical adsorption capacity ($Q_{\max}$) or rate constant (k_{app})	Limitations and concerns	Ref.
Metal oxides	Anatase TiO_2	Size: 10–30 nm; surface area: 50–200 $m^2\ g^{-1}$; band gap: ~3.2 eV	**Photocatalysis** (UV-A)	Dyes, phenols, pharmaceuticals, pesticides, pathogens	70–90% degradation of various organics in lab studies k_{app}: 0.01–0.1 min^{-1} (for organics)	Requires UV irradiation; efficiency reduced by NOM scavenging of ROS; potential for phototoxicity to non-target organisms	193–197
Metal oxides	Magnetite (Fe_3O_4)	Size: 20-50 nm; superparamagnetic; PZC: ~6.5	Adsorption	Arsenic (As(III/V)), lead (Pb^{2+}), chromium (Cr(VI))	$Q_{\max}$: 10–100 mg g^{-1} (for heavy metals). Notably, >10 mg g^{-1} for As(V)	Susceptible to oxidation; performance is pH-dependent; potential for leaching of iron ions. Magnetic recovery is energy-intensive at scale	198–200
Carbon-based	Graphene oxide (GO), carbon nanotube	Single to few layers; high surface area: >500 $m^2\ g^{-1}$; oxygen functional groups	**Adsorption** (π–π stacking, electrostatic, H-bonding); **photocatalysis** (UV-A)	Dyes (*e.g.*, methylene blue), PAHs, heavy metals (Pb^{2+}, Cd^{2+}), pharmaceuticals	$Q_{\max}$: 200–500 mg g^{-1} (for dyes/metals)	Aggregates in hard water; potential for membrane disruption ("nano-blade" effect); difficult to recover from water	201–205
Zero-valent metals	nZVI	Size: 20–100 nm; high surface area: 20–60 $m^2\ g^{-1}$; $E^0 = -0.44$ V	Chemical reduction	Chlorinated solvents (TCE), Cr(VI), As(V), nitrates	k_{obs}: 0.005–0.05 min^{-1} (for TCE degradation)	Rapidly passivated by oxide layers in oxic water; high aggregation tendency; potential toxicity *via* Fenton reactions	206–209
Metal nanoparticles	Silver nanoparticles (AgNPs)	Size: 10–100 nm; high surface area; surface plasmon resonance	Antimicrobial action *via* Ag^+ ion release and ROS generation; catalytic reduction	Pathogens (bacteria, viruses), dyes, halogenated organics	Excellent disinfectant properties; effective catalyst for reducing certain organic pollutants	High toxicity to non-target aquatic organisms; Ag^+ release is a secondary contamination pathway; efficacy reduced by sulfidation in anoxic waters	132, 133, 210, 211
Metal oxides/hydroxides	Calcium peroxide (*n*-CaO_2)	Size: 50–200 nm; reactive oxygen source; alkaline	Slow release of H_2O_2 for *in situ* chemical oxidation (ISCO); pH modification; precipitation of metals	Petroleum hydrocarbons (BTEX), chlorinated solvents, heavy metals (as phosphates/carbonates)	Provides slow, sustained release of oxidants for groundwater remediation; effective for precipitating metals and fluoride	Can cause uncontrolled, drastic increase in local pH; potential for soil pore clogging; reactivity can be passivated by carbonate formation on the surface	212–214
Mixed metal oxides	Indium tin oxide (ITO)	Size: 20–80 nm; transparent conductive oxide; n-type semiconductor	**Photocatalysis** (UV-A); electrocatalysis	Dyes, phenols, some organic pollutants	Removal: 75–98% Moderate photocatalytic activity; useful as a stable electrode in electrochemical degradation systems	Very high cost of indium limits large-scale application; less efficient photocatalyst than TiO_2 or ZnO; potential for leaching of toxic In^{3+} and Sn^{4+} ions	215, 216
Metal oxides	Zinc oxide (ZnO)	Size: 20–70 nm; surface area: 10-50 $m^2\ g^{-1}$; band gap: ~3.3 eV; PZC: ~9.0	Photocatalysis (UV-A); adsorption; antimicrobial action *via* Zn^{2+} release	Dyes, phenols, pharmaceuticals, pathogens	Removal: 69–95% High photocatalytic efficiency, comparable to TiO_2; effective adsorbent for certain pollutants	Highly soluble under acidic conditions, leading to significant Zn^{2+} leaching and high toxicity ("Trojan Horse" effect); subject to photocorrosion	217–220
Carbon-based	Carbon nanotubes (CNTs)	High aspect ratio (fibrous); high surface area: ~150 to 1587 $m^2\ g^{-1}$; strong sp^2 carbon structure	Adsorption (π–π stacking, hydrophobic); catalyst support; filtration membrane component	PAHs, PCBs, heavy metals (when functionalized), dyes, pharmaceuticals	$Q_{\max}$: 50–350 mg g^{-1} Exceptional adsorption capacity for aromatic organic pollutants; excellent support for catalytic or photocatalytic nanoparticles	Aggregates in hard water; difficult to recover/disperse; potential for physical toxicity due to fibrous shape ("nano-blade" effect)	221–224

PZC: point of zero charge. The pH at which the particle surface has a net neutral charge; efficacy values are highly dependent on experimental conditions (*e.g.*, pH, ENM dose, initial pollutant concentration); NOM: natural organic matter; PAHs: polycyclic aromatic hydrocarbons.

photocatalysts boosts pollutant degradation rates by 3–5-fold, it concurrently amplifies the phototoxicity risk to algae by sustaining high extracellular ROS flux.[36–39] The work of Pigosso *et al.*,[40] for instance, exemplifies a common strategy of band-gap engineering to improve photocatalytic efficiency; by doping ZnO with nitrogen, the band gap was narrowed to 2.89 eV, enabling visible-light absorption and achieving almost complete degradation of tetracycline within 120 minutes.

While such studies demonstrate high efficacy, this performance is typically achieved under highly optimised laboratory conditions. A critical perspective must acknowledge that in real-world aquatic systems, the efficiency of photocatalysis is severely attenuated by light scattering from suspended solids and the absorption of photons by natural organic matter (NOM), which also acts as a potent scavenger of ROS, thereby reducing the quantum yield of pollutant degradation.

Beyond photocatalysis, metal oxides, particularly iron oxides such as magnetite (Fe_3O_4) and maghemite (γ-Fe_2O_3), are effective adsorbents for heavy metals.[8,41,42] Their utility is significantly enhanced by their superparamagnetic nature, which permits facile magnetic separation post-treatment—a crucial advantage for practical implementation. A compelling demonstration is the work of Oladipo *et al.*,[8] who developed a magnetite-based adsorbent with high saturation magnetisation (39.55 emu g^{-1}) and reported impressive removal capacities (72.8–83.4 mg g^{-1}) for various divalent metal cations. However, such high capacities, often determined in single-component aqueous systems, must be interpreted with caution. In environmentally relevant scenarios, the presence of competing cations and complexing ligands in multi-contaminant wastewater invariably leads to a significant reduction in both the rate and extent of target pollutant adsorption due to competitive binding at surface sites.

- **Carbon-based ENMs: broad-spectrum adsorbents**

Carbonaceous nanomaterials, principally carbon nanotubes (CNTs) and graphene-family materials (graphene oxide, GO; reduced graphene oxide, rGO), are defined by their unparalleled specific surface areas and highly accessible graphitised π-electron systems.[12,43–45] Their primary remediation mechanism is physisorption, governed by a combination of non-covalent forces, including dispersive π–π stacking interactions with aromatic contaminants (*e.g.*, PAHs, PCBs) and general van der Waals forces.[46]

The chemical functionalisation of their surfaces, particularly the introduction of oxygen-containing moieties in GO (*e.g.*, carboxyl, hydroxyl groups), imbues them with hydrophilic character and provides sites for electrostatic attraction and hydrogen bonding. This dramatically enhances their affinity for a broader range of pollutants, including heavy metal cations and polar organics.[47,48] A critical challenge for this class of materials, however, lies in their colloidal behaviour and regeneration. Their low density and high aspect ratio can lead to challenging solid–liquid separation, and while their adsorptive capacity is high, effective and economic regeneration remains a significant practical barrier compared to traditional adsorbents like granular activated carbon.

- **Zero-valent metals: potent reducing agents**

Nanoscale zero-valent iron (nZVI) is the most prominent and widely deployed ENM for *in situ* groundwater remediation, acting as a powerful reducing agent.[49,50] Structurally, nZVI particles are characterised by a metallic iron (Fe^0) core encapsulated by a mixed-valence iron oxide/hydroxide shell, which mediates electron transfer to contaminants. With a standard reduction potential (E^0) of −0.44 V, the Fe^0 core can reductively dehalogenate chlorinated organic solvents and reduce mobile, high-oxidation-state metals and metalloids to less toxic, immobile forms.[51,52] A key example is the reduction of hexavalent chromium:

$$2CrO_4^{2-} + 3Fe^0 + 16H^+ \rightarrow 2Cr^{3+} + 3Fe^{2+} + 8H_2O$$

The study by Zhang *et al.*,[49] which integrated nZVI with biochar, highlights a common strategy to mitigate the rapid aggregation and surface passivation that plagues bare nZVI. While the composite showed near-complete Cr(VI) removal, the study also astutely noted the inhibitory effect of co-adsorbed humic acid. This observation underscores the central challenge for nZVI: its high intrinsic reactivity is also its Achilles' heel. The rapid oxidation of the Fe^0 core leads to the growth of the passivating oxide shell, diminishing its reductive power over time.[51] Therefore, a critical assessment of nZVI performance must always consider the trade-off between initial reactivity and long-term operational longevity in complex geochemical environments.

- **Nanocomposites: towards synergistic functionality**

To surmount the intrinsic limitations of single-component ENMs, significant research effort is now directed towards designing multifunctional nanocomposites. For example, decorating carbon nanostructures with TiO_2 (*e.g.*, TiO_2–CNT) acts as an electron sink, promoting charge separation and retarding the recombination of the photogenerated electron–hole pair, thereby increasing the quantum yield.[53]

The work of Joseph *et al.*,[53] which utilised a CNT/TiO_2 nanohybrid for the selective reduction of nitrobenzene, is an elegant illustration of this principle, achieving nearly 100% conversion with excellent selectivity. Similarly, immobilising nZVI on supports like bentonite or biochar improves its dispersion and stability.[49] While these composite materials represent a clear advancement in rational design for enhanced efficacy, they concurrently introduce greater complexity in predicting their environmental fate.

2.2. Critical evaluation of remediation performance

While the operational principles of remediation ENMs are well-established in theory, a critical appraisal of their reported performance reveals a significant disjunction between laboratory potential and real-world applicability.

- **The lab-to-field disparity: contextualising performance metrics**

The presence of dissolved organic matter (DOM) introduces a formidable variable. The predominantly reported effect of DOM is antagonistic, primarily through competitive adsorption (blocking active sites) and scavenging of therapeutic oxidants.[54] The work of Awfa *et al.*[55] provides a clear quantitative illustration of this inhibition, where the presence of NOM reduced the apparent photodegradation rate constant (k_{app}) for pharmaceuticals by nearly an order of magnitude.

Conversely, Dong *et al.*[56] observed that certain fractions of DOM promoted photocatalytic degradation *via* photosensitisation. The resolution of this apparent contradiction lies in the specific molecular composition of the DOM and the surface chemistry of the ENM.[54,55,57] Therefore, any performance claim that does not account for the specific nature and concentration of background organic matter must be considered environmentally irrelevant.

- **The selectivity dilemma: balancing efficacy against ecological perturbation**

The reactivity of remediation ENMs exists on a spectrum that presents a fundamental design dilemma. On one end, broad-spectrum agents such as photocatalytic TiO_2 or Fenton-based nZVI systems generate highly reactive, non-selective species like the hydroxyl radical. This is advantageous for mineralising complex mixtures of organic contaminants.[58,59] The critical drawback, however, is that this indiscriminate reactivity does not distinguish between a pollutant molecule and a non-target biological structure. A quantitative illustration of this dilemma is observed in comparative toxicity studies: ENMs engineered for maximal ROS yield (*e.g.*, defective TiO_2) often exhibit LC_{50} values for sensitive species like *Daphnia magna* that are orders of magnitude lower ($1\ \mathrm{mgL^{-1}}$) than their less reactive counterparts, creating a direct correlation between remediation potential and ecological hazard.[60,61]

On the other end of the spectrum, ENM surfaces can be functionalised with specific ligands or imprinted polymers to achieve high selectivity for a single contaminant, such as Hg^{2+} or Pb^{2+}.[62–64] While effective, its utility is limited in multi-contaminant mixtures. The central challenge for the field is to move towards materials with tuneable reactivity—activated only under specific conditions—thus optimising the trade-off between remedial power and ecological safety.

- **Material integrity and longevity**

For any nanoremediation technology to be economically viable, long-term stability is paramount. In realistic continuous-flow systems, ENM efficacy is subject to gradual decline through surface fouling and chemical passivation (*e.g.*, oxidation of the Fe^0 core in nZVI).[65,66] Furthermore, the dissolution of metal-based ENMs constitutes a secondary contamination pathway. The release of toxic constituent ions (*e.g.*, Zn^{2+} from ZnO, Ag^+ from AgNPs) into treated water effectively replaces one contamination problem with another.[67,68] A critical evaluation must therefore adopt a lifecycle perspective, assessing not only the initial efficacy but also the material's robustness and its "end-of-life" fate.

3. Environmental fate and transformation: the bridge between application and exposure

The remediation performance detailed in the preceding section represents the ENM's function at the point of application—a transient and idealised state. Once released into a complex aquatic milieu, an ENM is no longer a static entity but a labile one, subject to a suite of transport phenomena and physicochemical transformations that collectively govern its environmental fate and speciation. The ecotoxicological risk posed by a remediation ENM is therefore not a function of its pristine, as-synthesised properties, but is instead determined by the dynamically evolving characteristics of the "**environmentally aged**" **nanoparticle**—defined here as a particle that has undergone significant physicochemical alteration (*e.g.*, surface coating, aggregation, partial dissolution) relative to its pristine state.

This process of transformation, conceptualised in Fig. 2, alters fundamental properties including effective particle size, surface charge, chemical composition, and dissolution kinetics, which in turn dictate the material's transport, bioavailability, and hazard potential.[69–71] Critical transformations include aggregation and agglomeration with natural colloids (heteroaggregation), dissolution and reprecipitation, surface oxidation or reduction, and the formation of an adsorbed layer of natural organic matter, termed the "eco-corona". The eco-corona is defined as a dynamic, environmentally acquired coating of natural organic matter (NOM), proteins, and other biomolecules that rapidly adsorbs to the nanoparticle surface upon contact with aquatic media, fundamentally altering its biological identity.

A predictive understanding of nanotoxicity is therefore impossible without first critically evaluating the environmental processes that bridge the gap between initial application and eventual biological exposure. This section deconstructs this critical pathway.

3.1. Release scenarios: defining the initial boundary conditions

The initial concentration, chemical form, and spatial distribution of ENMs in an aquatic system are dictated by the mode of application, which establishes the "initial boundary conditions" for all subsequent fate and transport processes. A primary and critical distinction must be drawn between direct and indirect release pathways.

- **Direct application (*in situ* remediation):** this pathway involves the intentional, large-scale introduction of ENMs directly into a contaminated environment. The archetypal example is the injection of concentrated nanoscale zero-valent iron (nZVI) slurries (often in the $\mathrm{g\ L^{-1}}$ range) into groundwater plumes to form permeable reactive barriers.[67,70] This scenario creates a localised, high-concentration "hotspot" where the fate of the relatively pristine, as-synthesised ENMs is initially dominated by the immediate,

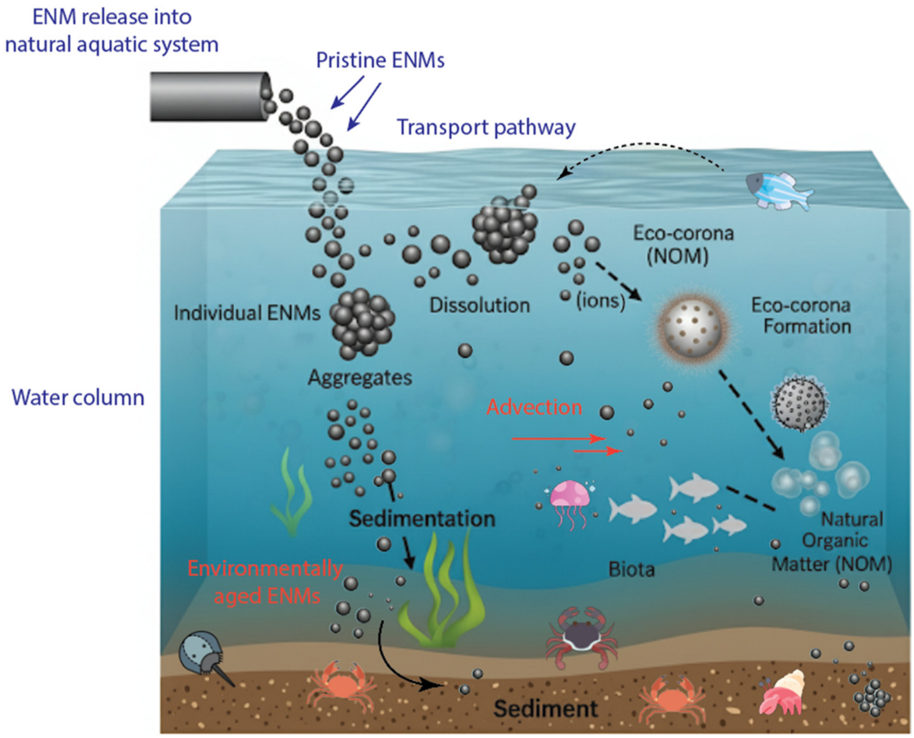

Fig. 2 Conceptual diagram illustrating the environmental fate and transformation cascade of a remediation ENM following its release into a natural aquatic system. The journey begins with the pristine ENM, which undergoes critical physicochemical transformations within the water column, including **aggregation** (homo- and heteroaggregation), **dissolution** (ion release), and the formation of an **eco-corona** *via* adsorption of natural organic matter (NOM). These transformations, occurring during transport *via* advection, dictate the ENM's ultimate environmental speciation, its interactions with biota, and its potential for deposition into the sediment compartment *via* sedimentation.

and often aggressive, geochemistry of the subsurface environment.

- **Indirect release (*ex situ* treatment effluents):** this pathway involves the discharge of ENMs from centralised water or wastewater treatment facilities where they are used as part of the purification process. In this scenario, the ENMs are extensively modified *before* they even enter the receiving aquatic environment. Within a treatment plant, they interact intimately with the microbial communities and high organic load of the activated sludge, leading to significant aggregation and the formation of a mature eco-corona.[67,69–73] Consequently, the material released *via* the final effluent is not the pristine ENM, but rather a "**pre-aged**" **nanocomposite** whose environmental behaviour is substantially different from its as-synthesised counterpart.

- **Chronic leaching from immobilised systems:** a third, distinct pathway involves the slow, chronic release of ENMs that are immobilised within a fixed-bed reactor or integrated into a filtration membrane. Over extended operational periods, mechanical abrasion and chemical degradation can lead to the gradual shedding or leaching of the nanomaterials.[74,75] This results in a long-term, low-concentration release of potentially fragmented or weathered ENMs, representing a chronic rather than acute source of environmental exposure.

A critical methodological gap exists here: the overwhelming majority of laboratory-based fate and ecotoxicology studies use pristine ENMs dispersed in simplified media as their model system. This approach may approximate the direct application scenario but poorly represents the pre-aged state of materials from indirect release pathways, which are arguably the more widespread source of environmental loading. Recognising the distinct initial state of the ENM as defined by its release pathway is therefore a prerequisite for designing environmentally relevant experiments and developing accurate exposure models.

3.2. Key physico-chemical transformations in water

Upon introduction into any natural aquatic system, an ENM is immediately subjected to a suite of interconnected transformation processes that fundamentally and dynamically alter its physical and chemical identity. These processes, dictated by both the ENM's intrinsic properties and the local water chemistry (*e.g.*, pH, ionic strength, redox potential, DOM composition), govern the material's ultimate environmental speciation and toxicological profile.

- **Aggregation and sedimentation: altering transport and bioavailability**

Due to their high surface energy-to-volume ratio, nanoparticles possess a strong thermodynamic driving force to aggregate in aqueous suspension to minimise the interfacial free energy. This process occurs *via* homoaggregation (ENM–ENM interaction) or, more commonly in natural waters, heteroaggregation with suspended natural colloids like clays and cellular debris.[76–79] While the colloidal stability of ENMs is classically described by

DLVO theory (balancing van der Waals attraction V_A and electrostatic repulsion V_R), environmental systems are far from ideal.[80] In high ionic strength waters (*e.g.*, seawater >500 mM), the electrical double layer is compressed, leading to diffusion-limited aggregation regimes with rate constants (k_{agg}) approaching the Smoluchowski limit ($\sim 10^{-17}$ m^3 s^{-1}).[79,81]

The kinetics of aggregation are critically dependent on water chemistry. Recent quantitative data indicate that this aggregation can be rapid; for example, the hydrodynamic diameter of TiO_2 nanoparticles can increase from ~25 nm to >1 μm within 30 minutes in hard water, drastically increasing their sedimentation rates.[82,83] The study by Bélteky *et al.*[84] critically demonstrates that this environmental fate is pre-conditioned by the synthetic route; silver nanoparticles stabilised with green tea extract (AgNP@GT) exhibited greater colloidal stability across different pH and electrolyte conditions than those capped with citrate (AgNP@C). This highlights a crucial insight: the "environmental fate" of an ENM begins in the synthesis flask.

- **Dissolution and ion release: the 'Trojan Horse' pathway**

For ENMs composed of non-inert materials (*e.g.*, ZnO, CuO, AgNPs), dissolution is a paramount transformation pathway. It releases constituent metal ions into the aqueous phase, creating a mixed exposure scenario.[85]

The dissolution rate (dC/dt) is governed by the Noyes–Whitney framework but is critically modulated by environmental factors.[86] Kinetic data show that dissolution half-lives ($t_{1/2}$) vary by orders of magnitude: ZnO nanoparticles may exhibit a $t_{1/2}$ of mere hours in acidic waters (pH 5) *versus* weeks in neutral, NOM-rich waters where surface passivation occurs.[87,88] This pathway is the mechanistic basis of the "Trojan Horse" hypothesis.[71–73,75]

- **Surface transformations and the eco-corona: defining the bio-nano interface**

The surface of an ENM is its active interface with the environment. Upon immersion, a dynamic "eco-corona" of adsorbed DOM rapidly forms.[54,57] This corona evolves *via* the Vroman effect, where high-affinity molecules displace lower-affinity ones.[89] This layer profoundly alters the bio-nano interface; for instance, a humic acid corona can increase the negative zeta potential of an ENM to <−20 mV, enhancing colloidal stability *via* electrosteric repulsion, while simultaneously masking surface defects.[84,90,91]

Surface functionalisation critically defines the biological outcome. The work by Shivaji *et al.*[91] demonstrates that bio-functionalising CdS quantum dots with a biocompatible shell successfully hindered the leaching of toxic Cd^{2+} ions. Beyond corona formation, direct chemical transformations like sulfidation (*e.g.*, $2Ag^+ + HS^- \rightarrow Ag_2S + H^+$) can effectively detoxify ENMs by locking toxic ions into insoluble mineral phases, reducing acute toxicity by up to 99% compared to the pristine ionic form.[71,92–94]

3.3. Impact of transformations on bioavailability and reactivity

Environmental transformations are not mere modifications; they represent a critical **decoupling mechanism** that separates the initial properties of an ENM from its ultimate biological effect. The pristine, as-synthesised nanoparticle is largely a toxicological abstraction; in any real environmental scenario, organisms are exposed to an aged, transformed entity whose bioavailability and intrinsic chemical reactivity differ profoundly from its nascent state.[95]

Aggregation and sedimentation directly re-route the primary vector of ecological exposure. By increasing the effective particle size, aggregation reduces bioavailability to pelagic organisms like phytoplankton but concentrates the ENM payload in benthic ecosystems, increasing exposure for deposit-feeders.[96] Perhaps the most profound modulator is the eco-corona. This layer acts as the ENM's "biochemical passport", governing cellular uptake.[97–99] A dense corona can sterically hinder direct contact, mitigating membrane disruption.[97] Conversely, it can facilitate uptake *via* receptor-mediated endocytosis if specific biomolecules are recognised.[71,73] Crucially, these transformations also redefine chemical reactivity. The passivation of nZVI's reactive Fe^0 core by an oxide shell diminishes its pollutant degradation capacity and its potential to induce reductive stress.[100]

Similarly, the work of Yang *et al.*[93] illustrates the complexity: algal EPS promoted Ag^+ release (toxifying) but also facilitated sulfidation to Ag_2S (detoxifying). This reveals that the net toxicological outcome is determined by the kinetic balance between competing pathways.

3.4. Critical assessment of fate modelling

Predictive environmental fate models are indispensable tools in a modern regulatory context, providing the predicted environmental concentrations (PECs) required for quantitative risk assessment.[101–104] However, a critical assessment reveals significant uncertainty propagation across fate models, as summarised in Table 2.

For ENMs, a suite of models (*e.g.*, MendNano, nanoDUFLOW, SimpleBox4Nano) has been developed, typically by adapting existing multimedia frameworks for conventional chemicals and incorporating nano-specific modules for processes like aggregation, dissolution, and sedimentation.[104–106] While these models represent a crucial first step, a critical assessment reveals a significant gap between their conceptual architecture and the environmental complexity they purport to describe, leading to profound uncertainty in exposure prediction. A primary limitation is the oversimplification of foundational theories (*e.g.*, Smoluchowski coagulation) for non-ideal environmental systems.[107] These theories fail to account for the complex fractal geometries of real aggregates, the dominant role of heteroaggregation with chemically diverse natural colloids, or the steric and hydration forces imparted by a mature eco-corona, leading to high structural uncertainty in the models.[104,105,108,109]

Even if model structures were perfect, they are constrained by a parameterization impasse. Input parameters like Hamaker constants are difficult to measure for corona-coated ENMs.[110] The most significant issue is the validation gap: most models are calibrated with laboratory data and lack

Table 2 Comparison of key multimedia environmental fate models for ENMs

Model feature	MendNano	SimpleBox4Nano	WASP8
Core approach	Dynamic, mechanistic mass balance	Steady-state, multimedia box model	Dynamic, compartmental water quality model
Key processes	Aggregation, dissolution, sedimentation	Aggregation, attachment, dissolution	Transport, transformation, benthic flux
Spatial resolution	Flexible (compartmental)	Regional/continental scale	High resolution (water body segments)
Limitations	High data requirement for kinetic parameters	Assumes instantaneous equilibrium (steady-state)	Complex parameterisation for specific ENMs
Best use case	Dynamic systems with variable release	Screening-level regional risk assessment	Site-specific water quality management

robust validation against field measurements, leading to PEC estimates that can span orders of magnitude.[111–113] This uncertainty remains a primary impediment to the responsible governance of nanoremediation.

3.5. Analytical hurdles: the challenge of detecting and characterising transformed ENMs

The preceding critique is underpinned by the immense difficulty in detecting and characterising ENMs *in situ*. The challenge is distinguishing trace ENMs from natural nanoparticles (NNPs) and characterising their speciation. For quantitative detection, single-particle ICP-MS (spICP-MS) is state-of-the-art, distinguishing dissolved and particulate fractions.[114,115] However, it cannot easily distinguish engineered from natural particles of the same element. To address speciation, multi-method approaches are required (Table 3). Techniques like field-flow fractionation (FFF) offer separation by size, while TEM/SEM provide visual confirmation but introduce preparation artefacts.[116,117] Synchrotron-based XAS (XANES/EXAFS) is the gold standard for chemical speciation (*e.g.*, distinguishing Fe^0 from Fe-oxides), but is limited by accessibility and sensitivity.[118,119] In summary, the analytical barrier is the primary reason for the validation gap in fate modelling and remains a formidable obstacle to tracking the transformation of ENMs *in vivo*.

4. The “Risk” edge: mechanistic nanotoxicity in aquatic systems

Once an environmentally transformed ENM makes contact with a biological receptor, the “risk” dimension of the benefit–risk paradigm is realised. The ensuing toxicity is not a monolithic endpoint but a dynamic pathological cascade, originating from molecular-level perturbations that can propagate through successive levels of biological organisation to manifest as adverse outcomes for the whole organism and population. Crucially, the bioavailability of the ENM at this interface is directly dictated by the environmental fate processes described in section 3 (*e.g.*, aggregation state, corona composition), creating a continuum from release to exposure. A mechanistic understanding is therefore paramount, as it moves beyond a descriptive, black-box approach towards a predictive toxicology. This section dissects the critical stages in the nanotoxicity cascade, adopting the OECD adverse outcome pathway (AOP) framework to link molecular initiating events (MIEs) through key events (KEs) to adverse outcomes (AOs).[120] The primary mechanistic pathways are conceptualised in Fig. 3 and representative toxicological endpoints are synthesised in Table 4.

4.1. Crossing the threshold: uptake, translocation, and biodistribution

For an ENM to exert systemic or internal toxicity, it must first traverse a biological barrier. The efficiency of this process—governing the internal dose and subsequent biodistribution—is dictated by the complex interplay at the **bio-nano interface**: the interaction between the environmentally-conditioned ENM (with its eco-corona) and the physiological surfaces of the organism.[121]

In primary producers like microalgae, the initial barrier is the cell wall. Lacking active phagocytic mechanisms, ENM interaction is dominated by adsorption to this external surface. This alone can induce physical stress, block light and nutrient transport, and cause direct membrane damage through localised oxidative phenomena.[85,121–124] While the cell wall's porous structure (typically 5–20 nm) may permit the passive entry of very small ENMs (<10 nm), its primary role appears to be protective.[125,126] This is central to the ongoing debate regarding the ultimate toxic agent for soluble ENMs like AgNPs. Studies using cell wall-free mutants, such as those by Piccapietra *et al.*,[127] clearly demonstrate that the wall limits the uptake of dissolved Ag^+. This suggests that a major toxicity vector is the dissolution of ENMs in the immediate vicinity of the cell, creating a high local concentration of toxic ions that then diffuse inwards, rather than the wholesale internalisation of the particles themselves.[126,127] Definitive proof of widespread particle translocation across the algal cell membrane remains analytically challenging.

For invertebrates such as the filter-feeding crustacean *Daphnia magna*, the principal uptake route is ingestion, leading to direct exposure of the gut epithelium.[126,128] Systemic translocation from the gut is generally inefficient, hindered by the peritrophic membrane and the gut lining, but is highly dependent on the ENM's properties. The critical role of the surface coating was elegantly demonstrated by Yan and Wang,[126] who showed that citrate- and fluorogen-coated AgNPs were internalised by algal cells *via* different pathways (apical zone *vs.* endocytosis). This finding is

Table 3 Critical comparison of advanced analytical techniques for ENM characterisation in aquatic media

Technique	Principle of operation	Information provided	Strengths	Critical limitations in environmental matrices
spICP-MS (single-particle inductively coupled plasma-mass spectrometry)	Individual nanoparticles are ionised in plasma; a mass spectrometer detects the resultant ion bursts to count and quantify particles elementally	Particle number concentration; mass-equivalent size distribution; dissolved *vs.* particulate fractions	**Highly sensitive** (ng L^{-1} detection); quantitative; high throughput; distinguishes particles from dissolved ions	**Blind to particle aggregation state and shape.** Cannot distinguish between engineered and natural nanoparticles (NNPs) of the same element. Lower size detection limit (~10–20 nm). Not applicable to carbon-based ENMs
FFF-MALS/DLS/ICP-MS (field-flow fractionation coupled to detectors)	A gentle, liquid-phase separation technique that sorts particles based on hydrodynamic size in a thin channel, without a stationary phase	High-resolution hydrodynamic size distribution; aggregation state; particle shape information (MALS); elemental composition (ICP-MS)	Excellent for characterising **polydisperse and complex samples.** Preserves aggregation state much better than batch methods. Can be hyphenated with multiple detectors for rich data	**Technically complex to operate**. Separation can be influenced by particle-membrane interactions, leading to artefacts. Lower throughput and less sensitive than spICP-MS for trace quantification
TEM/SEM–EDX (transmission/scanning Electron microscopy with energy dispersive X-ray)	A high-energy electron beam scans a prepared sample to generate a high-magnification image. EDX provides localised elemental analysis	Direct visualisation of particle **morphology, primary size, and structure**. Elemental mapping of individual particles and aggregates	**Unmatched spatial resolution**. Provides direct, unambiguous visual evidence of particle form and association with biological or mineral matrices	**Not an *in situ* technique**. Sample preparation (drying, fixing, vacuum) introduces **severe artefacts**, destroying the hydrated eco-corona and altering the aggregation state. Poor statistical representation of the bulk sample
DLS (dynamic light scattering)	Measures time-dependent fluctuations in scattered light intensity from particles undergoing Brownian motion to calculate hydrodynamic size	*Z*-average hydrodynamic diameter; polydispersity index (PDI)	**Rapid, non-invasive, and widely accessible** for routine analysis of colloidal stability in simple systems	**Highly unreliable for environmentally relevant, polydisperse samples.** The signal is overwhelmingly biased by larger particles (intensity $\propto r^6$). Provides no information on concentration or chemical composition
XAS (X-ray absorption spectroscopy; including XANES & EXAFS)	A tunable synchrotron X-ray beam excites core electrons; the absorption spectrum reveals the element's local atomic and chemical environment	**Elemental oxidation state**; coordination environment (*e.g.*, oxide *vs.* sulfide); nearest-neighbour identity and bond distances	The **gold standard for chemical speciation**. Can definitively identify chemical transformations *in situ* in wet, frozen, or solid samples	Requires access to a **synchrotron radiation facility**. Relatively high concentrations are needed for a good signal. Provides bulk-averaged information, not data on individual particle heterogeneity

profound, as it shows that the surface chemistry does not merely alter the *quantity* of uptake, but the fundamental *biological mechanism* by which it occurs.

This leads to the environmentally critical pathway of **trophic transfer.** The bioaccumulation of ENMs is dominated by dietary exposure for many invertebrates. As Zhao and Wang[128] estimated, over 70% of silver accumulated by *Daphnia magna* can originate from their algal diet. This process transfers not just the ENM, but a complex cocktail of the aged particle, its corona, and any associated ions. As Lekamge *et al.*[124] demonstrated, the surface coating of the AgNPs dictated their initial accumulation in algae and, consequently, their subsequent diet-borne transfer efficiency to daphnids, underscoring that surface properties can have cascading effects up the food web.

In vertebrates like fish, multiple exposure routes exist. The gills represent a vast, delicate surface area providing a direct pathway to the systemic circulation. Ingestion leads to gut exposure, and dermal contact provides a minor route.[122,123] Once internalised, ENMs are typically distributed *via* the circulatory system, with evidence showing rapid translocation and accumulation in key filtering and detoxification organs analogous to the reticuloendothelial system, such as the liver, kidney, and spleen.[123,124] The work by Rességuier *et al.*[123] using polymer-based nanoparticles (PLA-NPs) in zebrafish larvae vividly illustrated this, showing efficient internalisation by endothelial cells and macrophages and widespread vascular distribution within an hour. This rapid biodistribution establishes the landscape of target organs where the

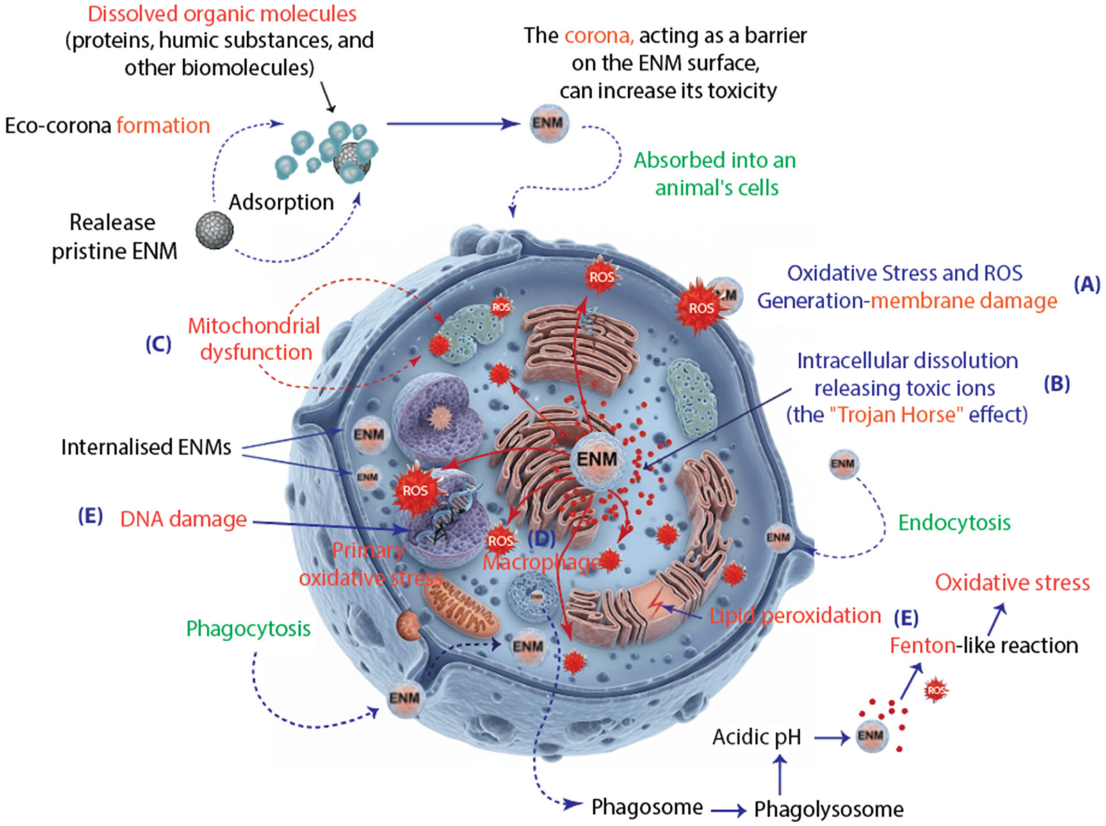

Fig. 3 Cellular-level schematic of the primary mechanistic pathways of nanotoxicity following the uptake of an ENM by an aquatic organism's cell. The cascade is initiated by cellular internalisation, leading to multiple, interconnected molecular initiating events. These include: (A) direct physical or chemical damage to the cell membrane; (B) intracellular dissolution releasing toxic ions (the "Trojan Horse" effect); (C) disruption of mitochondrial function; (D) generation of reactive oxygen species (ROS) leading to widespread oxidative stress, such as lipid peroxidation and (E) oxidative DNA damage (genotoxicity) within the nucleus.

molecular and cellular perturbations that constitute the core of mechanistic nanotoxicity will subsequently unfold. Table 5 summarizes the distinct uptake routes across key trophic levels.

4.2. Core mechanistic toxicity pathways – a critical synthesis

Following uptake and translocation, the interaction of ENMs with the subcellular environment initiates a complex and interconnected network of pathological events.[129] While the specific outcomes are diverse, the underlying molecular initiating events often converge upon a few core mechanistic pathways. The central thesis of mechanistic nanotoxicity is that an ENM's physicochemical properties (*e.g.*, surface reactivity, dissolution kinetics, crystalline defects, redox potential) directly govern which of these pathways are triggered. A critical synthesis of these mechanisms reveals that they are not discrete events but a web of cause-and-effect, in which oxidative stress functions as the central *convergence hub*, radiating damage to lipids, proteins, and nucleic acids.

4.2.1. Oxidative stress: the central hub of nanotoxicity. Oxidative stress, broadly defined as the disequilibrium between pro-oxidant flux and antioxidant defenses, is arguably the most pervasive and consequential driver of ENM-induced toxicity. The imbalance manifests in uncontrolled oxidation of biomacromolecules and collapse of redox homeostasis, propagating cytotoxicity and tissue dysfunction.[130,131] Quantitative thresholds for this effect are often material-specific; for instance, TiO_2 nanoparticles typically trigger measurable intracellular ROS increases at concentrations >1 mgL^{-1} in zebrafish embryos, whereas highly soluble AgNPs can induce oxidative stress at concentrations as low as 10 $\mu g\ L^{-1}$.[132,133]

Two mechanistic routes underlie this process:

A. Direct ROS generation at the nano-nio interface (primary oxidative stress)

This route exemplifies the "double-edged sword" of nanotechnology: the very reactivity that makes ENMs attractive for catalysis, remediation, and antimicrobial action is transposed into toxicological liability upon biological exposure.

- **Photocatalytic ENMs (TiO_2, ZnO)**

In photic zones, internalised or membrane-associated photocatalysts can absorb photons, generating electron–hole pairs (e^-/h^+) that react with water and oxygen to produce ROS. The non-selective and highly damaging hydroxyl radical ($^{\bullet}OH$) is a key product.[57] While studies on cultured cells

Table 4 Mechanistic toxicity endpoints of remediation ENMs linking to the primary ENM properties

ENM class	Example ENM	Model organism(s)	Primary toxicity mechanism(s)	Key mechanistic endpoint(s)	Modulating factors	Ref.
Metal oxides	Anatase TiO_2	*R. subcapitata* (algae); *D. magna* (crustacean); *D. rerio* (zebrafish)	**Oxidative stress** (phototoxicity)	Increased intracellular ROS; lipid peroxidation (MDA levels);[a] upregulation of antioxidant enzyme genes (*e.g.*, *cat*, *sod*); DNA damage (8-OHdG lesions)	**UV irradiation is required.** Toxicity is mitigated by NOM corona formation, which can quench ROS or block surface sites	225–227
Metal oxides	ZnO	*D. rerio* (zebrafish)	**Oxidative stress** Physical effects	Increased ROS production; induction of stress-related gene expression (*e.g.*, *hsp70*); evidence of gut blockage and gill adhesion at high concentrations	Toxicity is generally lower than for other ENMs. Aggregation state is a key determinant of physical effects	228, 229
Carbon-based	Graphene oxide (GO)	Bacteria (*e.g.*, *E. coli*); algae	**Membrane disruption** Oxidative stress	Loss of membrane integrity (*e.g.*, LDH leakage assay);[b] physical wrapping/envelopment of cells; depletion of intracellular glutathione (GSH)	Toxicity is strongly dependent on sheet size and sharpness of edges. NOM corona can passivate sharp edges and reduce direct membrane contact	230, 231
Zero-valent metals	nZVI	*D. magna*; *D. rerio*; *Chlorella pyrenoidosa*; *Paraprionospio patiens*	**Oxidative stress** (Fenton-like reactions)	Severe lipid peroxidation; significant upregulation of oxidative stress response genes; mitochondrial membrane depolarisation	**Oxygen is required.** Toxicity is driven by corrosion products (Fe^{2+}) and is highest for bare, un-coated nZVI. Surface coatings and NOM can reduce corrosion and mitigate toxicity	232–234
Metal nanoparticles	Silver nanoparticles (AgNPs)	*D. magna* (crustacean); *Danio rerio* (zebrafish); *R. subcapitata* (algae)	**Ion release** (trojan horse); oxidative stress	Inhibition of key enzymes *via* Ag^+ binding to thiol groups (*e.g.*, Na^+/K^+-ATPase); increased intracellular ROS; lipid peroxidation (MDA levels); upregulation of metallothionein genes	**Water chemistry is critical.** Toxicity is drastically reduced by sulfidation (formation of insoluble Ag_2S). Presence of chloride can precipitate AgCl. NOM corona can reduce ion dissolution rate and direct particle contact	235–237
Carbon-based	Carbon nanotubes (CNTs/MWCNTs)	*D. magna* (crustacean); *Danio rerio* (zebrafish); algae	Physical/mechanical damage; oxidative stress; inflammation	Physical abrasion of gut epithelium and gills; impairment of filter-feeding; induction of inflammatory cytokine genes (*e.g.*, *IL-1β*, *TNF-α*); ROS generation leading to oxidative DNA damage (8-OHdG)	**Physicochemical properties are dominant.** Toxicity is highly dependent on length, rigidity, and aspect ratio ("fibre-type toxicity"). Aggregation state significantly alters bioavailability and physical effects. NOM corona can passivate surfaces	238–241

Notes: [a] MDA: malondialdehyde, a common biomarker for lipid peroxidation. [b] LDH: lactate dehydrogenase, a cytosolic enzyme that is released upon loss of membrane integrity. Endpoints are representative and not exhaustive. The magnitude of the effect is dose- and time-dependent.

Table 5 Comparative uptake routes and translocation potential of ENMs across aquatic trophic levels

Trophic level	Model organism	Primary uptake route	Translocation potential	Key barrier
Primary producers	Microalgae (*R. subcapitata*)	Adsorption to cell wall; pore diffusion (<10 nm)	Low (limited by cell wall)	Cell wall (cellulose/pectin)
Primary consumers	Zooplankton (*D. magna*)	Ingestion (filter-feeding)	Moderate (gut → hemolymph)	Peritrophic membrane
Secondary consumers	Fish (*Danio rerio*)	Gills (direct); ingestion; dermal	High (systemic circulation)	Gill epithelium; gut lining

demonstrate the potent cytotoxicity of photoactivated TiO_2,[134–137] the environmental relevance for internalised particles in aquatic organisms (away from direct UV exposure) is likely limited.

- **Redox-active ENMs (nZVI, CuO, CeO_2)**

Unlike photocatalysts, these ENMs generate ROS through sustained redox cycling in the absence of external irradiation. Their catalytic involvement in Fenton-like and Haber–Weiss chemistry continuously converts relatively benign metabolic by-products ($O_2^{\bullet-}$, H_2O_2) into $^{\bullet}OH$. The archetypal Fe-driven cycle illustrates this process:[138–140]

$$Fe^0 + 2H^+ \rightarrow Fe^{2+} + H_2 \quad \text{(Corrosion)}$$

$$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^{\bullet} + OH^- \quad \text{(Fenton Reaction)}$$

$$O_2^{\bullet-} + Fe^{3+} \rightarrow O_2 + Fe^{2+} \quad \text{(Haber–Weiss Cycle)}$$

Here, the regenerative cycling between Fe^{2+} and Fe^{3+} sustains ROS production with catalytic efficiency. Importantly, the same principle extends to other redox-active ENMs. CuO nanoparticles, for example, can shuttle between Cu^{2+}/Cu^+, driving analogous redox cascades. A critical study by Moschini *et al.*[130] revealed that the density of crystalline defects in CuO ENMs directly amplified intrinsic ROS generation and cytotoxicity—concretely linking atomic-scale structure to toxicological potency. In contrast, CeO_2 exhibits dualistic behavior:[141] depending on its Ce^{3+}/Ce^{4+} ratio and local environment, it may function either as a pro-oxidant (*via* cycling and ·OH production) or as a radical scavenger, underscoring the nuanced, context-dependent toxicological outcomes of redox-active ENMs.

B. Disruption of cellular redox homeostasis (secondary oxidative stress)

Independent of direct surface catalysis, ENMs perturb intracellular redox equilibrium by interfering with mitochondrial function. Perturbations to the electron transport chain (ETC)—either through direct ENM interaction or *via* released ions—facilitate premature electron leakage to molecular oxygen, yielding superoxide. The resultant ROS cascade follows canonical enzymatic processing:

$$2O_2^{\bullet-} + 2H^+ \overset{SOD}{\rightarrow} H_2O_2 + O_2$$

$$2H_2O_2 \overset{CAT}{\rightarrow} 2H_2O + O_2$$

When antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT) are overwhelmed, and non-enzymatic reserves like glutathione (GSH) are depleted, redox collapse ensues. This exacerbates cellular vulnerability, converting localized oxidative stress into a global cytotoxic program.

- **Damage to cellular structures and macromolecules**

The uncontrolled production of ROS and direct physical interactions with ENMs lead to widespread structural and functional damage.

○ **Membrane disruption and lipid peroxidation:** the cell membrane is a primary target. Physical damage can occur from sharp, acicular ENMs like CNTs puncturing the bilayer.[142,143] More insidiously, ROS can initiate a destructive radical chain reaction of lipid peroxidation, compromising membrane integrity. This proceeds *via* three stages:

i. Initiation: $LH + {}^{\bullet}OH \rightarrow L^{\bullet} + H_2O$ (where LH is a lipid).

ii. Propagation: $L^{\bullet} + O_2 \rightarrow LOO^{\bullet}$; then $LOO^{\bullet} + LH \rightarrow LOOH + L^{\bullet}$.

iii. Termination: formation of non-radical products, including cytotoxic aldehydes like malondialdehyde (MDA).

○ **Genotoxicity and DNA damage:** damage to genetic material is a severe endpoint. Indirect genotoxicity is the most common route, where $^{\bullet}OH$ attacks DNA bases. A key lesion is the formation of 8-hydroxy-2′-deoxyguanosine (8-OHdG), a widely used biomarker for oxidative DNA damage.[144–146] Direct genotoxicity involves the translocation of small ENMs into the nucleus, where they can physically interfere with DNA replication or bind to the phosphate backbone, though definitive evidence for this in aquatic species remains sparse.[145]

○ **Protein corona-pathology and enzyme inhibition:** upon internalisation, the formation of a protein "bio-corona" is immediate. This interaction can be pathological; adsorption onto a high-curvature surface can induce protein unfolding and aggregation, leading to loss of function.[147–149] Furthermore, released ions can directly inhibit enzyme activity. The potent toxicity of silver ions (Ag^+), for instance, is largely due to their high affinity for thiol groups (–SH) in cysteine residues, which are often critical components of enzyme active sites:[150]

$$\text{Enzyme–SH} + Ag^+ \rightarrow \text{Enzyme–S–Ag} + H^+ \quad \text{(Irreversible Inhibition)}$$

- **The trojan horse effect: deconvoluting particle *vs.* ion toxicity**

For soluble ENMs (ZnO, CuO, AgNPs), a central mechanistic question is the relative contribution of the particle itself *versus* its dissolved ions. The "Trojan Horse" hypothesis posits that the nanoparticle form facilitates uptake, subsequently dissolving within the acidic confines of organelles like lysosomes to deliver a highly concentrated dose of toxic ions.[136,151] The pH-dependent dissolution of ZnO is a classic example:

$$ZnO_{(s)} + 2H^+_{(aq)} \rightarrow Zn^{2+}_{(aq)} + H_2O_{(l)}$$

While this mechanism is compelling and supported by significant evidence,[152] a critical perspective acknowledges the immense difficulty in experimentally deconvoluting these two toxicity vectors. The observed effects are almost always a combination of direct particle interactions at the surface and the toxicological consequences of the released ions.

- **Higher-order responses: inflammation and immunotoxicity**

In multicellular organisms, cellular stress escalates to inflammation. Mechanistically, this is driven by the upregulation of specific pro-inflammatory cytokines such as IL-1β, IL-6 and TNF-α. Chronic expression of these markers serves as a key event linking initial exposure to tissue-level fibrosis and organ failure. In fish, phagocytic immune cells like macrophages recognise ENMs as foreign invaders and attempt to clear them, a process that can trigger the release of pro-inflammatory cytokines and further ROS production (a "respiratory burst").[122,123,137,141] While this is a protective response, chronic, unresolved inflammation, or "frustrated phagocytosis" by fibrous ENMs, can lead to tissue damage and fibrosis. As shown by Pei *et al.*,[131] ZnO NPs can activate the NLRP3 inflammasome in hepatocytes, leading to pyroptotic cell death and the release of inflammatory mediators, demonstrating a clear link between an ENM, a specific molecular pathway, and organ-level injury.

C. Emerging mechanisms: while ROS generation is well-established, recent research highlights more subtle, sub-lethal mechanisms:

- **Autophagy modulation:** ENMs like graphene oxide (GO) can induce "autophagic flux blockade," where the accumulation of autophagosomes leads to cell death even in the absence of massive ROS. This aligns with the findings of the Hu and Zhou on graphene risks, which identified physical sequestration and membrane stress as key drivers of cytotoxicity independent of oxidative potential.[153]
- **Mitochondrial dynamics:** beyond simple dysfunction, ENMs can disrupt mitochondrial fission/fusion dynamics, impairing cellular energy metabolism long before cell death occurs.
- **Epigenetic alterations:** chronic exposure to sub-lethal doses of ENMs (*e.g.*, CuO) has been linked to DNA methylation changes, potentially affecting gene expression in future generations.[154]

D. Reproductive toxicity: recent evidence underscores the sensitivity of reproductive organs. For example, exposure to PbSe nanoparticles has been shown to induce significant male reproductive damage in rat models *via* blood-testis barrier disruption, a mechanism likely conserved in aquatic vertebrates.[155] This highlights a critical "hidden" risk that standard acute toxicity assays miss.

4.3. Influence of environmental factors and ENM transformations on toxicity mechanisms

The mechanistic toxicity pathways detailed above represent the intrinsic hazard potential of an ENM. However, this potential is rarely, if ever, fully expressed in a natural aquatic environment. The suite of transformations discussed in section 3 acts as a dynamic environmental filter, profoundly modulating, and in some cases completely rewriting, the toxicological behaviour of the nanoparticle prior to and during its interaction with an organism. Understanding this modulation is key to bridging the gap between laboratory-derived hazard data and real-world ecological risk.

The formation of a natural organic matter (NOM) eco-corona is arguably the most immediate and universal modulator. By adsorbing to the ENM, this layer typically acts as a passivating steric barrier, "quenching" the high surface energy of the pristine material. This directly mitigates toxicity mechanisms that depend on the raw nano-bio interface, such as direct catalytic ROS generation and physical membrane disruption.[99] However, this passivation is a double-edged sword. The corona is far less effective at preventing toxicity driven by the dissolution and subsequent release of toxic ions—the trojan horse pathway remains largely intact.[136,151] Furthermore, the specific composition of the corona is critical; low-molecular-weight fulvic acids can act as chelators that accelerate ion release, whereas high-molecular-weight humic acids provide more effective steric stabilisation. In certain cases, the corona can even amplify toxicity, as seen when adsorbed humic substances act as photosensitisers on the surface of TiO_2, increasing its photocatalytic ROS production under natural solar radiation.[156]

Aggregation, another inevitable process, fundamentally shifts the primary mode of toxicity from the **biochemical to the biophysical**. Large aggregates, often exceeding several hundred nanometres, are typically too large for efficient cellular internalisation by non-phagocytic cells. This reduces the internal dose and consequently lessens the impact of intracellular toxicity pathways like mitochondrial disruption and genotoxicity. However, this decrease in biochemical hazard can be replaced by a significant increase in physical or mechanical toxicity. These large agglomerates can clog the delicate filter-feeding apparatus of zooplankton like *Daphnia*, abrade sensitive gill epithelia in fish leading to hypoxia and secondary infections, and cause physical shading or smothering of benthic primary producers.[157,158]

Finally, direct chemical transformations of the ENM core can fundamentally rewrite its hazard profile. The sulfidation of AgNPs to the highly insoluble and biologically inert mineral Ag_2S, a common process in anoxic sediments, **effectively deletes the dominant toxicity pathway** for pristine AgNPs by shutting down the release of toxic Ag^+ ions.[92,93] Similarly, the progressive oxidation of an nZVI particle's reactive Fe^0 core to a more benign iron (oxy)hydroxide shell concurrently neutralises both its remedial efficacy and its capacity to generate ROS *via* Fenton chemistry. This highlights a central principle for realistic risk assessment: the toxicity of an aged, environmentally transformed ENM is often mechanistically distinct from, and usually less acute than, its pristine counterpart. A major limitation of early nanotoxicology was its reliance on data from pristine materials in clean laboratory media; a modern assessment must consider how the hazard profile itself evolves along the ENM's environmental life cycle.

4.4. Critiquing methodologies

The elucidation of the mechanistic pathways detailed above is contingent on a suite of experimental assays. However, the uncritical application of methods developed for soluble, molecular toxicants to the particulate and reactive nature of ENMs has generated a body of literature that must be interpreted with significant caution. The validity of nanotoxicity data is fundamentally challenged by methodological artefacts, inappropriate dose metrics, and the conceptual difficulty of scaling from molecular events to ecological outcomes.

A primary and pervasive issue is the direct interference of ENMs with common optical and fluorescent assays. High-surface-area materials, particularly carbon-based ENMs, can adsorb colorimetric or fluorescent reporter dyes (*e.g.*, the formazan product in MTT assays or the dichlorofluorescein probe in ROS assays), physically removing them from solution and leading to a significant underestimation of the biological effect (a false-negative).[159–161] Conversely, optically active ENMs like AgNPs, AuNPs, and TiO_2 can scatter and absorb light, interfering with spectrophotometric measurements and producing false-positives. Beyond these physical interferences, redox-active ENMs can chemically interact with the assay probes themselves in cell-free conditions. This necessitates the use of a rigorous suite of abiotic, particle-only controls and, critically, the validation of any single finding with multiple, orthogonal assays that rely on different detection principles.

Furthermore, the conventional dose metric of mass concentration (*e.g.*, mg L^{-1}) is a profoundly inadequate and often misleading descriptor for the biological activity of ENMs. Unlike soluble chemicals, the toxicity of nanoparticles is predominantly a surface-area-driven phenomenon. Two ENM suspensions at identical mass concentrations but with different primary particle sizes will present vastly different total surface areas and particle numbers to a biological system, resulting in dramatically different toxic potentials. This has led to a strong consensus that dose should, at a minimum, be reported in terms of **particle number and total surface area** in addition to mass. The failure to adopt standardised and relevant dosimetry remains a major barrier to comparing results across studies and establishing robust dose–response relationships.

Conceptually, there is a necessary shift away from traditional ecotoxicological tests focused on mechanistically opaque apical endpoints (*e.g.*, mortality, LC_{50}) towards frameworks that embrace causality. The **adverse outcome pathway (AOP)** framework has been rightly championed as a powerful tool for this purpose.[120,162] An AOP provides a structured linkage from a molecular initiating event (MIE), through a cascade of cellular and physiological key events (KEs), to a final adverse outcome (AO) relevant to risk assessment.[120] While this provides an invaluable conceptual roadmap, the classical **linear structure of an AOP is a poor fit for the multi-modal nature of ENMs**. A single ENM can trigger multiple MIEs simultaneously (*e.g.*, membrane disruption, catalytic ROS generation, and ion release), initiating a network of interacting pathways rather than a single, linear chain. The development of quantitative "AOP Networks" is a logical next step, but this presents a formidable challenge in complexity.

Finally, the ultimate challenge is the extrapolation of mechanistic data, often derived from *in vitro* mono-cultures or simple model organisms, to predict ecologically relevant impacts. Bridging the immense **scale-translation gap**—from molecular perturbation in a single cell to altered population dynamics and community structure in a complex, fluctuating ecosystem—is a critical frontier for environmental nanoscience. It is this gap between understanding a mechanism and predicting its ecological consequence that currently represents the greatest uncertainty in the risk half of the nanoremediation equation.

4.5. Beyond the cell: trophic transfer and ecosystem-level consequences

A complete toxicological assessment cannot be confined to the molecular and cellular perturbations within a single organism. The ultimate ecological risk of deploying remediation ENMs is a function of their impacts on population dynamics, inter-species interactions, and foundational ecosystem processes. The current over-reliance on single-species, laboratory-based toxicity tests, while mechanistically informative, is ecologically reductionist and provides an incomplete picture of potential environmental harm. A systems-level perspective is therefore not an afterthought, but a necessity.

A primary concern at the ecosystem level is the potential for **trophic transfer and biomagnification**. As established in section 4.1, dietary uptake is a dominant exposure pathway for primary consumers like zooplankton, which ingest ENM-laden algae and detritus.[163,164] This provides a clear vector for the entry of ENMs into aquatic food webs. The critical question, however, is whether the concentration of these materials increases at successively higher trophic levels, as is the case for legacy contaminants like mercury and PCBs. To date, the evidence for classical biomagnification of ENMs is limited and often contradictory. For many ENM types, **biodiminution** (a decrease in concentration up the food chain) appears to be a more common phenomenon. This is attributable to several factors: the low assimilation efficiency of particulate or aggregated material across gut epithelia, leading to high rates of egestion; and active depuration or biotransformation of internalised ENMs (*e.g.*, sulfidation of AgNPs within the gut lumen), which reduces the bioavailable fraction for a predator.[165,166] However, **the absence of biomagnification does not equate to an absence of risk**. The trophic transfer of even non-magnifying concentrations can still deliver a bioactive dose to sensitive predators and represents a critical pathway for chronic, long-term exposure throughout the food web.

Perhaps more insidious, and far less studied, are the potential impacts of remediation ENMs on **microbial communities and biogeochemical cycling**.[167,168] Aquatic ecosystems are built upon the metabolic activity of complex microbial consortia that drive the foundational cycles of carbon, nitrogen, phosphorus, and sulphur. Many remediation ENMs, particularly those containing silver, copper, or zinc, possess potent, broad-spectrum antimicrobial properties. Their release into an aquatic system can therefore disrupt the structure and function of natural microbial communities, such as benthic biofilms. This could have profound consequences for ecosystem health by impairing critical processes. For example, inhibition of key microbial guilds involved in the nitrogen cycle, such as nitrifying bacteria (which convert ammonia to nitrate) or denitrifying bacteria, could alter the nutrient dynamics and productivity of an entire system.

In conclusion, a truly predictive ecotoxicology for nanomaterials must look beyond the cell and the individual. While classical biomagnification appears to be less of a concern than for legacy contaminants, the risks associated with trophic transfer and subtle, long-term perturbations of microbial-driven ecosystem functions remain poorly understood and represent a major knowledge gap. Future research must therefore embrace greater ecological complexity, moving beyond simplified laboratory assays towards mesocosm experiments and ecosystem function endpoints to probe these subtle but potentially significant environmental impacts.

5. The nexus: critically analyzing the benefit–risk balance

The preceding sections establish the fundamental paradox of nanoremediation: the very physicochemical properties meticulously engineered for high remedial efficacy are often the direct drivers of ecotoxicological hazard. A responsible and sustainable path forward for these potent technologies demands a paradigm shift—moving beyond siloed evaluations of benefit and risk towards an integrated, mechanistic analysis of their nexus. This section deconstructs the shared principles that govern both remediation and toxicity. It critically examines the direct correlations between these mechanisms, uses illustrative case studies to explore this duality in practice, and analyses the pivotal role of the aquatic environment in modulating both sides of the benefit–risk equation, a concept visually synthesised in Fig. 4.

5.1. Deconstructing the mechanistic overlap

The "double-edged sword" hypothesis is not merely a convenient analogy; it is a reflection of a shared thermodynamic and kinetic basis for the desired remedial action and the undesired biological harm. This mechanistic overlap is not coincidental but is rooted in fundamental principles of surface and solution-phase chemistry. The correlations can be categorised into several key classes.

- **Generation of non-selective reactive species:** this is the most conspicuous mechanistic link, exemplified by photocatalysts like TiO_2 and other advanced oxidation process (AOP) materials. The therapeutic action relies on the generation of powerfully oxidising species, principally the hydroxyl radical ($^{\bullet}OH$). With a standard redox potential of +2.8 V, the $^{\bullet}OH$ radical is one of the most powerful oxidants known, capable of indiscriminately mineralising a vast range of recalcitrant organic pollutants.[57,135] This very **non-selectivity** is simultaneously its greatest liability. The radical does not distinguish between a C–H bond in a pesticide and a C–H bond in a lipid membrane or a DNA base, making it a primary molecular initiating event for oxidative stress and cellular damage in non-target organisms.[135,169]
- **Intrinsic redox potential and reactivity:** for materials like nanoscale zero-valent iron (nZVI), the remedial function is predicated on their intrinsic redox potential. The strong reducing power of the Fe^0 core ($E^0(Fe^{2+}/Fe^0) = -0.44$ V) is essential for the reductive dechlorination of solvents or the detoxification of Cr(VI).[170,171] However, this same thermodynamic drive to donate electrons can be repurposed within a biological system. It can directly reduce endogenous

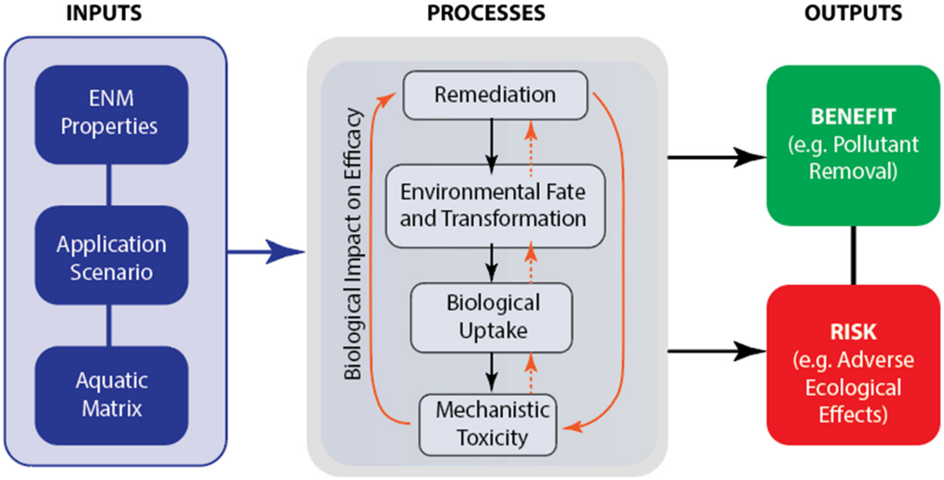

Fig. 4 A conceptual framework for the integrated benefit–risk assessment of a nanoremediation technology. The model illustrates the flow from initial **inputs** (intrinsic ENM properties, application scenario, and the aquatic matrix) to a central nexus of dynamic **processes**. Within this nexus, remediation efficacy, environmental fate, biological uptake, and mechanistic toxicity are shown as critically interlinked, with key feedback loops influencing each other. These complex interactions ultimately lead to two distinct sets of **outputs**: the intended environmental **benefit** (*e.g.*, pollutant removal) and the unintended ecological **risk** (*e.g.*, adverse effects on non-target organisms).

molecules, disrupting sensitive cellular redox signaling pathways, or more commonly, reduce dissolved oxygen to initiate the Fenton reaction cascade, leading to the production of the same damaging $^{\bullet}$OH radical discussed above.[172] The fundamental chemical property is conserved; only the reaction partner changes from an environmental contaminant to a biological molecule.

- **High surface energy and adsorptive capacity:** for sorbent materials like graphene oxide or CNTs, their efficacy is a function of their vast specific surface area and the presence of functionalities that drive strong adsorptive interactions. These forces—van der Waals, hydrophobic interactions, π–π stacking, and hydrogen bonding—are precisely the same forces that govern their adhesion to biological surfaces.[46] The strong affinity for binding an organic pollutant is mechanistically analogous to the strong, and often disruptive, adhesion to the phospholipid and protein components of a cell membrane, which can lead to physical stress, lipid extraction, or protein denaturation.[173,174]

This direct mechanistic coupling presents a profound challenge. Strategies that narrowly focus on maximising a single performance metric—such as "reactivity" or "surface area"—are inherently flawed, as they will almost certainly amplify the potential for hazard in parallel. This implies that the future of sustainable nanoremediation lies not in simply making materials more powerful, but in making them smarter. The grand challenge is **mechanistic decoupling**: the rational design of materials that can maintain high affinity and reactivity towards specific target pollutants while minimising non-specific and harmful interactions with biological systems.

5.2. Case studies in mechanistic duality

Examining archetypal remediation ENMs for which extensive data exist on both efficacy and toxicology provides a clear, practical illustration of the benefit–risk conflict.

- **Titanium dioxide (TiO_2):** the archetype of collateral damage as a photocatalyst, nano-TiO_2 is highly effective for the oxidative degradation of organic pollutants. Its remedial benefit is a direct, linear function of its quantum yield for generating reactive oxygen species (ROS) under UV irradiation. The risk, however, is phototoxicity, which is governed by precisely the same mechanism. Extensive studies have demonstrated that UV-irradiated TiO_2 causes severe oxidative damage to the membranes, proteins, and DNA of algae, daphnids, and fish.[136,137,151] For this ENM, the benefit (pollutant mineralisation) and the risk (ecotoxicity) are **mechanistically inseparable**, both originating from the indiscriminate reactivity of photogenerated ROS. Strategies to improve performance, such as doping to create visible-light activity, may alter the energy input but do not resolve the fundamental reliance on ROS, meaning the collateral damage potential is an intrinsic design feature.
- **Nanoscale zero-valent iron (nZVI):** a case of programmed instability the high efficacy of nZVI as a reductant is a direct consequence of its thermodynamic instability in water. This "programmed instability" makes it a potent agent for detoxifying chlorinated solvents and heavy metals like Cr(VI). Yet, the very corrosion process that drives its remedial action is also a primary source of its toxicity. The inevitable oxidation of Fe^0 in oxic waters produces Fe^{2+} ions, which can participate in Fenton chemistry to generate the highly damaging $^{\bullet}$OH radical, inducing severe oxidative stress in exposed organisms.[172,175] Therefore, the benefit and risk are two facets of the same core chemical process. Surface coatings designed to slow this corrosion can temper the toxicity but simultaneously reduce the material's remedial reactivity, creating a direct and challenging trade-off between environmental safety and functional performance.
- **Silver nanoparticles (AgNPs):** the trojan horse dilemma as a potent antimicrobial agent, AgNPs have been explored for controlling biofouling or harmful algal blooms.[176,177] Their benefit is derived almost entirely from their function as a high-surface-area delivery vehicle for toxic silver ions (Ag^+), which are released *via* oxidative dissolution. The risk to non-target aquatic organisms is, of course, identical: toxicity mediated by the release of Ag^+ ions that disrupt enzyme function and membrane integrity. For AgNPs, the benefit and risk are not merely correlated; they are mechanistically identical. The only distinction is the intended *versus* the unintended target. This presents a profound management challenge, as it is impossible to enhance the desired function without concurrently amplifying the potential for ecological harm.

5.3. The environmental matrix: a dynamic modulator of the Nexus

The aquatic environment is not a passive stage for these interactions but an active and complex chemical system that dynamically modulates both sides of the benefit–risk equation. Water chemistry acts as a complex lens, capable of amplifying, attenuating, or even decoupling the relationship between remedial efficacy and toxicological hazard.[165,178] For instance, high water hardness can attenuate AgNP toxicity by providing competing cations (Ca^{2+}, Mg^{2+}) that reduce Ag^+ uptake at the gills, yet this same water chemistry promotes aggregation, which can reduce the active surface area available for any desired catalytic function.[111,151,179]

The role of NOM is particularly illustrative of this decoupling potential. The formation of a dense, high-molecular-weight humic corona typically passivates the ENM surface. This can simultaneously reduce its efficacy (*e.g.*, blocking catalytic sites) and its surface-reactivity-driven toxicity (*e.g.*, preventing direct membrane contact).[180–182] However, this same corona may have a negligible effect on toxicity pathways driven by ion dissolution if it does not significantly alter the material's dissolution kinetics.[182] The environment, therefore, does not act as a simple dimmer switch, turning benefit and risk up or down in unison. It selectively modulates different mechanisms, creating a complex, non-linear, and highly context-dependent benefit–risk profile. A critical failure of many laboratory assessments is the use of simplified, clean media, which generates environmentally naive conclusions that are invalid for real-world systems.

5.4. Towards quantitative risk–benefit assessment

A truly holistic evaluation requires a quantitative framework for weighing the societal and environmental benefits of remediation against the potential ecological risks, which can be operationalized using a decision-tree approach (Fig. 5). This remains a formidable challenge, primarily due to the problem of **incommensurable endpoints**. Remediation performance is quantified in process-based units (*e.g.*, % removal, adsorption capacity in mg g^{-1}, rate constants in s^{-1}), while ecotoxicity is measured with biological effect concentrations (*e.g.*, EC_{50} or NOEC in mg L^{-1}). A quantitative comparison requires a common functional framework, which can be conceptually expressed as:

$$\text{Net Impact} = f(\text{Performance Metrics}) - g(\text{Hazard Metrics})$$

Here, f and g are complex, multi-dimensional functions modulated by a vector of environmental parameters. The minus sign represents a societal weighting or trade-off, not a simple mathematical subtraction. Defining these functions is a grand challenge, as it requires placing a value on disparate outcomes: for example, how does one weigh the benefit of removing a known carcinogen against the risk of reducing the reproductive output of a keystone invertebrate species?.[183]

To date, most assessments remain qualitative. Promising quantitative tools like **lifecycle assessment (LCA)** and **multi-criteria decision analysis (MCDA)** offer a structured path forward.[184] MCDA can integrate diverse data types and allow stakeholders to assign weights to different criteria (*e.g.*, efficacy, cost, acute toxicity, chronic effects, manufacturing impacts) to rank technology options. However, the output of

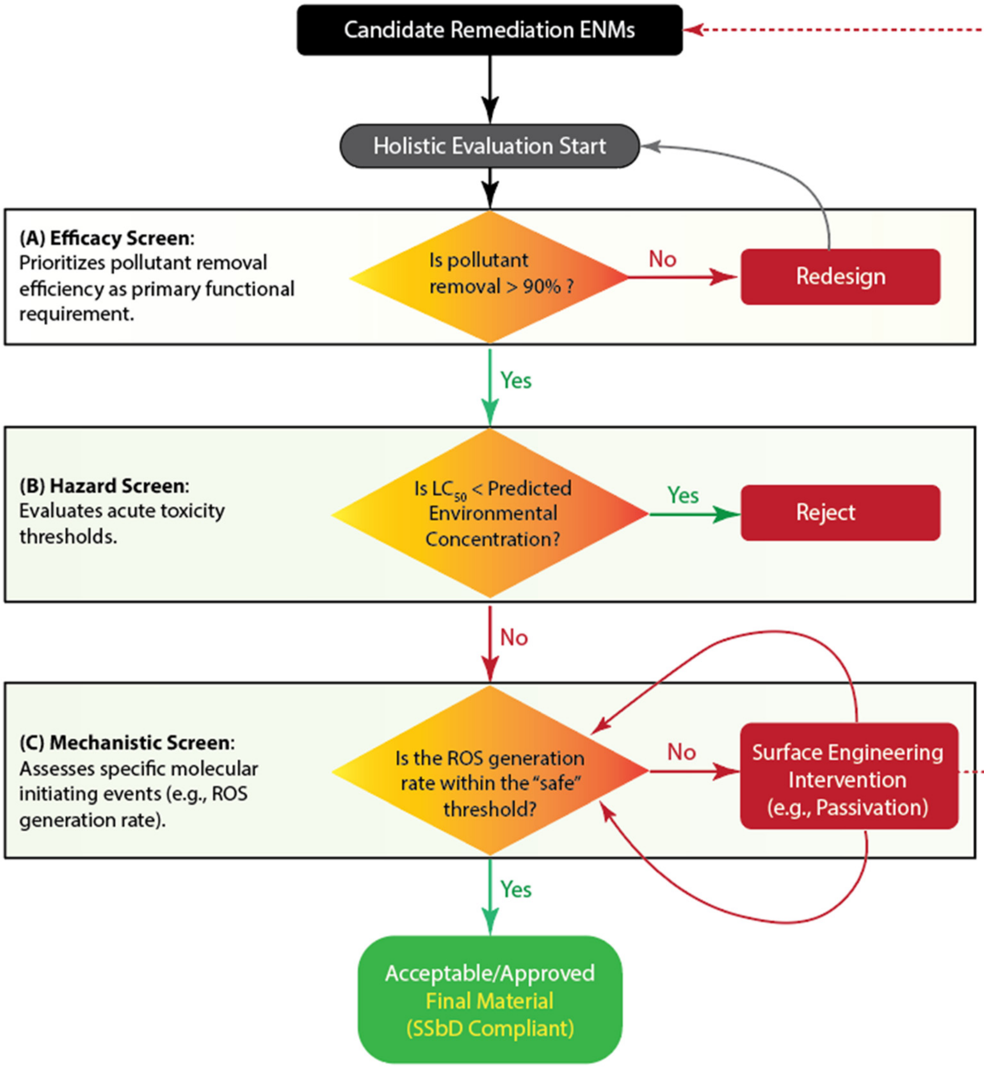

Fig. 5 A decision-tree framework for the operationalized benefit–risk assessment of remediation ENMs. The schematic outlines a hierarchical screening process designed to filter candidate materials based on sequential "go/no-go" criteria.

an MCDA is highly sensitive to these weighting factors, which can be subjective. It is a powerful tool for structured decision-making, not an objective arbiter of truth. Ultimately, the development of any robust risk–benefit model is critically dependent on populating it with comprehensive, environmentally realistic data. Without such frameworks, the selection of nanoremediation technologies risks being driven by the most easily measured metric—remedial efficiency—potentially externalising significant and unquantified ecological costs.

5.5. A call for a "Safe-and-Sustainable-by-Design" (SSbD) paradigm

The mechanistic overlaps, environmental modulations, and assessment challenges detailed throughout this section underscore the inherent limitations of a conventional, *post hoc* approach to risk assessment. The traditional paradigm of "synthesise first, test for safety later" is not only inefficient but is ill-suited for a technology where the drivers of function and hazard are so deeply intertwined. A more intelligent and responsible approach is required. This necessitates a fundamental shift towards a proactive **safe-and-sustainable-by-design (SSbD)** framework, where considerations of ecological safety and lifecycle sustainability are not an afterthought, but are integral components of the material innovation process from its inception.[185,186] The SSbD framework defined by the European Commission's Joint Research Centre (JRC) 2023 report, which integrates safety assessment into the earliest stages of innovation, is advocated.

Design thresholds for SSbD: to operationalize this, specific quantitative design targets for remediation ENMs are proposed:

- **Dissolution stability:** <5% ion release over the operational lifetime (*e.g.*, *via* doping or core–shell structures).
- **Surface charge (zeta potential):** target values within ±10 mV to promote controlled aggregation and settling post-treatment, reducing water column exposure.
- **Band gap engineering:** tuning E_g to visible light range (>400 nm) to minimize UV-driven phototoxicity to non-target surface organisms.

Embedding mechanistic toxicology into ENM design is pivotal to ensuring efficacy and safety co-evolve. By targeting these specific physicochemical levers, a transition can be made from a reactive "risk management" stance to a proactive "safety engineering" paradigm.

The implementation of an SSbD paradigm for remediation ENMs would be guided by several core principles aimed at resolving the benefit–risk conflict:

1. Pursuit of mechanistic decoupling: as previously stated, the central goal is to rationally design materials that can decouple remedial efficacy from ecotoxicological hazard (Fig. 6). This moves beyond simply maximising reactivity and towards engineering selectivity. Potential strategies include developing "smart" materials that remain relatively inert until activated by a specific trigger unique to the contaminated environment (*e.g.*, a particular pH, redox potential, or the presence of the target pollutant itself). Another avenue is advanced surface engineering to create coatings that are selectively permeable to contaminants but act as a robust physical and chemical barrier to biological macromolecules, thus preventing the non-specific interactions that initiate toxicity.[186]

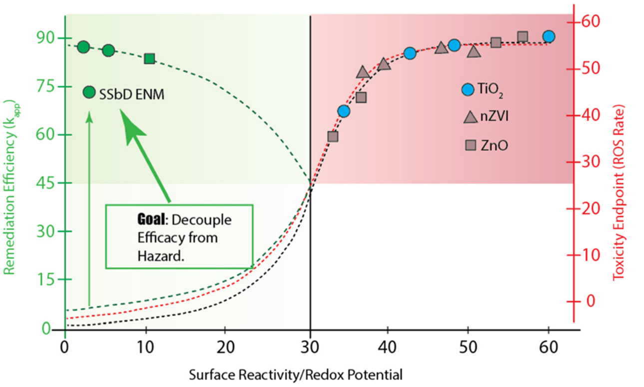

Fig. 6 The mechanistic duality map, a conceptual plot illustrating the core challenge of nanoremediation and the goal of a safe-and-sustainable-by-design (SSbD) approach. The map correlates a key physicochemical property driving ENM activity (*e.g.*, surface reactivity) with dual and competing outcomes: remediation efficiency (left *y*-axis, in green) and a key mechanistic toxicity endpoint (right *y*-axis, in red). Conventional high-performance ENMs (*e.g.*, nZVI, TiO$_2$) often occupy a high-efficacy, high-risk space. The explicit goal of SSbD, as indicated by the arrow, is to rationally design novel materials that occupy a high-efficacy, low-risk space, thereby **mechanistically decoupling** therapeutic function from ecological hazard.

2. Designing for a benign environmental fate: the "end-of-life" of the ENM should be a primary design parameter. Rather than striving for maximum persistence, an SSbD approach would involve programming the material for a specific functional lifetime. This could include designing ENMs that, after their task is complete, are triggered to aggregate and sediment, or more elegantly, to dissolve into bio-essential and non-toxic constituent ions (*e.g.*, iron oxides dissolving into bioavailable iron). This requires a deep understanding of the material's long-term transformation pathways under relevant environmental conditions.

3. Adoption of lifecycle thinking: the scope of SSbD must extend beyond the use-phase to encompass the entire lifecycle of the nanomaterial.[187] This includes selecting sustainable and non-toxic precursors, employing energy-efficient and scalable "green synthesis" routes that minimise hazardous waste, and ensuring that the recovery and regeneration of the ENMs (where applicable) is both feasible and environmentally sound.

Implementing a true SSbD framework is a formidable challenge. It demands a level of predictive understanding of nano-bio interactions that the field is only beginning to attain. It requires a move away from disciplinary silos towards a new, deeply integrated model of co-design, where materials chemists, (eco)toxicologists, environmental scientists, and modelling experts collaborate from the very outset of the innovation process. While difficult, the SSbD paradigm represents the most promising—and perhaps the only—pathway to unlock the full, transformative potential of nanoremediation without imposing an unacceptable and unforeseen burden of ecological risk.

6. Synthesis: critical gaps, challenges, and future directions

The analysis presented in this review confirms that while nanoremediation holds substantial, and perhaps transformative, promise for addressing critical water quality challenges, its progression towards sustainable implementation is constrained by a series of interconnected scientific gaps and methodological hurdles. The central tension between remedial efficacy and ecological safety is not a simple trade-off but a complex, mechanistically-rooted duality. Advancing the field beyond its current state requires a concerted and strategic effort to move from a descriptive to a predictive science. This concluding section synthesises the key knowledge gaps that define the research frontier, outlines the methodological barriers that must be overcome, and charts a critical path forward guided by a new paradigm of proactive and integrated technology development.

6.1. The frontiers: key knowledge gaps

Despite a burgeoning literature, our understanding of the long-term, ecosystem-level consequences of deploying remediation ENMs remains fundamentally incomplete. Several critical knowledge gaps define the frontier of the field:

- **Chronic and sub-lethal mechanistic toxicity:** the current body of knowledge is overwhelmingly dominated by studies of acute exposure at high, often environmentally unrealistic, concentrations. There is a profound deficit of data on the mechanistic impacts of chronic, low-dose exposures. It is highly plausible that such long-term exposures trigger entirely different and more subtle toxicity pathways, such as endocrine disruption, metabolic reprogramming, or even transgenerational epigenetic effects, which are invisible in standard 96 hour acute assays.[188,189]
- **Toxicity of the "Nano-Waste Stream":** in any real application, aquatic ecosystems will be exposed to a complex "nano-waste stream" comprising a mixture of the original ENMs, their various environmentally transformed progeny, the target pollutants, and the pollutant degradation by-products. The combined toxicological effect of this mixture—which may be additive, synergistic, or antagonistic—is almost entirely uncharacterised and represents a more realistic exposure scenario than any single-component study.
- **Consequences of trophic transfer:** while the trophic transfer of ENMs from primary producers to consumers is now well-established, the ultimate consequences remain ambiguous. The focus is shifting from the question of whether classical biomagnification occurs (for which evidence is limited) to the more nuanced question of how the chronic ingestion of ENMs affects the long-term health, fitness, and reproductive success of higher-trophic-level organisms.[124,126,163]
- **Impact of multi-stage environmental aging:** as this review has stressed, ENMs are subject to continuous transformation. While the impact of a simple humic acid corona is relatively well-studied, the effect of complex, multi-stage aging (*e.g.*, sequential corona formation, partial dissolution, oxidation, and aggregation) on the ultimate toxic mechanism is a major unknown. The biological response is to these aged forms, making the focus on pristine ENMs a significant source of uncertainty.
- **Linking molecular mechanisms to ecological impacts:** a formidable gap persists between laboratory-derived molecular data and meaningful ecological outcomes. While AOPs can conceptually link a molecular initiating event to an adverse outcome in an individual, predicting how this translates to population-level effects (*e.g.*, reduced population viability) or community-level impacts (*e.g.*, altered species composition or impaired ecosystem functions like primary productivity) requires a new generation of integrated ecological models that are still in their infancy for nanomaterials.

6.2. The barriers: overcoming methodological hurdles

Progress on these frontiers is fundamentally constrained by significant methodological barriers. Closing the knowledge gaps requires a parallel advancement in our experimental and analytical capabilities:

- **Developing artefact-free, relevant mechanistic assays:** as noted, the uncritical use of conventional toxicity assays has likely generated a body of data compromised by ENM-specific artefacts. There is a pressing need for validated, interference-free methods and high-throughput screening (HTS) platforms that can probe mechanistic endpoints (*e.g.*, transcriptomics, metabolomics) and assess the bio-reactivity of the ENM *with its eco-corona intact*.[190]
- **Achieving true *in situ* characterisation:** progress on understanding environmental aging is directly limited by our inability to accurately characterise ENMs within complex matrices. This "linchpin challenge" requires the development of novel analytical techniques capable of real-time, *in situ* measurement of key ENM properties (*e.g.*, aggregation state, corona composition, dissolution rate) within exposure media and even biological tissues, moving beyond the static, *ex situ* snapshots provided by current methods.
- **Bridging the lab-to-field chasm:** the difficulty in extrapolating from controlled laboratory systems to heterogeneous ecosystems is a perennial challenge. For nanomaterials, this is amplified by their dynamic behaviour. While resource-intensive, well-designed mesocosm studies are an indispensable "gold standard" for validating lab findings and models in a more realistic context. Developing validated, tiered testing strategies that intelligently combine *in vitro*, *in vivo*, and mesocosm data is essential for making this challenge more tractable.

6.3. The path forward: a vision for sustainable nanoremediation

Addressing this formidable array of gaps and challenges requires more than incremental progress; it demands a paradigm shift. The future of nanoremediation must be guided by a proactive, integrated, and systems-level approach.

- **Embracing safe-and-sustainable-by-design (SSbD):** as argued in section 5.5, the SSbD paradigm must become the guiding principle of the field.[187] This involves embedding safety and sustainability assessments into the earliest stages of material design, with a focus on mechanistic decoupling (preserving efficacy while minimising hazard) and designing for a benign fate (*e.g.*, controlled dissolution into non-toxic components).
- **Advancing predictive mechanistic modelling:** to make SSbD a reality, a new generation of computational models is needed. The field must move beyond empirical QSARs towards mechanism-based, systems toxicology models that can quantitatively predict the dynamics of AOP networks based on an ENM's intrinsic properties and the environmental context.
- **Integrating lifecycle assessment (LCA):** a technology is not sustainable if its production creates a greater environmental burden than its application resolves. Integrating mechanistic toxicity and fate data into formal LCA frameworks is the only way to conduct a true "net environmental benefit" analysis, accounting for impacts from precursor synthesis to end-of-life disposal.[191,192]
- **Informing fit-for-purpose regulation:** current chemical regulatory frameworks like REACH are ill-equipped for the complexities of ENMs. The mechanistic and fate data generated by the research community must be used to inform a necessary evolution towards more nuanced, property-based regulations that consider parameters beyond mass concentration, such as surface area, reactivity, and transformation potential.

In conclusion, the duality of the nanoparticle—its immense potential for both benefit and risk—presents both a grand scientific challenge and a profound responsibility. The journey from a powerful chemical tool to a truly sustainable environmental solution requires a steadfast and collaborative commitment to the principles of mechanistic science, ecological realism, and proactive design. The ultimate success of nanoremediation will be measured not only by the pollutants it removes from water but by the wisdom and foresight with which it is conceived and deployed.

7. Conclusions

The application of engineered nanomaterials to aquatic remediation is governed by a fundamental mechanistic duality: the unique physicochemical properties that drive high remedial efficacy are inextricably and often identically linked to the pathways that initiate ecotoxicological harm. This critical review has demonstrated that the environmental fate and transformation of these materials—processes of aggregation, dissolution, and corona formation—are not confounding factors but are central to the narrative, acting as a dynamic environmental filter that continuously reshapes the benefit–risk nexus. This reality renders a simple, siloed evaluation of 'benefit' *versus* 'risk' scientifically untenable for these complex, dynamic materials.

The deep-seated connection between intended function and unintended hazard demands a paradigm shift away from the traditional, reactive model of "synthesise first, test for safety later". The future of sustainable nanoremediation hinges on the widespread adoption of a proactive and integrated safe-and-sustainable-by-design (SSbD) philosophy. This approach elevates mechanistic toxicology from a downstream regulatory hurdle to an indispensable, *a priori* design tool. The central scientific goal must be mechanistic decoupling—the rational modulation of surface chemistry, dissolution kinetics, and electronic properties to strategically sharpen the "benefit" edge of the sword while simultaneously blunting the "risk" edge.

Achieving this vision is a grand challenge that requires more than just clever materials chemistry; it demands a new research culture founded on deep interdisciplinarity. It necessitates the integration of advanced *in situ* analytics, predictive mechanism-based models, and holistic lifecycle assessment frameworks. Ultimately, the successful transition of nanoremediation from a powerful proof-of-concept to a globally trusted environmental solution will be measured not just by the contaminants it removes, but by the wisdom and foresight with which it is designed and deployed. Only through such a mechanistically-informed and safety-conscious perspective can the full potential of nanotechnology for ensuring global water security be realised responsibly and sustainably.

Author contributions

Akeem Adeyemi Oladipo: conceptualization, data curation, writing – review & editing, writing – original draft, visualization.

Conflicts of interest

There are no conflicts to declare.

Abbreviation

AOP	Adverse outcome pathway
As(V)	Arsenate
CNTs	Carbon nanotubes
Cr(VI)	Hexavalent chromium
DLVO	Derjaguin–Landau–Verwey–Overbeek
DOM	Dissolved organic matter
EC_{50}	Median effective concentration
ENMs	Engineered nanomaterials
GO	Graphene oxide
HTS	High-throughput screening
LCA	Lifecycle assessment
MCDA	Multi-criteria decision analysis
MIEs	Molecular initiating events
NNPs	Natural nanoparticles
NOEC	No-observed-effect concentration

NOM	Natural organic matter
nZVI	Nanoscale zero-valent iron
PAHs	Polycyclic aromatic hydrocarbons
PCBs	Polychlorinated biphenyls
PECs	Predicted environmental concentrations
POPs	Persistent organic pollutants
QSAR	Quantitative structure–activity relationship
rGO	Reduced graphene oxide
ROS	Reactive oxygen species
SbD	Safer-by-design
sp-ICP-MS	Single-particle inductively coupled plasma-mass spectrometry
TCE	Trichloroethylene
TiO_2	Titanium dioxide
ZnO	Zinc oxide
8-OHdG	8-Hydroxy-2′-deoxyguanosine

Data availability

No new data were created or analyzed in this study. Data sharing is not applicable to this article as no new data were generated.

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