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Review

New Insights for Exploring the Risks of Bioaccumulation, Molecular Mechanisms, and Cellular Toxicities of AgNPs in Aquatic Ecosystem

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Abstract: Silver nanoparticles (AgNPs) are commonly used in numerous consumer products, including textiles, cosmetics, and health care items. The widespread usage of AgNPs results in their unavoidable discharge into the ecosystem, which pollutes the aquatic, groundwater, sediments, and marine environments. These nanoparticles (NPs) activate the production of free radicals reactive species in aquatic organisms that interrupt the functions of DNA, cause mitochondrial dysfunction, and increase lipid peroxidation, which terminates the development and reproduction both in vivo and in vitro. The life present in the aquatic ecosystem is becoming threatened due to the release and exploitation of AgNPs. Managing the aquatic ecosystem from the AgNP effects in the near future is highly recommended. In this review, we discussed the background of AgNPs, their discharge, and uptake by aquatic organisms, the mechanism of toxicity, different pathways of cytotoxicity, and bioaccumulation, particularly in aquatic organisms. We have also discussed the antimicrobial activities of AgNPs along with acute and chronic toxicity in aquatic groups of organisms.

Keywords: AgNPs; marine ecosystem; toxicity; bioaccumulation; pollutants; molecular mechanisms

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1. Introduction

Water is the most important natural resource on the planet, and its existence in its purest form is essential for all living organisms since life would be unimaginable without water [1]. Physical, chemical, and biological factors and their interactions significantly impact water quality [2]. According to recently released United Nations (UN) statistics, about 60% of the world's population may be living in water-stressed areas by 2025 [3]. It may severely impact many areas of contemporary life, including food and energy shortages, which could lead to increased disease rates. As the world's population and activities grow, so does wastewater production. Excessive discharge of toxic pollutants into water bodies has adverse effects on human health, aquatic organisms, and ecosystems because they change the physical and chemical characteristics of the water, rendering it unfit for use by people, animals, and plants [3].

Nanotechnology has become a significant scientific and economic development in water conservation. However, the widespread use of nanomaterials, toxic compounds, and effluents released from industrial processes has ultimately negatively affected the different modes of life [4]. Nanomaterials are synthesized in various forms, sizes, shapes, and functionalities with their extensive applications. Nano-sized products have been established in drug delivery, tissue engineering, diagnostics, energy, environmental remediation, chemical sensors, and agricultural sciences [5]. They have exceptional physicochemical properties in comparison to the bulk metals from which they are extracted and have made numerous advances in various fields [6,7]. The most micro-genic approach to AgNP synthesis gained attention because of its wide biocompatibility and good stability [8].

AgNPs' broad-spectrum antibacterial activity has been exploited in several marketable products such as textiles, household appliances, food packaging, shampoos, toothpaste, water filters, and medical devices; all of these contribute to the excessive discharge of AgNPs into the aquatic ecosystem through use and manufacturing [9,10]. This substantial rise in the number of products available in the market, including AgNPs, raises the risk of their release and aggregation in the environment and triggers harmful effects on the organisms continuously exposed to these materials [11]. Recent advances in designing novel engineered nanomaterials have expanded social and environmental awareness, making it much more essential to consider the future consequences of synthesis and the use of AgNPs [12].

In daily life, nanomaterials are widely used and readily discharged into the aquatic ecosystem, a significant source of water pollution [13]. About 2500 metric tonnes of silver are discharged into the environment each year as a result of industrial waste and emissions, with an additional 150 metric tonnes ending up in sewage sludge and 80 metric tonnes going into surface waterways [14]. They can cause environmental pollution with different particle morphology and composition of an element, intentionally or unintentionally.

In terms of heavy metal toxicity, silver ion is second only to mercury and is thus classified in the highest toxicity class along with cadmium, chromium (VI), copper, and mercury [15]. For all the metals, the interaction of adjacent locations influences the connection of silver ions through different ligands, and these impacts persuade the toxicity and bioavailability [16–18]. AgNPs cause multiple toxic effects on aquatic groups of organisms because of their exposure conditions (e.g., pH, conductivity, media composition, and temperature) and physicochemical properties, i.e., size and surface coating [19–21]. For example, sulfide and organic matter have strong silver sensitivity in freshwater environments, and possibly control of silver ions speciation decreases silver bioavailability. Along with all the discussion of AgNPs and their toxicity processes, we have noticed that no work has been done to identify the agent that detoxifies the nanoparticles (NPs) [22]. So, there is also a need for urgent attention to establishing the biological control and bioremediation of AgNPs to reduce silver toxicity through microorganisms such as bacteria, fungi, protozoa, or algae to save the ecosystem and the lives of aquatic organisms.

This review discussed the background of AgNPs, their discharge and uptake by aquatic organisms, the mechanism of toxicity of AgNPs, and bioaccumulation in the

aquatic ecosystem and its life. The present study discussed the antimicrobial activity of AgNPs, along with acute and chronic toxicity. The present study also highlighted the toxic effects of AgNPs on aquatic groups of organisms.

2. Discharge of AgNPs into the Aquatic Environment

Global production of AgNPs currently ranges between 420 and 500 tonnes per year [22,23]. Silver nanoparticles can be released directly and indirectly into the environment throughout their life cycle (manufacture, transport, use, and disposal). An example of direct release could be discharged from transport accidents and all types of spills. The indirect release could be due to discharge from wastewater treatment plants which receive discarded nanoparticles at the end of their life cycle [20]. The surface water compartment (rivers, seas) receives the discharge of AgNPs either directly effluents from wastewater treatment plants or from other environmental compartments. Several ways, like residual industrial material, chemical spills, runoff, or washing machines, increase the discharge of AgNPs into the aquatic ecosystem, including synthesis, the integrating of NPs inside the products, using different things containing NPs, and recycling/disposing of things [21]. Silver nanoparticles can be highly toxic to aquatic organisms due to the same mechanisms of toxicity for terrestrial species. Marine invertebrates and small fish make good indicators of aquatic toxicity due to their small size and high degree of susceptibility to environmental pollutants. Zebrafish (*Danio rerio*) are a model organism for aquatic studies since the number of available materials and data on these fish make them efficient for streamlining ecotoxicological testing. Hence, aquatic ecosystems receive an important amount of the released AgNPs [22]. In that case, the possibility of silver ion concentration in the natural environment may rise locally to a level of mass that may typically match or surpass the peak rates of dissolved silver ions found in the polluted waters [24], which would intensify apprehensions around environmental hazards [25].

In the aquatic environment, the fate of AgNPs is determined by their capping agents [26]. Polyvinylpyrrolidone (PVP) and polymer-based citrate are the most commonly used capping agents that increase the risk of heavy metal toxicity in aquatic organisms. PVP and citrate-based AgNPs induced metal toxicity in the gut epithelial tissues of *Mytilus galloprovincialis* [27]. It is demonstrated that the accumulation of PVP and citrate in coelomocytes of *Nereis diversicolor* increased DNA damage and lysosomal membrane permeability. Environmental exposure to PVP and citrate also damages the pancreatic tissues in *Litopenaeus vannamei* [28]. Another study investigated that exposure of 200 µg/L PVP-AgNPs to *Cyprinus carpio* increased the damage to the brain and gills [29]. Uptake of PVP and citrate-based AgNPs by *Ampelisca abdita* and *Americamysis bahia* also damages their skin epithelial tissues [4]. PVP and citrate-based AgNPs also aggregate with the microalgae, *Raphidocelis subcapitata*, causing faster segmentation of filaments [30]. The antioxidant defense system arises from glutathione peroxidase and catalase in aquatic organisms that lower the toxic effects of PVP and citrate AgNPs [31].

Dissolution and Toxicity in Fresh and Marine Water

A recent study reported the toxicity of silver ions and Ag-NPPVP for *Ceramium tenuicorne* and *Tisbe battagliai*. It was found that *Ceramium tenuicorne* exhibited EC_{50} values of 2312.2 and 26.6 mg/L against silver ions and Ag-NPPVP, while *Tisbe battagliai* showed only EC_{50} values of 90.9 and 7.9 mg/L, respectively 27. The EC_{50} values for *Scenedesmus* sp. and *Thalassiosira* sp. were 89.92 ± 9.68 and 107.21 ± 7.43 µg/L, respectively [20,32]. A low EC_{50} value (0.055 mg/L) for *A. salina* was also determined with AgNPs (2–18 nm) at concentrations between 0 and 1.5 mg/L [33]. The toxicity of PVP-coated AgNPs in *Daphnia* sp. from different boreal lakes was evaluated and found to have an LC_{50} ranging from 34 to 292 µg/L [34]. In contrast to silver ion, the toxicity of Ag-NPPVP enhanced substantially with increasing salinity. However, despite extensive characterization, it was not feasible to link particle behaviour with an increase in toxicity and salinity. The findings indicate that the observed toxicity is caused by free ionic silver complexing in solution and an unknown possible particle-related impact [35]. Pham [36] estimated the effect of AgNPs on the algal species in marine (*Thalassiosira* sp.) and freshwater (*Scenedesmus* sp.)

environments, and his findings described that AgNPs are more hazardous in freshwater than in marine environments. At a specific concentration, AgNPs caused a change in cell width, a decrease in chlorophyll-a content, and an increase in total lipid synthesis in the studied microalgae. The morphological properties of microalgae may be utilized as an efficient method for monitoring NPs in water. Table 1 shows the Percentage of dissolved silver ions in different aquatic ecosystems (freshwater and marine water). It has been demonstrated that one of the main mechanisms of toxicity of AgNPs is related to Ag dissolved from AgNPs, which in both media has a relationship with the size and exposed surface of the AgNPs [24,37]. The dissolution of silver ions from AgNPs and their ionic membrane potential are discussed in Table 1. The dissolution of silver ions from AgNPs in freshwater is 1.8–4% ppb (20 nm), 24.5 (40 nm), 75 (30 nm), 27 (42 nm), and <0.1% (100 nm), and in fresh marine water 35–95% ppb (20 nm) and 0.38–1.25% ppb (100 nm).

Table 1. Percentage of dissolved silver ions in different aquatic ecosystems (freshwater and marine water).

Nanoparticles	Size (nm)	Dissolution of Silver Ions from AgNPs over Time (ppb)			Ionic Membrane Potential (mV)		
Type of Nanoparticles	Size of Nanoparticles	Time (h)	Freshwater (%)	Marine Water (%)	Freshwater	Marine Water	References
AgNPs	40	0–24	24.5	30–38	−25.3	16.5 ± 2	[27]
AgNPs	20	0–48	1.8–4	35–95	−17.5	−6.5 ± 1	[37]
AgNPs	30	0–48	118	15–30	−19.6	−15.8 ± 2	[30]
AgNPs	50	0–48	0.3–1.4	14–23	−16.2	−5.6 ± 2	[37]
AgNPs	30	0–48	75	32.38	−32.5	−11.7 ± 1	[24]
AgNPs	42	0–72	45	20–38	−17.8	−15.8 ± 1	[29]
AgNPs	100	0–48	<0.1	0.38–1.25	−21.7	−12.6 ± 2	[37]
AgNPs	20	0–96	200	35–45	−23.4	−19.4 ± 1	[35]
AgNPs	42	0–48	27	40–55	−16.5	−2.5 ± 2	[6]

3. Uptake and Bioaccumulation of AgNPs

3.1. Bioaccumulation of AgNPs in Aquatic Organis

Bioaccumulation is a primary method for determining the risks and hazards of AgNPs. Hazard evaluation needs analysis of all consequences and exposure. The xenobiotic consequences of bioaccumulation and exposure are frequently a condition for toxicity because the organism must preserve the chemical until it can show toxic behavior. Bioaccumulation is the simple method of evaluating the developments that affect bioavailability, the concentration of pollutants an organism absorbs from the ecological medium [38].

The structural and physicochemical features of AgNPs reflect the particles' ability to remain suspended in a solution that is isolated from the dissolved organic matter and microbes due to their interaction with aquatic biota [33,34]. The literature review we observed about the uptake and bioaccumulation of AgNPs in living organisms is provided in Table 2. Algal cells can accumulate NPs due to the permeability of NPs to the outer cell wall. Adsorption (or connection) of AgNPs to zooplankton is believed to include respiratory epithelium, appendages, digestive tract epithelium, and exoskeleton, and these NPs usually bioaccumulate in the consumers of zooplankton. Isolated hepatopancreatic digestive cells from mussels were seen to endocytose NPs, while invaginated cell membrane particles were produced in the lysosomal degradative compartment of cells [39]. Almost 70% of the AgNPs contained in Daphnia are obtained through food consumption. Silver ion makes zooplankton sick when they eat algae that have been exposed to AgNP [40].

About 63 tons of nano-silver are annually projected to flow into stream water bodies throughout the globe, with concentrations in aquatic environments predicted to range from 0.03 to 0.32 micrograms per liter [41]. Although the extra consumption of AgNPs released

into the aquatic environment will rise in the coming years. It is estimated that the predicted environmental concentrations (PECs) for AgNPs in surface water ranged from 0.088 to 10,000 ng/L [42]. AgNPs provide an excessive amount of attention in the aquatic ecosystem because ionic silver is considered to be one of the most harmful metals for aquatic life (prokaryotes, small fishes, marine and freshwater invertebrates), where the range of lethal concentration is low, like 1.25–10 µg/L (see Table 2) [43,44].

Mechanisms of Bioaccumulation

In aquatic organisms such as fishes, the NPs enter across gills or external surface epithelia [61]. The NPs enter the cell through the cell membrane at the cellular level. The plasma membrane has a complex system that allows selective substances into the cells; the translocation of substances inside the cell membrane occurs through pores and protein carriers. NPs are transported inside the plasma membrane through the invagination and vesicle formation that enclose material and transport these vesicles inside the membrane, whereas other translocating NPs enter via endocytosis [62]. If the uptake of AgNPs is prevailed by endocytosis, producing a nano environment of silver ions concentration becomes favorable for fast ion absorption [63]. These NPs can generate reactive oxygen species (ROS) that cause damage to cells by attacking membrane, protein, and DNA [64].

3.2. Ecological and Biological Toxic Effects of AgNPs

The toxicity of AgNPs directly correlates with their alteration within ecological and biological media and the release of silver ions with surface oxidation [65,66]. Silver ions are highly susceptible to bioconcentration in organisms as they are consistent with chemical reactions through cell membrane transporters of ions [67]. In aquatic invertebrates, some evidence suggests that silver ions increase toxicity if it is present in NPs due to subsequent release as ionic silver ions from NPs [68]. Studies demonstrated that AgNPs are less harmful than silver ions, remaining in the ionic form [69,70].

Various studies reported that AgNPs conjugated with the protein of the membrane and trigger signaling pathways, which leads to inhibition and proliferation of cells [71,72]. AgNPs often reach inside the cell through endocytosis or diffusion to induce mitochondrial disruption, producing ROS that damage proteins and inhibit cell proliferation [73,74]. Oxidative stress as ROS production reaches the capacities of antioxidant cellular defense mechanisms [75]. Oxidative disruption is always concerned with the degradation of glutathione, sulfhydryl group-containing protein, and improvements in the functioning of multiple antioxidant enzymes [76]. The toxicity significance for AgNPs is the association of nano and ionic silver with sulphur comprising macromolecules like proteins [77,78].

Algae play a vital role in all aquatic ecosystems and produce oxygen in aquatic habitats; thus, AgNPs pollution may severely impact algae functions [79]. NPs often cause harm to multicellular organisms via their respiratory system or skin, while unicellular organisms such as microalgae may be harmed generally by NPs [80]. AgNP toxicity, includes the collapse of proton pumps, membrane adhesion, increased cell porosity, DNA damage, inactivation of proteins and enzymes, destruction of lipopolysaccharide molecules, ribosome denaturation, production of ROS, and suppression of DNA synthesis [81,82].

Table 2. Uptake concentration of AgNPs by aquatic organisms.

Aquatic Organisms	Isolated Strains	NP Size (nm)	Concentration Range	Duration	Uptake Endpoints	Reference
Diatom	*Thalassiosira weissflogii*	10	10 mg/L	48 h	Cellular distribution	[45]
Aquatic bacterium	*Pseudomonas fluorescens*	30–50	0.002–2 mg/L	24 h	Aggregation by nanoscale film formation	[9]
Eastern mud snails, Juvenile hard clams, Grass shrimp, Cordgrass, Biofilms	*Vibrio harveyi*	20–80	1.62 mg/L	60 d	Bioaccumulation and trophic transfer	[34]
Nematode	*Caenorhabditis elegans*	<100	up to 0.5 mg/L	24 h	Uptake/adsorption to body	[46]
		7–25	5–50 mg/L	24 h	Uptake/transgenerational transfer to body	[47]
Earthworm	*Eisenia fetida*	30–50	10–20 mg/L	28 d	Bioaccumulation in a concentration-dependent manner	[48]
		10–50	<0.1 mg/L	48 h	Unpredictable	[48]
		29–39	1 mg/L	28 d	Possible body distribution	[49]
Water flea	*Daphnia magna*	40–50	up to 5 mg/L	8 h	Uptake and bioaccumulation	[40]
Zebrafish embryos	*Danio rerio*	5–15	1.62 mg/L	120 h	Uptake in embryos through chorion pore canals	[50]
		11.3	0.48 mg/L	21 h	Adsorption to embryos	[51]
		20–30	1×10^{-8}–2×10^{-8} mg/L	24 h	Penetrated skin and blood tube as aggregated particles	[52]
		20–30	0–4 mg/L	10 d	Bioaccumulation in muscle and intestine	[53]
		20–30	10 mg/L	48 h	Possible body uptake	[54]
Eurasian perch	*Perca fluviatilis*	30–40	0.000063–0.0003 mg/L	25 h	Possible to adsorb into gill	[55]
Rainbow trout	*Oncorhynchus mykiss*	5–15	10–20 mg/L	48 h	Cellular compartmentalization, transport over epithelial layers	[56]
Japanese medaka	*Oryzias latipes*	30–50	20 mg/L	7 d	Bioaccumulation in liver and gill	[57]
Zucchini	*Cucurbita pepo*	100	1000 mg/L	12 d	Translocation through shoots	[58]
Thale cress	*Arabidopsis thaliana*	20–80	10–20 mg/L		Uptake and accumulation of roots	[59]
Common grass	*Lolium multiflorum*	6–25	0–40 mg/L	24 h	Uptake into roots and shoots	[60]

Daphnia magna died after exposure to AR-AgNPs and silver ions, and the higher the concentration, the higher the death rate. The 48-h LC_{50} of AR-AgNPs and silver ions to daphnids was 1.86 ± 0.12 and 1.30 ± 0.07 mg/L, respectively, suggesting silver ions' higher toxicity in Daphnia, leading to mortality than AR-AgNPs. After 24 h of acute toxicity testing and removing dead zebrafish from a medium containing AR-AgNPs, two distinct abnormalities in the deceased zebrafish were identified. Ascites (fluid build-up in the belly) and colour alteration (fading colour) were among the abnormalities seen in fish exposed to 25 mg/L of AR-AgNPs [83]. This added to the evidence that AR-AgNPs had a negative impact on zebrafish development. The toxicity of AgNPs to aquatic species is typically caused by the release of free Ag ions [84]. The toxicity of silver ions to organisms may be attributed to ion regulatory disruption and competitive suppression of potassium or sodium ion-dependent adenosine triphosphate (Na+, K+-ATPase), which hindered absorption of Na+ by Daphnia. When AgNPs get into living cells, they may cause ROS and oxidative stress, which can lead to detrimental consequences such as membrane lipid peroxidation, mitochondrial damage, DNA damage, and cell death [85].

3.3. Toxicity of AgNPs on Algal Cells

The cytotoxic effects of AgNPs are based on their size, environmental influences, concentration, exposure time, and other cell properties. The cell wall in algae is a binding site for any reciprocal action with AgNPs since it serves as a barrier to the entry of the AgNPs from the surroundings. The majority of the algal cell wall is composed of proteins, carbohydrates, and cellulose (polysaccharides and glycoproteins) [86]. The algal cell wall acts as a semi-permeable sieve and also filters the large NPs via a smaller particle transition. The smaller size and broader surface area of the AgNPs make it possible to pass through the cell wall pores to the plasma membrane [78]. Cell reproduction may affect the permeability of cell walls, and newly manufactured holes may become more permeable to AgNPs. It was observed that newly formed holes are more prominent than previous ones owing to the effect of AgNPs on algal cells that may lead to an increase in nano-silver absorption in algal cells [87].

Some recent studies have shown the toxicity concerns of AgNPs. AgNPs are incredibly toxic in in-vitro studies at concentrations of 5–10 μg/L and particle sizes of 10–100 nm [88,89]. The surface coating agents of AgNPs like amino acids, sodium dodecyl sulfate, citric acid, and acetyl trimethyl ammonium bromide are non-covalently bound to AgNPs and discharged into the biological and environmental medium [90,91]. The formation of silver oxide through surface oxidation of AgNPs and silver oxide discharge into silver ions occurs in various biological and ecological media [92]. Due to this property, these particles enter the cell via passive and active transport and get assembled into silver oxide, causing mitochondrial dysfunction [93].

From previous studies, it can be inferred that AgNPs can enter the cell membrane, particularly inside the mitochondria. However, it is unclear whether nanomaterials can cause damage to the mitochondria across the cell or invade secondary organelles for oxidative impairment [94]. AgNPs can contact the cell membrane protein, triggering the cell signalling pathways to produce ROS, leading to protein and nucleic acid damage. The initiation of the strong attraction of Ag for sulfur of protein eventually causes apoptosis and inhibits cell proliferation [95]. The anti-proliferative action of AgNPs and the toxicity mechanism of AgNPs are shown in Figure 1. Wu and Zhou determined the toxicity of AgNPs to freshwater fish cell lines and their embryos [96]. This example is based on research work that used sub-chronic toxicity procedures to assess AgNPs bioaccumulation and its impact on adult medaka's histology and antioxidant protection mechanisms. Purified AgNPs were well dissolved in water, and after 14 days of exposure to the fish, a significant accumulation of Ag in the gills, liver, and intestinal tissues was observed. The functions of lactate dehydrogenase and antioxidant enzymes in the liver were dose-dependent. Reduction in glutathione and lipid peroxidation was dose-dependent both in the liver and gills. Exposure to AgNPs also caused histological lesions in fish tissues. Toxicological

endpoints and metal transport analysis showed that AgNPs caused tissue-specific toxicity, especially in the liver.

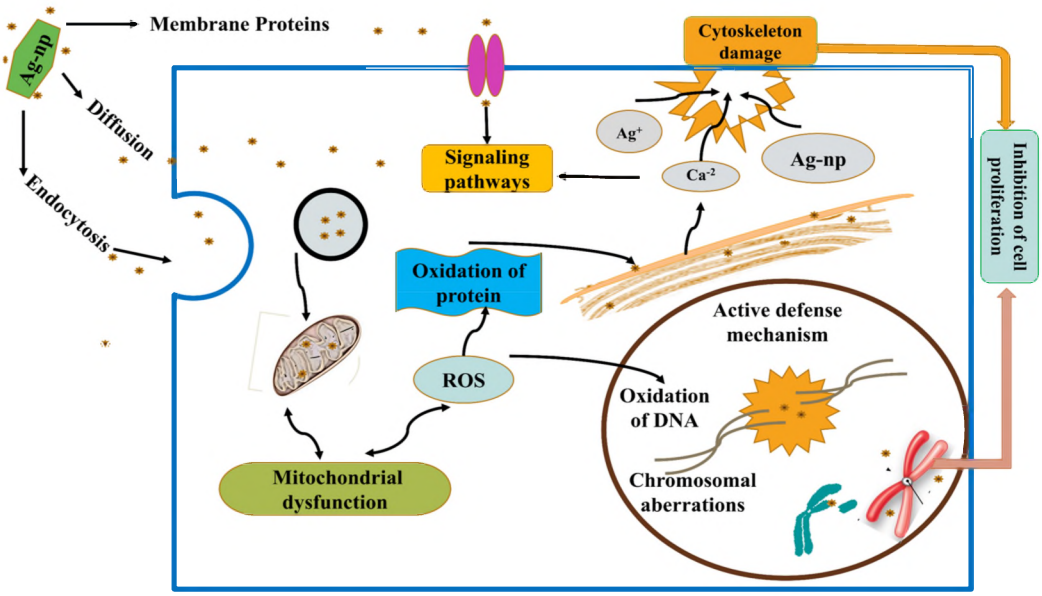

Figure 1. Proposed mechanism of AgNPs toxicity. This figure is reproduced from Asharani et al. [97].

3.4. Different Toxicological Pathways of AgNPs

Cell death mechanisms are broadly classified into necrosis and apoptosis. Apoptosis is an uncontrolled cellular explosion that is dependent on the enzyme caspases, whereas necroptosis and autophagy are caspase-independent. Programmed cell death is a natural process in which cells commit suicide in respond to precise signals, including exogenous cell-damaging (infectious or physical) and endogenous tissue-specific mediators depending upon the critical physiological situation. The apoptosis mechanism consists of three pathways: intrinsic or mitochondrial-mediated, extrinsic or DR-mediated, and execution [98]. Mitochondrial-mediated apoptotic or intrinsic pathways elicited by intracellular and extracellular stress release cytochrome c from the inter-membrane space of mitochondria into the cytosol [99]. The extrinsic pathway is activated through apoptotic signals when extracellular ligands, including (TNF-related apoptosis ligand; TRAIL), (Fas ligand; Fas-L), (tumour necrosis factor ligand; TNF) are bonded to the extracellular domain of trans-membrane death receptors; DRs such as TRAIL, CD95/Apo-1/Fas, type 1 TNF/TNFR1 receptors [100]. The extrinsic apoptosis phase is well defined by ligands and receptor association models (TNF-α; TNFR) and (FasL; FasR). Death-inducing signalling complex, DISC is expressed when specific death-ligand is triggered [98]. In the execution pathway, the intrinsic and extrinsic pathways are converged at the same point of the execution phase, the final approach of apoptosis [99].

3.4.1. AgNPs Regulate Apoptotic Pathways

The AgNPs cause cytotoxicity in various routes by inducing apoptosis and cell death [101]. In-vitro induction of AgNPs increases reactive oxygen free radicals inside the cell, which initiates apoptotic signalling through PKB (Protein Kinase B), TP53 (Tumor Protein 53) [102]. Overproduction of ROS causes PKB down-regulation and enhances p38 mitogen-activated protein kinase (p38 MAPK. Meanwhile, poly ADP ribose polymerase (PARP) declines in response to significant changes in caspase-3 and P53 proteins [103]. Thus, AgNPs can persuade apoptosis via subsequent TP53 signalling pathways (Figure 2).

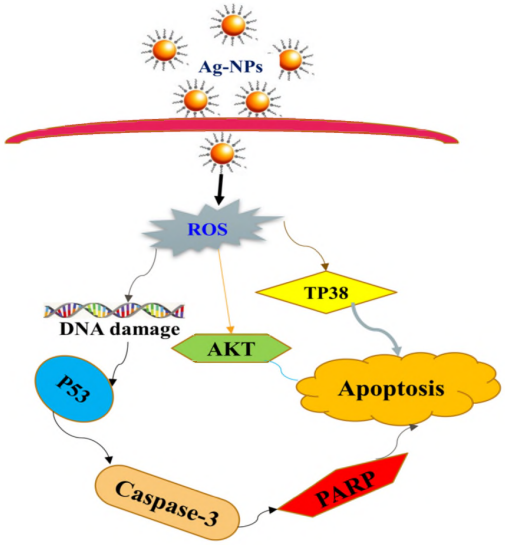

Figure 2. Apoptosis induced by tumor protein 53 (TP53) mediated signalling pathway, protein kinase B (PKB), p38 mitogen-activated protein kinase (p38 MAPK) activation suppresses the ROS produced by AgNPs. This figure is reproduced/modified from Li et al.; Akter et al. [103,104].

3.4.2. Mechanism of Apoptosis by AgNPs

The mitochondria are an important center of the apoptosis signal. AgNPs use mitochondria for signalling apoptosis. AgNPs affect the mitochondrial membrane's permeability, which damages the mitochondria's integrity, thus initiating apoptosis based on Jun amino-terminal kinases (JNK) facilitating cysteine-dependent aspartate specific-protease [105,106]. Loss of mitochondrial membrane potential causes B cell lymphoma-2 (BCL-2) down-regulation, pro-apoptotic Bax protein (BAX) upregulation, and discharge of cytochrome C inside the cytosol. JNK may affect the downregulation of Bcl-2 by phosphorylation. The cascade caused by cytochrome C is released inside the cytosol, leading to cysteine-dependent aspartate specific-protease (caspase 3) initiated via apoptotic protease activating factor-1 (APAF-1) and cysteine-dependent aspartate specific-protease (caspase 9) [107]. Thus, AgNPs can persuade apoptosis through the JNK-mediated mitochondrial and caspase-dependent pathways (Figure 3).

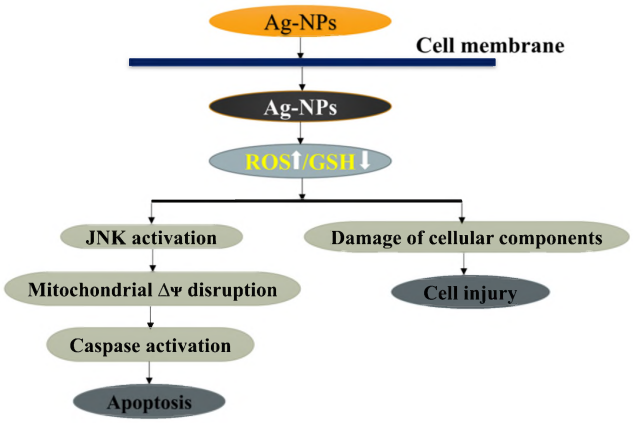

Figure 3. An anticipated pathway of AgNPs generates ROS production of intracellular glutathione (GSH) reduction, impairment to the cellular components, and eventually cell apoptosis. This figure is reproduced/modified from Akter et al.; Piao et al. [104,106].

3.4.3. Trojan-Horse Mechanism

In 2007, Limbach and colleagues proposed the Trojan-horse mechanism [108], further expanding as potential bioavailability and toxicity alterations linked to NP-pollutant as-

sociations [109]. The so-called "Trojan-horse" mechanism, in which NPs are incorporated inside cells and subsequently release large amounts of hazardous ions, has been suggested as a characteristic in AgNP cellular absorption [110]. Su et al. [111] investigated that Trojan horse greatly impacts several biochemical pathways involving substances adsorbed on NPs and present in cells simultaneously.

Interestingly, bacterial membranes often have negative charges that bind cationic silver but not AgNPs. As a result, silver ions may readily bind to bacterial membranes and produce a more severe toxicological reaction. Furthermore, larger AgNPs may survive in the environment; therefore, a Trojan-horse process transforms AgNPs into free silver ions pools, which can continually release silver ions [110]. In most cases, silver ions toxicity is caused by binding to proteins/peptides or DNA, inhibiting intracellular signal transmission. However, oxidative stress may be possible depending on the superoxide intermediates found during the oxidative dissolution processes [112]. Researchers found that O_2^- is more likely generated in the oxidative process of AgNPs with oxygen [113].

For example, RAW 265 cells phagocytose AgNPs; both in culture medium and cytosol, active cells are accessible rather than damaged cells. Trojan-horse mechanism promoted the discharge of NPs from the damaged cell [104]. The disappearance of AgNPs across the scratched cells proposes that NPs inside the cell ionize and cause cell damage. AgNPs phagocytosis can produce ROS, which stimulates inflammatory TNF-α (Tumor Necrosis Factor-alpha) signals. The cell membrane damage and apoptosis are due to the rising level of TNF-α. So, the hypothesis triggering the ionization of AgNPs across the cell is stated as a process of Trojan-horse mechanism [114].

Navarro and colleagues found that AgNPs' environmental toxicity is greatly influenced by their size and shape and the Trojan-horse mechanism, which promotes the release of silver ions within cells [115]. Smaller AgNPs offer higher antibacterial/antifungal effectiveness than bigger AgNPs [116]. Several studies concluded that the AgNPs were more hazardous to nitrifying bacteria than silver ions or silver chloride colloids [13,117,118]. These findings support the Trojan horse mechanism, though the authors hypothesized that AgNPs could attach to the outside cell membranes and induce oxidative stress without compromising the membrane.

Moreover, silver ion release is a significant toxicological mechanism for AgNPs in the environment because toxicity has been found mainly in the aqueous phase and is related to the levels of free silver ions [119]. When distributed in aquatic environments, ionic silver is highly hazardous to some species, including bacteria, phytoplankton, and fish [120]. It is believed that the ion's toxicity stems from its attraction to thiols (HS-), which are found in proteins and enzymes [121]. Similarly, studies indicate that when sulfide and thiosulfate are present in water to bind with silver ions, their toxicity to microorganisms considerably decreases because silver is no longer accessible [121,122]. The Trojan-horse effect has been proposed as the mechanism for inhalation toxicity of AgNPs (Figure 4).

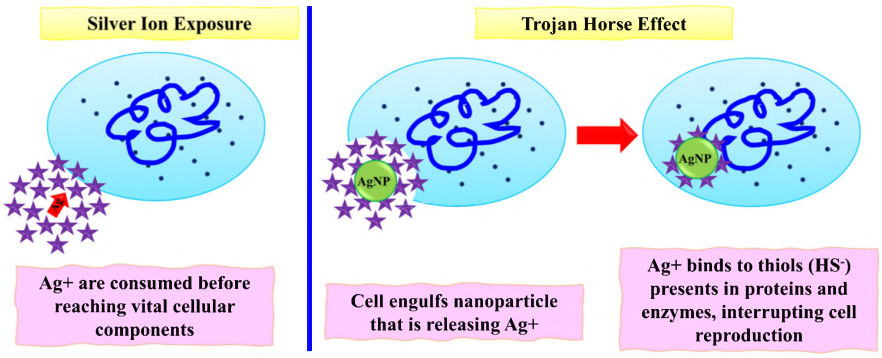

Figure 4. Silver ion exposure vs. Trojan-horse effect. This figure is reproduced/modified from Quadros and Marr [123].

4. Pharmacological Activities of AgNPs

4.1. Antimicrobial Activities of AgNPs

AgNPs possess excellent pharmacological activities against bacteria, fungi, and yeasts in aquatic groups of organisms. A study conducted by Moustafa et al. [124] investigated the antibacterial potential of AgNPs in marine organisms. They reported that these NPs showed excellent antibacterial properties against *S. agalactiae* and *V. alginolyticu* [124]. Another study conducted by Ghetas et al. [125] investigated that AgNPs possess antibacterial action against *S. agalactiae*, *A. hydrophila*, *V. alginolyticus*, and antifungal action against *F. moniliform*, and *C. albicans* [125]. AgNPs also possess antiviral activities in some aquatic organisms. White spot syndrome and hepatopancreatic parvoviruses are common viruses in aquatic organisms. A study conducted by Quinonez et al. [126] showed that a 1000 ng dose significantly reduced mortality by up to 50% and thus a potential role in controlling the White spot syndrome in striped harlequin, bumblebee red cherry shrimps. AgNPs also showed interaction with the envelope of hepatopancreatic parvoviruses and inhibited their viral replication in *A. japonicus* and *L. vannamei* [127].

Some recent studies have been reported about the antibacterial action of AgNPs in aquatic organisms. The larger surface area of AgNPs shows strong interaction with the biological membranes of microorganisms [128]. Therefore, these particles attach to the bacterial surface and enter the cell membrane due to their small size. It is also described as highly poisonous to bacterial species. Their antibacterial potency can be improved [129]. Free radical production mainly targets the membrane lipids in the living organism, occurs with dissociation and disruption, and ultimately inhibits microorganism growth [130]. The same mass of silver ions and AgNPs show an equivalent inhibition of bacteria, *Staphylococcus aureus*, and *Escherichia coli* [131]. The silver ions are infused through the cell wall into bacteria; due to this cell wall breakdown, the cells' protein is denatured, and the organisms die [132,133]. The silver ions are small and positively charged and freely communicate negatively charged biomolecules in the bacterial cell wall [134,135].

Silver ions discharged from AgNPs and entered across the bacterial cell-like protein and peptidoglycan constituents, preventing them from additional replication [136]. Discharge of silver ions occurs through oxidizing agents, which oxidize the elemental silver and convert them into toxic forms. Organic groups such as protein and carbonyl inside bacteria's cell walls are donors of electrons instead of electrons acceptors. The silver atom cannot generate silver ions. Therefore, the production of silver ions approves oxidizing agents [137]. Rai et al. [138] have noted how AgNPs correlated with the *E. coli* cell wall, which interfered with all membrane sides, dissolved with the release of silver ions into the cell, and affected the transcriptional response. They also illustrated a new dimension of the target microorganism species' significance, as the antibacterial behavior of NPs often relies on the target microorganism species. Antibacterial activity is explained stepwise in the later section: (1) NPs attract electrostatically [139], (2) generation of the free radicals, permeability changes, respiration disturbance, intracellular contents linkage [140], (3) modulation of protein phosphotyrosine profiles, activated in the development of the cell cycle and the synthesis of capsular polysaccharides [141], (4) associations with SH-groups; synthesis of protein and its function inhibited [142], and (5) interact with DNA-phosphorus that contain phosphorus (Figure 5) [143,144].

4.2. Chronic and Acute Toxicity Effects of AgNPs

AgNPs influence the cellular processes in living organisms and increase the production of reactive species in aquatic organisms in both in vitro and in vivo. These reactive species disrupted mitochondrial DNA activity and lipid peroxidation, stopping embryonic development and reproduction [146]. When AgNPs are exposed at high concentrations by two folds, even with a slow time of exposure, the survival of organisms is reduced abruptly [147,148]. The generally established explanation for this phenomenon leads to silver ion blockage of the Na^+, K^+-ATPase and inhibits the incorporation of Na ions via the gill membranes. It triggers the failure of ion regulation and eventually leads to the

organism's death [149]. The acute toxicity of AgNP exposure has both a direct and indirect effect. ROS generation, protein denaturation, membrane deformation, and DNA disruption are all direct effects of AgNP free radicals. In contrast, the indirect effect includes the discharge of Ag ions from the AgNP suspension [150].

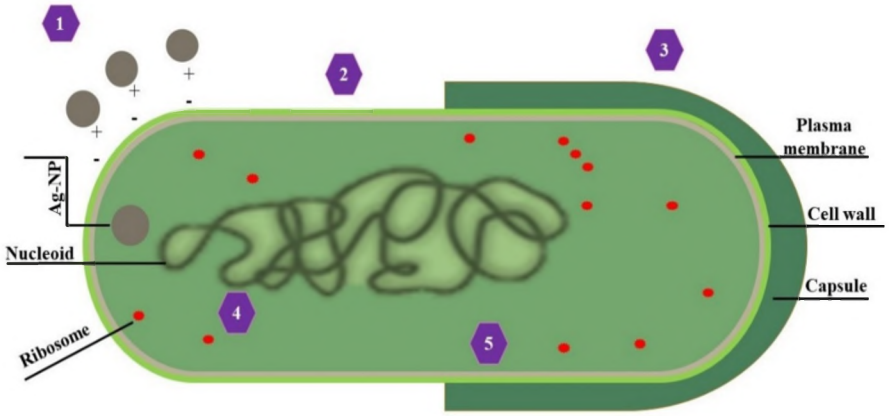

Figure 5. AgNPs mechanism proposed for antibacterial activity. This figure is reproduced/modified from Cui et al. [145].

Chronic toxicity tests utilizing minimal doses of exposure (ppb) and spanning the whole life cycle are required to accurately determine the toxicity of AgNPs in the aquatic environment. The chronic toxicity of AgNPs involves lipid peroxidation and oxidative stress caused by these NPs as free radicals [151]. The supply of food and the hurdles of purifying these NPs form the gut lines of the aquatic organisms [152]. In the chronic toxicity experiments, the waterborne AgNPs substantially reduce the proliferation of daphnids at a low concentration, i.e., 5 mg/L, suggesting that AgNPs provoke chronic toxicity in animals. Daphnid replication significantly decreased under the borne AgNP exposure when the algae were loaded with 0.1 mg/L AgNPs, far lower than the existing freshwater requirements [153]. AgNPs are affecting the organism's life in the aquatic ecosystem, as some of them are described in Table 3.

Many microorganisms, including bacteria, fungi, algae, and protozoa, are used as bioremediation to reduce the toxicity of inorganic and heavy metals like cobalt (Co), lead (Pb), copper (Cu), chromium (Cr), nickel (Ni), and zinc (Zn), which contaminate the aquatic ecosystem. Bacterial species of *Cellulosi microbium*, *Pseudomonas*, *Staphylococcus*, and *Enterobacter cloacae* [172] and fungal species like *Aspergillus niger*, *Aspergillus vesicolor*, *Phanerochaete chrysosporium*, *Sphaerotilus natans*, *Saccharomyces cerevisiae*, and *Gloeophyllum sepiarium* are used to minimize the toxicity effect [173,174]. Similarly, many species of algae; *Chlorella vulgaris* [175], *Spirogyra*, *Spirulina*, *Nostoc* sp., [176] and protozoa; *Tetrahymena rostrata* are also reported to detoxify the heavy metal concentration in the aquatic ecosystem [177]. The toxicity of silver particles is a severe threat to all living organisms in the aquatic ecosystem. There is also a need for urgent attention to establishing the biological control and bioremediation of AgNPs to reduce the silver toxicity through microorganisms (bacteria, fungi, protozoa) and algae to save the ecosystem's living aquatic life. However, from literature databases, we found a minimal number of studies published providing little detail on overcoming excessive bioaccumulation in an aquatic ecosystem. *Chromobacterium violaceum* is used for bioremediation purposes. They found that bacteria efficiently absorbed AgNPs released during cloth washing [178]. The morphological changes were observed in bacteria upon uptake of AgNPs. However, after subsequent culture, the original shape was restored to it.

Table 3. AgNPs toxicity and effects on different aquatic life.

Groups	Role of AgNPs in Toxicity/Malfunctioning of Cells/Organ/Organisms	Example	Reference
Protozoa	Ag ions destroy the sporozoites by entering the oocyst and ultimately break the oocyst wall	*Cryptosporidium parvum*	[154]
	The effects of protein-coated AgNPs (14.6 nm, Collargol) have shown in the viability, oxidative stress, and gene expression levels of ciliates species	*Tetrahymena thermophila*	[155]
Monera	AgNPs are highly toxic to bacteria, often associated with ion release and induction of oxidative stress. AgNPs serve as an antibacterial against bacterial tension and thus avoids its horrendous impact	Bacteria	[156,157]
	Inhibition of bacterial growth increased permeability due to the formation of "pits"	*Escherichia coli*	[134]
	The interaction of the bacterial cell with AgNPs causes Proton Motive Force dissipation leading to the death of the cell	*Staphylococcus aureus*	[158]
	Generation of ROS	Autotrophic nitrifying bacteria	[147]
	AgNPs caused toxicity in the membrane when they attached with less than ten nm-sized NPs	*Salmonella typhi*, *Pseudomonas aeruginosa* and *Vibrio cholera*	[159]
Fungi	AgNPs show antifungal activity, which suppresses the growth of fungal cells	*Aspergillus* sp., *Rhizoctonia solani*, *Sclerotinia sclerotiorum*, *S. minor*	[160]
Plant	AgNPs changed/inhibited seed's germination, the surface area of leaf, morphology, biomass, and growth potential	*Spirodela polyrhiza*	[161]
	Metabolic disorders arise, foliar proline accumulation is caused by a decrease in the contents of sugar. Total protein and chlorophyll, elongation of shoots and roots become reduced	*Lupinus termis*	[162]
	Repressed down-regulated induction of auxin receptor-related genetics, gravitropism of root, and reduction in root tips accumulation of auxins	*Arabidopsis thaliana*	[163]
	DNA damages when cytotoxicity enhances at lethal concentration; LC_{58}, i.e., up to 10 mg/L	*Allium cepa*	[164]
Animals	Apoptosis occurs when AgNPs directly contact the intestinal epithelium. In specific, typhlosole wherein the apoptosis impaired chloragogenous cells have a role like that of the liver invertebrate species or tissue in molluscs and arthropods	Oligochaetes, vertebrates, molluscs, arthropoda	[165]
	Acute toxicity/cause immobilization	*Daphnia magna*	[152]

Table 3. *Cont.*

Groups	Role of AgNPs in Toxicity/Malfunctioning of Cells/Organ/Organisms	Example	Reference
Algae	AgNPs increase toxicity and disrupts the Photosynthetic system, cell metabolism, and cell membrane. The percentage of overall NPs absorbed by algae cells was 21% and 31%, respectively, for both species	*Chlorella vulgaris*, *Raphidocelis subcapitata*	[166]
	AgNPs cause inhibitory effects on algae species	*Chlorella vulgaris Dunaliella tertiolecta*	[79]
	Chronic toxicity/Growth inhibition	*Euglena gracilis*	[167]
	Chronic toxicity/Growth inhibition	*Chlamydomonas reinhardtii*	[167]
Fishes	AgNPs induce changes in haematology parameters such as the mean corpuscular haemoglobin (MCH) and mean corpuscular volume (MCV) become decreased. In contrast, red blood cells (RBCs) and white blood cells (WBCs) become increased as the concentration (Conc.) of AgNPs increased	Rainbow trout (*Oncorhynchus mykiss)*	[154]
	AgNPs decreased the concentration of albumin (Al), globulin (Gl), and total proteins (Tp). In contrast, the concentration of alkaline phosphatase (ALP), Aspartic aminotransferase (AST), Glucose (Glu), Alanine aminotransferase (ALT), and total lipids (Tl) increases. At tissue and cell levels, pyknotic nuclei, proliferation of hepatocytes, cytoplasmic vacuolation, hepatic necrosis, central vein wall rupture, infiltrations of inflammatory cells, melanoma-macrophages aggregation, and apoptotic cells occurs in the liver of AgNPs-exposed fish	*Clarias gariepinus*	[168]
	Acute toxicity/Abnormality	*Oryzias latipes*	[167]
	Recent findings revealed that AgNPs had influenced the fish behaviour at the highest concentration (0.09 mg/L). The bioaccumulation AgNPs was found high in the liver, intestine, gills, and muscles. Moreover, the results revealed that at the highest concentration (0.09 mg/L), the bioaccumulation of AgNPs led to histopathological alterations, including gill damage leading to necrosis	*Cyprinus carpio*	[169]
	Acute toxicity/Abnormality in different functions	*Danio rerio*	[167]
Amphibians	The influence of AgNPs on stress and thyroid hormones is being studied with tadpole caudal fin cultures in vitro	*Lithobates catesbeianus*, *Rana catesbeiana*	[170,171]

5. Different Methods for Silver Ions Detection

At the nanomolar (nM) level, several techniques are employed for the detection of silver ions. Several ways of detecting silver ions depend on a mix of metal ion analysis and enzymatic or oxidative amplification mechanisms. It was identified and developed as a significant technique for increasing metal ion detection sensitivity. The biosensor technique has gotten a lot of interest because of its great sensitivity, short processing time, and ease of use [179]. These methods are based on nucleic acid interaction with metal ions in very low concentrations, which can be detected.

5.1. Biosensor

Silver ions could selectively connect via coordinating bonds with cytosine (C) molecules to form a strong C–Ag^+–C framework and transform single-stranded DNA into a double-helix structure. The C–Ag^+–C interaction is highly selective because the C–C mismatching interaction with silver ions is stronger than with other metal ions. It was observed that a colorimetric approach for silver ion detection was based on the interaction between methylene blue (MB) and C-rich single-stranded DNA (ssDNA). When the MB was absorbed onto the ssDNA surface, the MB color changed from blue to purple. However, in the presence of silver ions, the specific C–Ag^+–C pair is formed and removes the interaction between SSDNA and MB, returning to the blue color. Based on this feature, a new method for detecting silver ions by adding cysteine that removes base pairs C–Ag^+–C because it binds to silver ions instead of cytosine has been developed. The amount of free cysteine was critical for colorimetric detection using ABTS–H_2O_2 [180].

A new biosensor based on electro-chem-luminescence (ECL) of Ru (bpy) 2 (mcbpy-O-Su-ester) (PF6)2 for highly sensitive and selective silver ion detection. Based on deoxyribonucleic acid (DNA tetrahedron TS primer (STTS), this process consists of three hybridized oligonucleotides forming three dual-stranded DNAs, close to a Y-shaped DNA structure. The formation of DNA-TS makes the signal intensity change of Ru (bpy) 2 (mcbpy-O-Su-ester) (PF6) 2 at different concentrations of silver ions [181].

5.2. Chemical Sensor

The highly sensitive chemical colorimetric sensor approach for silver ions' detection in the picomolar (pM) range. This approach uses Pt nano-cubes coated with PVP as artificial peroxidases. The peroxidase substrates generate a colored signal that diminishes the existence of silver ions. This colorimetric approach will achieve an ultralow detection limit of 80 pM and a wide dynamic range of 102–104 nM. A colorimetric approach for silver ions' detection that measures the changes in SPR of modified AuNPs [182]. A colorimetric sensor based on ascorbic acid (AA) and AuNPs for silver ions' detection at concentrations of 2–28 μM in aqueous solutions. A nanocomposite membrane of Cu–(PAAc/PVA) by gamma radiation for Rapid Colorimetric Detection (RCD) of silver and mercury ions associated with significant changes of color of the Cu–(PAAc/PVA) membrane from yellow to dark green and pale gray color, respectively [183]. A carbon dot is another nonmetal ion used as a colorimetric sensor of silver ions. Carbon dots (CDs) have fluorescence emission properties that quench when their surface is attached to metal ions such as silver ions. The CDs have strong fluorescent emission at 479 nm when excited over 370 nm. This fluorescent emission was quenched by the existence of silver ions in an aqueous solution. Therefore, increased risks of AgNPs lead to health and environmental issues, cellular toxicities, and damage to the organisms in the aquatic system. It is urgently needed to discover novel detection methods that can control the increased discharge of heavy metals into human food items [184].

6. Conclusions

This review revealed the current significance of AgNPs as emerging environmental contaminants that can cause adverse effects on living tissues, damage vital organs, and pose a severe threat to aquatic life and their ecosystems. The widespread use of AgNPs results

in their unavoidable discharge into the ecosystem, which pollutes the aquatic environment. With every passing day, the accumulation of AgNPs increases in the biological system, creating cytotoxicity in the normal cells of aquatic life. The genotoxic and cytotoxic effects of AgNPs are based on their size, environmental influences, concentration, and exposure time. These NPs produce the reactive species in aquatic organisms that interrupt the functions of DNA, cause mitochondrial dysfunction, and increase lipid peroxidation, which terminates development and reproduction both in vivo and in vitro. These reactive species interrupt mitochondrial, DNA activity, and lipid peroxidation, stopping embryonic development and reproduction. This review will be helpful for drawing attention to establishing the biological control and bioremediation of AgNPs to reduce the silver toxicity through microorganisms, bacteria, fungi, protozoa, or algae to save the ecosystem and for living aquatic life. It will also be helpful for identifying the organisms that denature the toxic NPs effect so that its toxicity mechanisms can be reduced.

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References

1. Ahmad, A.; Mohd-Setapar, S.H.; Chuong, C.S.; Khatoon, A.; Wani, W.A.; Kumar, R.; Rafatullah, M. Recent advances in new generation dye removal technologies: Novel search for approaches to reprocess wastewater. *RSC Adv.* **2015**, *5*, 30801–30818. [CrossRef]
2. Vasistha, P.; Ganguly, R. Water quality assessment of natural lakes and its importance: An overview. *Mater. Today Proc.* **2020**, *32*, 544–552. [CrossRef]
3. Elbehiry, F.; Alshaal, T.; Elhawat, N.; Elbasiouny, H. Environmental-Friendly and Cost-Effective Agricultural Wastes for Heavy Metals and Toxicants Removal from Wastewater. In *The Handbook of Environmental Chemistry*; Springer: Berlin/Heidelberg, Germany, 2021.
4. Hullmann, A. *The Economic Development of Nanotechnology—An Indicators Based Analysis*; European Commission: Brussels, Belgium, 2006.
5. Klaine, S.J.; Alvarez, P.J.J.; Batley, G.E.; Fernandes, T.F.; Handy, R.D.; Lyon, D.Y.; Mahendra, S.; McLaughlin, M.J.; Lead, J.R. Nanomaterials in the environment: Behavior, fate, bioavailability, and effects. *Environ. Toxicol. Chem. Int. J.* **2008**, *27*, 1825–1851. [CrossRef]
6. Fabrega, J.; Luoma, S.N.; Tyler, C.R.; Galloway, T.S.; Lead, J.R. Silver nanoparticles: Behaviour and effects in the aquatic environment. *Environ. Int.* **2011**, *37*, 517–531. [CrossRef] [PubMed]
7. Xia, G.; Liu, T.; Wang, Z.; Hou, Y.; Dong, L.; Zhu, J.; Qi, J. The effect of silver nanoparticles on zebrafish embryonic development and toxicology. *Artif. Cells Nanomed. Biotechnol.* **2016**, *44*, 1116–1121. [CrossRef]
8. Vance, M.E.; Kuiken, T.; Vejerano, E.P.; McGinnis, S.P.; Hochella, M.F., Jr.; Rejeski, D.; Hull, M.S. Nanotechnology in the real world: Redeveloping the nanomaterial consumer products inventory. *Beilstein J. Nanotechnol.* **2015**, *6*, 1769–1780. [CrossRef] [PubMed]
9. Fabrega, J.; Zhang, R.; Renshaw, J.C.; Liu, W.-T.; Lead, J.R. Impact of silver nanoparticles on natural marine biofilm bacteria. *Chemosphere* **2011**, *85*, 961–966. [CrossRef] [PubMed]
10. Qamar, S.U.R.; Ahmad, J.N. Nanoparticles: Mechanism of biosynthesis using plant extracts, bacteria, fungi, and their applications. *J. Mol. Liq.* **2021**, *334*, 116040. [CrossRef]
11. Ottoni, C.A.; Simões, M.F.; Fernandes, S.; Dos Santos, J.G.; Da Silva, E.S.; de Souza, R.F.B.; Maiorano, A.E. Screening of filamentous fungi for antimicrobial silver nanoparticles synthesis. *AMB Express* **2017**, *7*, 1–10. [CrossRef]

12. Alexander, J.W. History of the medical use of silver. *Surg. Infect.* **2009**, *10*, 289–292. [CrossRef]
13. Fabrega, J.; Fawcett, S.R.; Renshaw, J.C.; Lead, J.R. Silver nanoparticle impact on bacterial growth: Effect of pH, concentration, and organic matter. *Environ. Sci. Technol.* **2009**, *43*, 7285–7290. [CrossRef] [PubMed]
14. McGillicuddy, E.; Murray, I.; Kavanagh, S.; Morrison, L.; Fogarty, A.; Cormican, M.; Dockery, P.; Prendergast, M.; Rowan, N.; Morris, D. Silver nanoparticles in the environment: Sources, detection and ecotoxicology. *Sci. Total Environ.* **2017**, *575*, 231–246. [CrossRef] [PubMed]
15. Christen, V.; Capelle, M.; Fent, K. Silver nanoparticles induce endoplasmatic reticulum stress response in zebrafish. *Toxicol. Appl. Pharmacol.* **2013**, *272*, 519–528. [CrossRef] [PubMed]
16. Keller, A.A.; McFerran, S.; Lazareva, A.; Suh, S. Global life cycle releases of engineered nanomaterials. *J. Nanopart. Res.* **2013**, *15*, 1–17. [CrossRef]
17. Caballero-Guzman, A.; Sun, T.; Nowack, B. Flows of engineered nanomaterials through the recycling process in Switzerland. *Waste Manag.* **2015**, *36*, 33–43. [CrossRef]
18. Ratte, H.T. Bioaccumulation and toxicity of silver compounds: A review. *Environ. Toxicol. Chem. Int. J.* **1999**, *18*, 89–108. [CrossRef]
19. Bianchini, A.; Wood, C.M. Mechanism of acute silver toxicity in Daphnia magna. *Environ. Toxicol. Chem. Int. J.* **2003**, *22*, 1361–1367. [CrossRef]
20. Kleiven, M.; Macken, A.; Oughton, D.H. Growth inhibition in Raphidocelis subcapita–Evidence of nanospecific toxicity of silver nanoparticles. *Chemosphere* **2019**, *221*, 785–792. [CrossRef]
21. Katuli, K.K.; Massarsky, A.; Hadadi, A.; Pourmehran, Z. Silver nanoparticles inhibit the gill Na+/K+-ATPase and erythrocyte AChE activities and induce the stress response in adult zebrafish (Danio rerio). *Ecotoxicol. Environ. Saf.* **2014**, *106*, 173–180. [CrossRef]
22. Brunetti, G.; Donner, E.; Laera, G.; Sekine, R.; Scheckel, K.G.; Khaksar, M.; Vasilev, K.; De Mastro, G.; Lombi, E. Fate of zinc and silver engineered nanoparticles in sewerage networks. *Water Res.* **2015**, *77*, 72–84. [CrossRef]
23. Pulit-Prociak, J.; Banach, M. Silver nanoparticles–a material of the future . . . ? *Open Chem.* **2016**, *14*, 76–91. [CrossRef]
24. Luoma, S.N. Silver nanotechnologies and the environment. *Proj. Emerg. Nanotechnol. Rep.* **2008**, *15*, 12–13.
25. Luoma, S.N.; Rainbow, P.S. *Metal Contamination in Aquatic Environments: Science and Lateral Management*; Cambridge University Press: Cambridge, UK, 2008; ISBN 0521860571.
26. Levard, C.; Hotze, E.M.; Lowry, G.V.; Brown, G.E., Jr. Environmental transformations of silver nanoparticles: Impact on stability and toxicity. *Environ. Sci. Technol.* **2012**, *46*, 6900–6914. [CrossRef] [PubMed]
27. Magesky, A.; Pelletier, É. Cytotoxicity and physiological effects of silver nanoparticles on marine invertebrates. *Cell. Mol. Toxicol. Nanopart.* **2018**, *1048*, 285–309.
28. Cozzari, M.; Elia, A.C.; Pacini, N.; Smith, B.D.; Boyle, D.; Rainbow, P.S.; Khan, F.R. Bioaccumulation and oxidative stress responses measured in the estuarine ragworm (Nereis diversicolor) exposed to dissolved, nano-and bulk-sized silver. *Environ. Pollut.* **2015**, *198*, 32–40. [CrossRef] [PubMed]
29. Lee, B.; Duong, C.N.; Cho, J.; Lee, J.; Kim, K.; Seo, Y.; Kim, P.; Choi, K.; Yoon, J. Toxicity of citrate-capped silver nanoparticles in common carp (Cyprinus carpio). *J. Biomed. Biotechnol.* **2012**, *2012*, 262670. [CrossRef]
30. Wang, H.; Ho, K.T.; Scheckel, K.G.; Wu, F.; Cantwell, M.G.; Katz, D.R.; Horowitz, D.B.; Boothman, W.S.; Burgess, R.M. Toxicity, bioaccumulation, and biotransformation of silver nanoparticles in marine organisms. *Environ. Sci. Technol.* **2014**, *48*, 13711–13717. [CrossRef]
31. Ribeiro, F.; Gallego-Urrea, J.A.; Goodhead, R.M.; Van Gestel, C.A.M.; Moger, J.; Soares, A.M.V.M.; Loureiro, S. Uptake and elimination kinetics of silver nanoparticles and silver nitrate by Raphidocelis subcapitata: The influence of silver behaviour in solution. *Nanotoxicology* **2015**, *9*, 686–695. [CrossRef]
32. Macken, A.; Byrne, H.J.; Thomas, K.V. Effects of salinity on the toxicity of ionic silver and Ag-PVP nanoparticles to Tisbe battagliai and Ceramium tenuicorne. *Ecotoxicol. Environ. Saf.* **2012**, *86*, 101–110. [CrossRef]
33. Thio, B.J.R.; Montes, M.O.; Mahmoud, M.A.; Lee, D.-W.; Zhou, D.; Keller, A.A. Mobility of capped silver nanoparticles under environmentally relevant conditions. *Environ. Sci. Technol.* **2012**, *46*, 6985–6991. [CrossRef]
34. Cleveland, D.; Long, S.E.; Pennington, P.L.; Cooper, E.; Fulton, M.H.; Scott, G.I.; Brewer, T.; Davis, J.; Petersen, E.J.; Wood, L. Pilot estuarine mesocosm study on the environmental fate of silver nanomaterials leached from consumer products. *Sci. Total Environ.* **2012**, *421*, 267–272. [CrossRef]
35. Blaser, S.A.; Scheringer, M.; MacLeod, M.; Hungerbühler, K. Estimation of cumulative aquatic exposure and risk due to silver: Contribution of nano-functionalized plastics and textiles. *Sci. Total Environ.* **2008**, *390*, 396–409. [CrossRef] [PubMed]
36. Pham, T.-L. Effect of silver nanoparticles on tropical freshwater and marine microalgae. *J. Chem.* **2019**, *2019*, 9658386. [CrossRef]
37. Mueller, N.C.; Nowack, B. Exposure modeling of engineered nanoparticles in the environment. *Environ. Sci. Technol.* **2008**, *42*, 4447–4453. [CrossRef] [PubMed]
38. Luoma, S.N.; Rainbow, P.S. Why is metal bioaccumulation so variable? Biodynamics as a unifying concept. *Environ. Sci. Technol.* **2005**, *39*, 1921–1931. [CrossRef] [PubMed]
39. Moore, A.; Weissleder, R.; Bogdanov, A., Jr. Uptake of dextran-coated monocrystalline iron oxides in tumor cells and macrophages. *J. Magn. Reson. Imaging* **1997**, *7*, 1140–1145. [CrossRef]
40. Zhao, C.-M.; Wang, W.-X. Biokinetic uptake and efflux of silver nanoparticles in Daphnia magna. *Environ. Sci. Technol.* **2010**, *44*, 7699–7704. [CrossRef]

41. Sohn, E.K.; Johari, S.A.; Kim, T.G.; Kim, J.K.; Kim, E.; Lee, J.H.; Chung, Y.S.; Yu, I.J. Aquatic toxicity comparison of silver nanoparticles and silver nanowires. *BioMed Res. Int.* **2015**, *2015*, 893049. [CrossRef]
42. Hartmann, S.; Beasley, A.; Mozhayeva, D.; Engelhard, C.; Witte, K. Defective defence in Daphnia daughters: Silver nanoparticles inhibit anti-predator defence in offspring but not in maternal Daphnia magna. *Sci. Rep.* **2020**, *10*, 8021. [CrossRef]
43. Farkas, J.; Christian, P.; Urrea, J.A.G.; Roos, N.; Hassellöv, M.; Tollefsen, K.E.; Thomas, K.V. Effects of silver and gold nanoparticles on rainbow trout (*Oncorhynchus mykiss*) hepatocytes. *Aquat. Toxicol.* **2010**, *96*, 44–52. [CrossRef]
44. Maurer-Jones, M.A.; Gunsolus, I.L.; Murphy, C.J.; Haynes, C.L. Toxicity of engineered nanoparticles in the environment. *Anal. Chem.* **2013**, *85*, 3036–3049. [CrossRef]
45. Miao, A.-J.; Schwehr, K.A.; Xu, C.; Zhang, S.-J.; Luo, Z.; Quigg, A.; Santschi, P.H. The algal toxicity of silver engineered nanoparticles and detoxification by exopolymeric substances. *Environ. Pollut.* **2009**, *157*, 3034–3041. [CrossRef]
46. Roh, J.; Sim, S.J.; Yi, J.; Park, K.; Chung, K.H.; Ryu, D.; Choi, J. Ecotoxicity of silver nanoparticles on the soil nematode Caenorhabditis elegans using functional ecotoxicogenomics. *Environ. Sci. Technol.* **2009**, *43*, 3933–3940. [CrossRef] [PubMed]
47. Meyer, J.N.; Lord, C.A.; Yang, X.Y.; Turner, E.A.; Badireddy, A.R.; Marinakos, S.M.; Chilkoti, A.; Wiesner, M.R.; Auffan, M. Intracellular uptake and associated toxicity of silver nanoparticles in Caenorhabditis elegans. *Aquat. Toxicol.* **2010**, *100*, 140–150. [CrossRef] [PubMed]
48. Shoults-Wilson, W.A.; Reinsch, B.C.; Tsyusko, O.V.; Bertsch, P.M.; Lowry, G.V.; Unrine, J.M. Effect of silver nanoparticle surface coating on bioaccumulation and reproductive toxicity in earthworms (Eisenia fetida). *Nanotoxicology* **2011**, *5*, 432–444. [CrossRef] [PubMed]
49. Heckmann, L.-H.; Hovgaard, M.B.; Sutherland, D.S.; Autrup, H.; Besenbacher, F.; Scott-Fordsmand, J.J. Limit-test toxicity screening of selected inorganic nanoparticles to the earthworm Eisenia fetida. *Ecotoxicology* **2011**, *20*, 226–233. [CrossRef] [PubMed]
50. Lee, K.J.; Nallathamby, P.D.; Browning, L.M.; Osgood, C.J.; Xu, X.-H.N. In vivo imaging of transport and biocompatibility of single silver nanoparticles in early development of zebrafish embryos. *ACS Nano* **2007**, *1*, 133–143. [CrossRef]
51. Nallathamby, P.D.; Lee, K.J.; Xu, X.-H.N. Design of stable and uniform single nanoparticle photonics for in vivo dynamics imaging of nanoenvironments of zebrafish embryonic fluids. *ACS Nano* **2008**, *2*, 1371–1380. [CrossRef]
52. Yeo, M.-K.; Kang, M.-S. Effects of nanometer sized silver materials on biological toxicity during zebrafish embryogenesis. *Bull. Korean Chem. Soc.* **2008**, *29*, 1179–1184.
53. Yeo, M.-K.; Pak, S.-W. Exposing zebrafish to silver nanoparticles during caudal fin regeneration disrupts caudal fin growth and p53 signaling. *Mol. Cell. Toxicol.* **2008**, *4*, 311–317.
54. Griffitt, R.J.; Luo, J.; Gao, J.; Bonzongo, J.; Barber, D.S. Effects of particle composition and species on toxicity of metallic nanomaterials in aquatic organisms. *Environ. Toxicol. Chem. Int. J.* **2008**, *27*, 1972–1978. [CrossRef]
55. Bilberg, K.; Malte, H.; Wang, T.; Baatrup, E. Silver nanoparticles and silver nitrate cause respiratory stress in Eurasian perch (Perca fluviatilis). *Aquat. Toxicol.* **2010**, *96*, 159–165. [CrossRef] [PubMed]
56. Farkas, J.; Christian, P.; Gallego-Urrea, J.A.; Roos, N.; Hassellöv, M.; Tollefsen, K.E.; Thomas, K.V. Uptake and effects of manufactured silver nanoparticles in rainbow trout (*Oncorhynchus mykiss*) gill cells. *Aquat. Toxicol.* **2011**, *101*, 117–125. [CrossRef] [PubMed]
57. Jung, Y.J.; Kim, J.Y.; Yang, S.Y.; Lee, B.G.; Kim, S.D. Bioaccumulation of silver nanoparticles in Oryzias latipes through aqueous exposure. *Korean Soc. Environ. Health Toxicol.* **2012**, *10*, 410–411.
58. Stampoulis, D.; Sinha, S.K.; White, J.C. Assay-dependent phytotoxicity of nanoparticles to plants. *Environ. Sci. Technol.* **2009**, *43*, 9473–9479. [CrossRef]
59. Ma, X.; Geiser-Lee, J.; Deng, Y.; Kolmakov, A. Interactions between engineered nanoparticles (ENPs) and plants: Phytotoxicity, uptake and accumulation. *Sci. Total Environ.* **2010**, *408*, 3053–3061. [CrossRef] [PubMed]
60. Yin, L.; Cheng, Y.; Espinasse, B.; Colman, B.P.; Auffan, M.; Wiesner, M.; Rose, J.; Liu, J.; Bernhardt, E.S. More than the ions: The effects of silver nanoparticles on Lolium multiflorum. *Environ. Sci. Technol.* **2011**, *45*, 2360–2367. [CrossRef]
61. Oberdörster, E. Manufactured nanomaterials (fullerenes, C60) induce oxidative stress in the brain of juvenile largemouth bass. *Environ. Health Perspect.* **2004**, *112*, 1058–1062. [CrossRef]
62. Moore, M.N. Do nanoparticles present ecotoxicological risks for the health of the aquatic environment? *Environ. Int.* **2006**, *32*, 967–976. [CrossRef]
63. Liu, J.; Hurt, R.H. Ion release kinetics and particle persistence in aqueous nano-silver colloids. *Environ. Sci. Technol.* **2010**, *44*, 2169–2175. [CrossRef]
64. Brown, D.M.; Wilson, M.R.; MacNee, W.; Stone, V.; Donaldson, K. Size-dependent proinflammatory effects of ultrafine polystyrene particles: A role for surface area and oxidative stress in the enhanced activity of ultrafines. *Toxicol. Appl. Pharmacol.* **2001**, *175*, 191–199. [CrossRef] [PubMed]
65. Reidy, B.; Haase, A.; Luch, A.; Dawson, K.A.; Lynch, I. Mechanisms of silver nanoparticle release, transformation and toxicity: A critical review of current knowledge and recommendations for future studies and applications. *Materials* **2013**, *6*, 2295–2350. [CrossRef] [PubMed]
66. Sharma, V.K. Stability and toxicity of silver nanoparticles in aquatic environment: A review. *Sustain. Nanotechnol. Environ. Adv. Achiev.* **2013**, *1124*, 165–179.

67. Zhang, L.; Wang, W.-X. Dominant role of silver ions in silver nanoparticle toxicity to a unicellular alga: Evidence from luminogen imaging. *Environ. Sci. Technol.* **2018**, *53*, 494–502. [CrossRef]
68. Huang, Z.; Zeng, Z.; Chen, A.; Zeng, G.; Xiao, R.; Xu, P.; He, K.; Song, Z.; Hu, L.; Peng, M. Differential behaviors of silver nanoparticles and silver ions towards cysteine: Bioremediation and toxicity to Phanerochaete chrysosporium. *Chemosphere* **2018**, *203*, 199–208. [CrossRef]
69. Abramenko, N.B.; Demidova, T.B.; Abkhalimov, E.V.; Ershov, B.G.; Krysanov, E.Y.; Kustov, L.M. Ecotoxicity of different-shaped silver nanoparticles: Case of zebrafish embryos. *J. Hazard. Mater.* **2018**, *347*, 89–94. [CrossRef]
70. Kvitek, L.; Vanickova, M.; Panacek, A.; Soukupova, J.; Dittrich, M.; Valentova, E.; Prucek, R.; Bancirova, M.; Milde, D.; Zboril, R. Initial study on the toxicity of silver nanoparticles (NPs) against Paramecium caudatum. *J. Phys. Chem. C* **2009**, *113*, 4296–4300. [CrossRef]
71. Braydich-Stolle, L.K.; Lucas, B.; Schrand, A.; Murdock, R.C.; Lee, T.; Schlager, J.J.; Hussain, S.M.; Hofmann, M.-C. Silver nanoparticles disrupt GDNF/Fyn kinase signaling in spermatogonial stem cells. *Toxicol. Sci.* **2010**, *116*, 577–589. [CrossRef]
72. Mohammadinejad, R.; Moosavi, M.A.; Tavakol, S.; Vardar, D.Ö.; Hosseini, A.; Rahmati, M.; Dini, L.; Hussain, S.; Mandegary, A.; Klionsky, D.J. Necrotic, apoptotic and autophagic cell fates triggered by nanoparticles. *Autophagy* **2019**, *15*, 4–33. [CrossRef]
73. Ahmadi, F.; Branch, S. Impact of different levels of silver nanoparticles (Ag-NPs) on performance, oxidative enzymes and blood parameters in broiler chicks. *Pak. Vet. J.* **2012**, *32*, 325–328.
74. Haase, A.; Rott, S.; Mantion, A.; Graf, P.; Plendl, J.; Thünemann, A.F.; Meier, W.P.; Taubert, A.; Luch, A.; Reiser, G. Effects of silver nanoparticles on primary mixed neural cell cultures: Uptake, oxidative stress and acute calcium responses. *Toxicol. Sci.* **2012**, *126*, 457–468. [CrossRef]
75. Perde-Schrepler, M.; Florea, A.; Brie, I.; Virag, P.; Fischer-Fodor, E.; Vâlcan, A.; Gurzău, E.; Lisencu, C.; Maniu, A. Size-dependent cytotoxicity and genotoxicity of silver nanoparticles in cochlear cells in vitro. *J. Nanomater.* **2019**, *2019*, 6090259. [CrossRef]
76. Awasthi, K.K.; Awasthi, A.; Kumar, N.; Roy, P.; Awasthi, K.; John, P.J. Silver nanoparticle induced cytotoxicity, oxidative stress, and DNA damage in CHO cells. *J. Nanopart. Res.* **2013**, *15*, 1898. [CrossRef]
77. Banerjee, V.; Das, K.P. Interaction of silver nanoparticles with proteins: A characteristic protein concentration dependent profile of SPR signal. *Colloids Surfaces B Biointerfaces* **2013**, *111*, 71–79. [CrossRef]
78. Tripathi, D.K.; Tripathi, A.; Singh, S.; Singh, Y.; Vishwakarma, K.; Yadav, G.; Sharma, S.; Singh, V.K.; Mishra, R.K.; Upadhyay, R.G. Uptake, accumulation and toxicity of silver nanoparticle in autotrophic plants, and heterotrophic microbes: A concentric review. *Front. Microbiol.* **2017**, *8*, 7. [CrossRef]
79. Oukarroum, A.; Bras, S.; Perreault, F.; Popovic, R. Inhibitory effects of silver nanoparticles in two green algae, Chlorella vulgaris and Dunaliella tertiolecta. *Ecotoxicol. Environ. Saf.* **2012**, *78*, 80–85. [CrossRef]
80. Moreno-Garrido, I.; Pérez, S.; Blasco, J. Toxicity of silver and gold nanoparticles on marine microalgae. *Mar. Environ. Res.* **2015**, *111*, 60–73. [CrossRef] [PubMed]
81. Hu, P.; Zhang, X.; Zhang, C.; Chen, Z. Molecular interactions between gold nanoparticles and model cell membranes. *Phys. Chem. Chem. Phys.* **2015**, *17*, 9873–9884. [CrossRef] [PubMed]
82. Xiang, L.; Fang, J.; Cheng, H. Toxicity of silver nanoparticles to green algae *M. aeruginosa* and alleviation by organic matter. *Environ. Monit. Assess.* **2018**, *190*, 667. [CrossRef]
83. Khoshnamvand, M.; Hao, Z.; Fadare, O.O.; Hanachi, P.; Chen, Y.; Liu, J. Toxicity of biosynthesized silver nanoparticles to aquatic organisms of different trophic levels. *Chemosphere* **2020**, *258*, 127346. [CrossRef]
84. Ottoni, C.A.; Neto, M.C.L.; Léo, P.; Ortolan, B.D.; Barbieri, E.; De Souza, A.O. Environmental impact of biogenic silver nanoparticles in soil and aquatic organisms. *Chemosphere* **2020**, *239*, 124698. [CrossRef] [PubMed]
85. Kruszewski, M.; Grzelak, A. Nanoparticle toxicity and reactive species: An overview. In *Toxicology*; Academic Press: Cambridge, MA, USA, 2021; pp. 11–21.
86. Sun, J.; Wan, J.; Zhai, X.; Wang, J.; Liu, Z.; Tian, H.; Xin, L. Silver nanoparticles: Correlating particle size and ionic Ag release with cytotoxicity, genotoxicity, and inflammatory responses in human cell lines. *Toxicol. Ind. Health* **2021**, *37*, 198–209. [CrossRef] [PubMed]
87. Li, M.; Liu, W.; Slaveykova, V.I. Effects of mixtures of engineered nanoparticles and metallic pollutants on aquatic organisms. *Environments* **2020**, *7*, 27. [CrossRef]
88. Arora, S.; Jain, J.; Rajwade, J.M.; Paknikar, K.M. Interactions of silver nanoparticles with primary mouse fibroblasts and liver cells. *Toxicol. Appl. Pharmacol.* **2009**, *236*, 310–318. [CrossRef]
89. Picó, Y.; Andreu, V. Analytical tools able to detect ENP/NM/MNs in both artificial and natural environmental water media. In *Ecotoxicology of Nanoparticles in Aquatic Systems*; CRC Press: Boca Raton, FL, USA, 2019; pp. 230–258. ISBN 1315158760.
90. McShan, D.; Ray, P.C.; Yu, H. Molecular toxicity mechanism of nanosilver. *J. Food Drug Anal.* **2014**, *22*, 116–127. [CrossRef]
91. Unrine, J.M.; Colman, B.P.; Bone, A.J.; Gondikas, A.P.; Matson, C.W. Biotic and abiotic interactions in aquatic microcosms determine fate and toxicity of Ag nanoparticles. Part 1. Aggregation and dissolution. *Environ. Sci. Technol.* **2012**, *46*, 6915–6924. [CrossRef]
92. Azimzada, A.; Tufenkji, N.; Wilkinson, K.J. Transformations of silver nanoparticles in wastewater effluents: Links to Ag bioavailability. *Environ. Sci. Nano* **2017**, *4*, 1339–1349. [CrossRef]
93. Sirelkhatim, A.; Mahmud, S.; Seeni, A.; Kaus, N.H.M.; Ann, L.C.; Bakhori, S.K.M.; Hasan, H.; Mohamad, D. Review on zinc oxide nanoparticles: Antibacterial activity and toxicity mechanism. *Nano-Micro Lett.* **2015**, *7*, 219–242. [CrossRef]

94. Sharifi, M.; Hosseinali, S.H.; Saboury, A.A.; Szegezdi, E.; Falahati, M. Involvement of planned cell death of necroptosis in cancer treatment by nanomaterials: Recent advances and future perspectives. *J. Control. Release* **2019**, *299*, 121–137. [CrossRef]
95. Fu, P.P.; Xia, Q.; Hwang, H.-M.; Ray, P.C.; Yu, H. Mechanisms of nanotoxicity: Generation of reactive oxygen species. *J. Food Drug Anal.* **2014**, *22*, 64–75. [CrossRef]
96. Wu, Y.; Zhou, Q. Silver nanoparticles cause oxidative damage and histological changes in medaka (Oryzias latipes) after 14 days of exposure. *Environ. Toxicol. Chem.* **2013**, *32*, 165–173. [CrossRef] [PubMed]
97. Asharani, P.V.; Hande, M.P.; Valiyaveettil, S. Anti-proliferative activity of silver nanoparticles. *BMC Cell Biol.* **2009**, *10*, 65. [CrossRef] [PubMed]
98. Fuchs, Y.; Steller, H. Programmed cell death in animal development and disease. *Cell* **2011**, *147*, 742–758. [CrossRef]
99. Quevedo, A.C.; Lynch, I.; Valsami-Jones, E. Silver nanoparticle induced toxicity and cell death mechanisms in embryonic zebrafish cells. *Nanoscale* **2021**, *13*, 6142–6161. [CrossRef]
100. Guicciardi, M.E.; Gores, G.J. Life and death by death receptors. *FASEB J.* **2009**, *23*, 1625–1637. [CrossRef]
101. Ferdous, Z.; Nemmar, A. Health impact of silver nanoparticles: A review of the biodistribution and toxicity following various routes of exposure. *Int. J. Mol. Sci.* **2020**, *21*, 2375. [CrossRef] [PubMed]
102. Kang, S.J.; Ryoo, I.; Lee, Y.J.; Kwak, M.-K. Role of the Nrf2-heme oxygenase-1 pathway in silver nanoparticle-mediated cytotoxicity. *Toxicol. Appl. Pharmacol.* **2012**, *258*, 89–98. [CrossRef]
103. Li, Y.; Guo, M.; Lin, Z.; Zhao, M.; Xiao, M.; Wang, C.; Xu, T.; Chen, T.; Zhu, B. Polyethylenimine-functionalized silver nanoparticle-based co-delivery of paclitaxel to induce HepG2 cell apoptosis. *Int. J. Nanomed.* **2016**, *11*, 6693. [CrossRef] [PubMed]
104. Akter, M.; Sikder, M.T.; Rahman, M.M.; Ullah, A.K.M.A.; Hossain, K.F.B.; Banik, S.; Hosokawa, T.; Saito, T.; Kurasaki, M. A systematic review on silver nanoparticles-induced cytotoxicity: Physicochemical properties and perspectives. *J. Adv. Res.* **2018**, *9*, 1–16. [CrossRef]
105. Hsin, Y.-H.; Chen, C.-F.; Huang, S.; Shih, T.-S.; Lai, P.-S.; Chueh, P.J. The apoptotic effect of nanosilver is mediated by a ROS-and JNK-dependent mechanism involving the mitochondrial pathway in NIH3T3 cells. *Toxicol. Lett.* **2008**, *179*, 130–139. [CrossRef]
106. Piao, M.J.; Kang, K.A.; Lee, I.K.; Kim, H.S.; Kim, S.; Choi, J.Y.; Choi, J.; Hyun, J.W. Silver nanoparticles induce oxidative cell damage in human liver cells through inhibition of reduced glutathione and induction of mitochondria-involved apoptosis. *Toxicol. Lett.* **2011**, *201*, 92–100. [CrossRef] [PubMed]
107. Quan, J.-H.; Gao, F.F.; Ismail, H.A.H.A.; Yuk, J.-M.; Cha, G.-H.; Chu, J.-Q.; Lee, Y.-H. Silver nanoparticle-induced apoptosis in ARPE-19 cells is inhibited by Toxoplasma gondii pre-infection through suppression of NOX4-dependent ROS generation. *Int. J. Nanomedicine* **2020**, *15*, 3695. [CrossRef] [PubMed]
108. Limbach, L.K.; Wick, P.; Manser, P.; Grass, R.N.; Bruinink, A.; Stark, W.J. Exposure of engineered nanoparticles to human lung epithelial cells: Influence of chemical composition and catalytic activity on oxidative stress. *Environ. Sci. Technol.* **2007**, *41*, 4158–4163. [CrossRef]
109. Baun, A.; Sørensen, S.N.; Rasmussen, R.F.; Hartmann, N.B.; Koch, C.B. Toxicity and bioaccumulation of xenobiotic organic compounds in the presence of aqueous suspensions of aggregates of nano-C60. *Aquat. Toxicol.* **2008**, *86*, 379–387. [CrossRef]
110. Hsiao, I.-L.; Hsieh, Y.-K.; Wang, C.-F.; Chen, I.-C.; Huang, Y.-J. Trojan-horse mechanism in the cellular uptake of silver nanoparticles verified by direct intra-and extracellular silver speciation analysis. *Environ. Sci. Technol.* **2015**, *49*, 3813–3821. [CrossRef]
111. Su, Y.; Yan, X.; Pu, Y.; Xiao, F.; Wang, D.; Yang, M. Risks of single-walled carbon nanotubes acting as contaminants-carriers: Potential release of phenanthrene in Japanese medaka (Oryzias latipes). *Environ. Sci. Technol.* **2013**, *47*, 4704–4710. [CrossRef]
112. Zhang, W.; Xiao, B.; Fang, T. Chemical transformation of silver nanoparticles in aquatic environments: Mechanism, morphology and toxicity. *Chemosphere* **2018**, *191*, 324–334. [CrossRef] [PubMed]
113. He, D.; Jones, A.M.; Garg, S.; Pham, A.N.; Waite, T.D. Silver nanoparticle− reactive oxygen species interactions: Application of a charging− discharging model. *J. Phys. Chem. C* **2011**, *115*, 5461–5468. [CrossRef]
114. Khan, F.R.; Misra, S.K.; Bury, N.R.; Smith, B.D.; Rainbow, P.S.; Luoma, S.N.; Valsami-Jones, E. Inhibition of potential uptake pathways for silver nanoparticles in the estuarine snail Peringia ulvae. *Nanotoxicology* **2015**, *9*, 493–501. [CrossRef]
115. Navarro, E.; Piccapietra, F.; Wagner, B.; Marconi, F.; Kaegi, R.; Odzak, N.; Sigg, L.; Behra, R. Toxicity of silver nanoparticles to Chlamydomonas reinhardtii. *Environ. Sci. Technol.* **2008**, *42*, 8959–8964. [CrossRef]
116. Khaydarov, R.R.; Khaydarov, R.A.; Gapurova, O. Remediation of contaminated groundwater using nano-carbon colloids. In *Nanomaterials: Risks and Benefits*; Springer: Berlin/Heidelberg, Germany, 2009; pp. 219–224.
117. Choi, O.K.; Hu, Z.Q. Nitrification inhibition by silver nanoparticles. *Water Sci. Technol.* **2009**, *59*, 1699–1702. [CrossRef] [PubMed]
118. Lok, C.-N.; Ho, C.-M.; Chen, R.; He, Q.-Y.; Yu, W.-Y.; Sun, H.; Tam, P.K.-H.; Chiu, J.-F.; Che, C.-M. Silver nanoparticles: Partial oxidation and antibacterial activities. *JBIC J. Biol. Inorg. Chem.* **2007**, *12*, 527–534. [CrossRef] [PubMed]
119. De Matteis, V.; Malvindi, M.A.; Galeone, A.; Brunetti, V.; De Luca, E.; Kote, S.; Kshirsagar, P.; Sabella, S.; Bardi, G.; Pompa, P.P. Negligible particle-specific toxicity mechanism of silver nanoparticles: The role of Ag+ ion release in the cytosol. *Nanomed. Nanotechnol. Biol. Med.* **2015**, *11*, 731–739. [CrossRef]
120. Jiang, H.S.; Yin, L.; Ren, N.N.; Xian, L.; Zhao, S.; Li, W.; Gontero, B. The effect of chronic silver nanoparticles on aquatic system in microcosms. *Environ. Pollut.* **2017**, *223*, 395–402. [CrossRef]
121. Rubino, F.M. Toxicity of glutathione-binding metals: A review of targets and mechanisms. *Toxics* **2015**, *3*, 20–62. [CrossRef]
122. Navarro, D.A.; Kirby, J.K.; McLaughlin, M.J.; Waddington, L.; Kookana, R.S. Remobilisation of silver and silver sulphide nanoparticles in soils. *Environ. Pollut.* **2014**, *193*, 102–110. [CrossRef] [PubMed]

123. Quadros, M.E.; Marr, L.C. Environmental and human health risks of aerosolized silver nanoparticles. *J. Air Waste Manag. Assoc.* **2010**, *60*, 770–781. [CrossRef] [PubMed]
124. Moustafa, E.M.; Khalil, R.H.; Saad, T.T.; Amer, M.T.; Shukry, M.; Farrag, F.; Elsawy, A.A.; Lolo, E.E.; Sakran, M.I.; Hamouda, A.H. Silver nanoparticles as an antibacterial agent in Oreochromis niloticus and Sparus auratus fish. *Aquac. Res.* **2021**, *52*, 6218–6234. [CrossRef]
125. Ghetas, H.A.; Abdel-Razek, N.; Shakweer, M.S.; Abotaleb, M.M.; Paray, B.A.; Ali, S.; Eldessouki, E.A.; Dawood, M.A.O.; Khalil, R.H. Antimicrobial activity of chemically and biologically synthesized silver nanoparticles against some fish pathogens. *Saudi J. Biol. Sci.* **2022**, *29*, 1298–1305. [CrossRef] [PubMed]
126. Romo-Quiñonez, C.R.; Álvarez-Sánchez, A.R.; Álvarez-Ruiz, P.; Chávez-Sánchez, M.C.; Bogdanchikova, N.; Pestryakov, A.; Mejia-Ruiz, C.H. Evaluation of a new Argovit as an antiviral agent included in feed to protect the shrimp Litopenaeus vannamei against White Spot Syndrome Virus infection. *PeerJ* **2020**, *8*, e8446. [CrossRef]
127. Ochoa-Meza, A.R.; Álvarez-Sánchez, A.R.; Romo-Quiñonez, C.R.; Barraza, A.; Magallón-Barajas, F.J.; Chávez-Sánchez, A.; García-Ramos, J.C.; Toledano-Magaña, Y.; Bogdanchikova, N.; Pestryakov, A. Silver nanoparticles enhance survival of white spot syndrome virus infected Penaeus vannamei shrimps by activation of its immunological system. *Fish Shellfish Immunol.* **2019**, *84*, 1083–1089. [CrossRef]
128. Rai, M.; Yadav, A.; Gade, A. Silver nanoparticles as a new generation of antimicrobials. *Biotechnol. Adv.* **2009**, *27*, 76–83. [CrossRef] [PubMed]
129. Agnihotri, S.; Mukherji, S.; Mukherji, S. Size-controlled silver nanoparticles synthesized over the range 5–100 nm using the same protocol and their antibacterial efficacy. *Rsc Adv.* **2014**, *4*, 3974–3983. [CrossRef]
130. Mendis, E.; Rajapakse, N.; Byun, H.-G.; Kim, S.-K. Investigation of jumbo squid (Dosidicus gigas) skin gelatin peptides for their in vitro antioxidant effects. *Life Sci.* **2005**, *77*, 2166–2178. [CrossRef]
131. Priyadarshini, S.; Gopinath, V.; Priyadharsshini, N.M.; MubarakAli, D.; Velusamy, P. Synthesis of anisotropic silver nanoparticles using novel strain, Bacillus flexus and its biomedical application. *Colloids Surfaces B Biointerfaces* **2013**, *102*, 232–237. [CrossRef]
132. Dibrov, P.; Dzioba, J.; Gosink, K.K.; Häse, C.C. Chemiosmotic mechanism of antimicrobial activity of Ag+ in Vibrio cholerae. *Antimicrob. Agents Chemother.* **2002**, *46*, 2668–2670. [CrossRef]
133. Hamouda, T.; Myc, A.; Donovan, B.; Shih, A.Y.; Reuter, J.D.; Baker, J.R. A novel surfactant nanoemulsion with a unique non-irritant topical antimicrobial activity against bacteria, enveloped viruses and fungi. *Microbiol. Res.* **2001**, *156*, 1–7. [CrossRef] [PubMed]
134. Sondi, I.; Salopek-Sondi, B. Silver nanoparticles as antimicrobial agent: A case study on E. coli as a model for Gram-negative bacteria. *J. Colloid Interface Sci.* **2004**, *275*, 177–182. [CrossRef] [PubMed]
135. Qamar, S.U.R. Nanocomposites: Potential therapeutic agents for the diagnosis and treatment of infectious diseases and cancer. *Colloid Interface Sci. Commun.* **2021**, *43*, 100463. [CrossRef]
136. Chaloupka, K.; Malam, Y.; Seifalian, A.M. Nanosilver as a new generation of nanoproduct in biomedical applications. *Trends Biotechnol.* **2010**, *28*, 580–588. [CrossRef]
137. Kędziora, A.; Speruda, M.; Krzyżewska, E.; Rybka, J.; Łukowiak, A.; Bugla-Płoskońska, G. Similarities and differences between silver ions and silver in nanoforms as antibacterial agents. *Int. J. Mol. Sci.* **2018**, *19*, 444. [CrossRef] [PubMed]
138. Rai, M.; Kon, K.; Ingle, A.; Duran, N.; Galdiero, S.; Galdiero, M. Broad-spectrum bioactivities of silver nanoparticles: The emerging trends and future prospects. *Appl. Microbiol. Biotechnol.* **2014**, *98*, 1951–1961. [CrossRef] [PubMed]
139. Alshareef, A.; Laird, K.; Cross, R.B.M. Shape-dependent antibacterial activity of silver nanoparticles on Escherichia coli and Enterococcus faecium bacterium. *Appl. Surf. Sci.* **2017**, *424*, 310–315. [CrossRef]
140. Abbas, Q.; Yousaf, B.; Ullah, H.; Ali, M.U.; Zia-ur-Rehman, M.; Rizwan, M.; Rinklebe, J. Biochar-induced immobilization and transformation of silver-nanoparticles affect growth, intracellular-radicles generation and nutrients assimilation by reducing oxidative stress in maize. *J. Hazard. Mater.* **2020**, *390*, 121976. [CrossRef]
141. Jia, Z.; Zhou, W.; Yan, J.; Xiong, P.; Guo, H.; Cheng, Y.; Zheng, Y. Constructing multilayer silk protein/Nanosilver biofunctionalized hierarchically structured 3D printed Ti6Al4 V scaffold for repair of infective bone defects. *ACS Biomater. Sci. Eng.* **2018**, *5*, 244–261. [CrossRef] [PubMed]
142. Marjaneh, R.M.; Rahmani, F.; Hassanian, S.M.; Rezaei, N.; Hashemzehi, M.; Bahrami, A.; Ariakia, F.; Fiuji, H.; Sahebkar, A.; Avan, A. Phytosomal curcumin inhibits tumor growth in colitis-associated colorectal cancer. *J. Cell. Physiol.* **2018**, *233*, 6785–6798. [CrossRef]
143. Ribeiro, A.P.C.; Anbu, S.; Alegria, E.; Fernandes, A.R.; Baptista, P.V.; Mendes, R.; Matias, A.S.; Mendes, M.; da Silva, M.F.C.G.; Pombeiro, A.J.L. Evaluation of cell toxicity and DNA and protein binding of green synthesized silver nanoparticles. *Biomed. Pharmacother.* **2018**, *101*, 137–144. [CrossRef]
144. Talebpour, Z.; Haghighi, F.; Taheri, M.; Hosseinzadeh, M.; Gharavi, S.; Habibi, F.; Aliahmadi, A.; Sadr, A.S.; Azad, J. Binding interaction of spherical silver nanoparticles and calf thymus DNA: Comprehensive multispectroscopic, molecular docking, and RAPD PCR studies. *J. Mol. Liq.* **2019**, *289*, 111185. [CrossRef]
145. Cui, L.; Chen, P.; Chen, S.; Yuan, Z.; Yu, C.; Ren, B.; Zhang, K. In situ study of the antibacterial activity and mechanism of action of silver nanoparticles by surface-enhanced Raman spectroscopy. *Anal. Chem.* **2013**, *85*, 5436–5443. [CrossRef]
146. Laban, G.; Nies, L.F.; Turco, R.F.; Bickham, J.W.; Sepúlveda, M.S. The effects of silver nanoparticles on fathead minnow (Pimephales promelas) embryos. *Ecotoxicology* **2010**, *19*, 185–195. [CrossRef]

147. Choi, O.; Hu, Z. Size dependent and reactive oxygen species related nanosilver toxicity to nitrifying bacteria. *Environ. Sci. Technol.* **2008**, *42*, 4583–4588. [CrossRef] [PubMed]
148. Zheng, Y.; Hou, L.; Liu, M.; Newell, S.E.; Yin, G.; Yu, C.; Zhang, H.; Li, X.; Gao, D.; Gao, J. Effects of silver nanoparticles on nitrification and associated nitrous oxide production in aquatic environments. *Sci. Adv.* **2017**, *3*, e1603229. [CrossRef] [PubMed]
149. Wood, C.M. Silver. In *Fish Physiology*; Elsevier: Amsterdam, The Netherlands, 2011; Volume 31, pp. 1–65. ISBN 1546-5098.
150. Lekamge, S.; Ball, A.S.; Shukla, R.; Nugegoda, D. The toxicity of nanoparticles to organisms in freshwater. *Rev. Environ. Contam. Toxicol.* **2018**, *248*, 1–80.
151. Kim, S.; Ryu, D. Silver nanoparticle-induced oxidative stress, genotoxicity and apoptosis in cultured cells and animal tissues. *J. Appl. Toxicol.* **2013**, *33*, 78–89. [CrossRef]
152. Van Hoecke, K.; Quik, J.T.K.; Mankiewicz-Boczek, J.; De Schamphelaere, K.A.C.; Elsaesser, A.; Van der Meeren, P.; Barnes, C.; McKerr, G.; Howard, C.V.; Meent, D. Van De Fate and effects of CeO2 nanoparticles in aquatic ecotoxicity tests. *Environ. Sci. Technol.* **2009**, *43*, 4537–4546. [CrossRef]
153. Zhao, C.; Wang, W. Comparison of acute and chronic toxicity of silver nanoparticles and silver nitrate to Daphnia magna. *Environ. Toxicol. Chem.* **2011**, *30*, 885–892. [CrossRef] [PubMed]
154. Cameron, P.; Gaiser, B.K.; Bhandari, B.; Bartley, P.M.; Katzer, F.; Bridle, H. Silver nanoparticles decrease the viability of Cryptosporidium parvum oocysts. *Appl. Environ. Microbiol.* **2016**, *82*, 431–437. [CrossRef]
155. Juganson, K.; Mortimer, M.; Ivask, A.; Pucciarelli, S.; Miceli, C.; Orupold, K.; Kahru, A. Mechanisms of toxic action of silver nanoparticles in the protozoan Tetrahymena thermophila: From gene expression to phenotypic events. *Environ. Pollut.* **2017**, *225*, 481–489. [CrossRef]
156. Greulich, C.; Braun, D.; Peetsch, A.; Diendorf, J.; Siebers, B.; Epple, M.; Köller, M. The toxic effect of silver ions and silver nanoparticles towards bacteria and human cells occurs in the same concentration range. *RSC Adv.* **2012**, *2*, 6981–6987. [CrossRef]
157. Marambio-Jones, C.; Hoek, E. A review of the antibacterial effects of silver nanomaterials and potential implications for human health and the environment. *J. Nanopart. Res.* **2010**, *12*, 1531–1551. [CrossRef]
158. Ruparelia, J.P.; Chatterjee, A.K.; Duttagupta, S.P.; Mukherji, S. Strain specificity in antimicrobial activity of silver and copper nanoparticles. *Acta Biomater.* **2008**, *4*, 707–716. [CrossRef] [PubMed]
159. Morones, J.R.; Elechiguerra, J.L.; Camacho, A.; Holt, K.; Kouri, J.B.; Ramírez, J.T.; Yacaman, M.J. The bactericidal effect of silver nanoparticles. *Nanotechnology* **2005**, *16*, 2346. [CrossRef] [PubMed]
160. Kuppusamy, P.; Ichwan, S.J.A.; Parine, N.R.; Yusoff, M.M.; Maniam, G.P.; Govindan, N. Intracellular biosynthesis of Au and Ag nanoparticles using ethanolic extract of Brassica oleracea L. and studies on their physicochemical and biological properties. *J. Environ. Sci.* **2015**, *29*, 151–157. [CrossRef] [PubMed]
161. Dietz, K.-J.; Herth, S. Plant nanotoxicology. *Trends Plant Sci.* **2011**, *16*, 582–589. [CrossRef] [PubMed]
162. Al-Huqail, A.A.; Hatata, M.M.; Al-Huqail, A.A.; Ibrahim, M.M. Preparation, characterization of silver phyto nanoparticles and their impact on growth potential of Lupinus termis L. seedlings. *Saudi J. Biol. Sci.* **2018**, *25*, 313–319. [CrossRef]
163. Sun, J.; Wang, L.; Li, S.; Yin, L.; Huang, J.; Chen, C. Toxicity of silver nanoparticles to Arabidopsis: Inhibition of root gravitropism by interfering with auxin pathway. *Environ. Toxicol. Chem.* **2017**, *36*, 2773–2780. [CrossRef]
164. Panda, K.K.; Achary, V.M.M.; Krishnaveni, R.; Padhi, B.K.; Sarangi, S.N.; Sahu, S.N.; Panda, B.B. In vitro biosynthesis and genotoxicity bioassay of silver nanoparticles using plants. *Toxicol. In Vitr.* **2011**, *25*, 1097–1105. [CrossRef] [PubMed]
165. Engelmann, P.; Bodó, K.; Najbauer, J.; Németh, P. Annelida: Oligochaetes (Segmented Worms): Earthworm immunity, quo vadis? Advances and new paradigms in the omics era. In *Advances in Comparative Immunology*; Springer: Berlin/Heidelberg, Germany, 2018; pp. 135–159.
166. Wang, F.; Guan, W.; Xu, L.; Ding, Z.; Ma, H.; Ma, A.; Terry, N. Effects of nanoparticles on algae: Adsorption, distribution, ecotoxicity and fate. *Appl. Sci.* **2019**, *9*, 1534. [CrossRef]
167. Kwak, J., II; Cui, R.; Nam, S.-H.; Kim, S.W.; Chae, Y.; An, Y.-J. Multispecies toxicity test for silver nanoparticles to derive hazardous concentration based on species sensitivity distribution for the protection of aquatic ecosystems. *Nanotoxicology* **2016**, *10*, 521–530. [CrossRef]
168. Naguib, M.; Mahmoud, U.M.; Mekkawy, I.A.; Sayed, A.E.-D.H. Hepatotoxic effects of silver nanoparticles on Clarias gariepinus; Biochemical, histopathological, and histochemical studies. *Toxicol. Rep.* **2020**, *7*, 133–141. [CrossRef]
169. Kakakhel, M.A.; Wu, F.; Sajjad, W.; Zhang, Q.; Khan, I.; Ullah, K.; Wang, W. Long-term exposure to high-concentration silver nanoparticles induced toxicity, fatality, bioaccumulation, and histological alteration in fish (Cyprinus carpio). *Environ. Sci. Eur.* **2021**, *33*, 1–11. [CrossRef]
170. Hammond, S.A.; Carew, A.C.; Helbing, C.C. Evaluation of the effects of titanium dioxide nanoparticles on cultured Rana catesbeiana tailfin tissue. *Front. Genet.* **2013**, *4*, 251. [CrossRef] [PubMed]
171. Hinther, A.; Vawda, S.; Skirrow, R.C.; Veldhoen, N.; Collins, P.; Cullen, J.T.; van Aggelen, G.; Helbing, C.C. Nanometals induce stress and alter thyroid hormone action in amphibia at or below North American water quality guidelines. *Environ. Sci. Technol.* **2010**, *44*, 8314–8321. [CrossRef] [PubMed]
172. Jafari, S.A.; Cheraghi, S.; Mirbakhsh, M.; Mirza, R.; Maryamabadi, A. Employing response surface methodology for optimization of mercury bioremediation by Vibrio parahaemolyticus PG02 in coastal sediments of Bushehr, Iran. *CLEAN–Soil Air Water* **2015**, *43*, 118–126. [CrossRef]

173. Ashokkumar, P.; Loashini, V.M.; Bhavya, V. Effect of pH, Temperature and biomass on biosorption of heavy metals by Sphaerotilus natans. *Int. J. Microbiol. Mycol.* **2017**, *6*, 32–38.
174. Benazir, J.F.; Suganthi, R.; Rajvel, D.; Pooja, M.P.; Mathithumilan, B. Bioremediation of chromium in tannery effluent by microbial consortia. *Afr. J. Biotechnol.* **2010**, *9*, 3140–3143.
175. Mariano, S.; Panzarini, E.; Inverno, M.D.; Voulvoulis, N.; Dini, L. Toxicity, bioaccumulation and biotransformation of glucose-capped silver nanoparticles in green microalgae Chlorella vulgaris. *Nanomaterials* **2020**, *10*, 1377. [CrossRef]
176. Al-Garni, S.M.; Ghanem, K.M.; Ibrahim, A.S. Biosorption of mercury by capsulated and slime layerforming Gram-ve bacilli from an aqueous solution. *Afr. J. Biotechnol.* **2010**, *9*, 6413–6421.
177. Igiri, B.E.; Okoduwa, S.I.R.; Idoko, G.O.; Akabuogu, E.P.; Adeyi, A.O.; Ejiogu, I.K. Toxicity and bioremediation of heavy metals contaminated ecosystem from tannery wastewater: A review. *J. Toxicol.* **2018**, *2018*, 2568038. [CrossRef]
178. Durán, N.; Marcato, P.D.; Alves, O.L.; Da Silva, J.P.S.; De Souza, G.I.H.; Rodrigues, F.A.; Esposito, E. Ecosystem protection by effluent bioremediation: Silver nanoparticles impregnation in a textile fabrics process. *J. Nanopart. Res.* **2010**, *12*, 285–292. [CrossRef]
179. Gao, Z.; Liu, G.G.; Ye, H.; Rauschendorfer, R.; Tang, D.; Xia, X. Facile colorimetric detection of silver ions with picomolar sensitivity. *Anal. Chem.* **2017**, *89*, 3622–3629. [CrossRef] [PubMed]
180. Maruthupandi, M.; Vasimalai, N. Nanomolar detection of L-cysteine and Cu2+ ions based on Trehalose capped silver nanoparticles. *Microchem. J.* **2021**, *161*, 105782. [CrossRef]
181. Hussain, F.; Hafeez, J.; Khalifa, A.S.; Naeem, M.; Ali, T.; Eed, E.M. In vitro and in vivo study of inhibitory potentials of α-glucosidase and acetylcholinesterase and biochemical profiling of M. charantia in alloxan-induced diabetic rat models. *Am. J. Transl. Res.* **2022**, *14*, 3824–3839.
182. Safavi, A.; Ahmadi, R.; Mohammadpour, Z. Colorimetric sensing of silver ion based on anti aggregation of gold nanoparticles. *Sens. Actuators B Chem.* **2017**, *242*, 609–615. [CrossRef]
183. Song, T.; Zhu, X.; Zhou, S.; Yang, G.; Gan, W.; Yuan, Q. DNA derived fluorescent bio-dots for sensitive detection of mercury and silver ions in aqueous solution. *Appl. Surf. Sci.* **2015**, *347*, 505–513. [CrossRef]
184. Wang, H.; Xue, L.; Jiang, H. Ratiometric fluorescent sensor for silver ion and its resultant complex for iodide anion in aqueous solution. *Org. Lett.* **2011**, *13*, 3844–3847. [CrossRef]