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Review

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Abstract

The extensive use of conventional pesticides has been a fundamental strategy in modern agriculture for controlling pests and increasing crop productivity; however, their improper application poses significant risks to human health and environmental sustainability. This review compiles scientific evidence linking pesticide exposure to oxidative stress and genotoxic damage particularly affecting rural populations and commonly consumed foods, even at levels exceeding the maximum permissible limits in fruits, vegetables, and animal products. Additionally, excessive pesticide use has been shown to alter soil microbiota, negatively compromising long-term agricultural fertility. In response to these challenges, recent advances in nanotechnology offer promising alternatives. This review highlights the development of nanopesticides designed for controlled release, improved stability, and targeted delivery of active ingredients, thereby reducing environmental contamination and increasing efficacy. Moreover, emerging nanobiosensor technologies, such as e-nose and e-tongue systems, have shown potential for real-time monitoring of pesticide residues and soil health. Although pesticides are still necessary, it is crucial to implement stricter laws and promote sustainable solutions that ensure safe and responsible agricultural practices. The need for evidence-based public policy is emphasized to regulate pesticide use and protect both human health and agricultural resources.

Keywords: oxidative stress; environmental sustainability; nanobiosensors; nanoparticles; antioxidant enzymes; nanopesticides

1. Introduction

Population standards regarding resource production have evolved in response to the requirements of their social and historical context, primarily during the mid-20th century, when advances in chemistry and technology enabled the development of chemical agents for various functions. Many of these developments were driven by military needs, such as the urgent need to ensure a stable food supply under the threat of armed conflicts or natural disasters, which encouraged the development of efficient agricultural strategies [1]. The use of pesticides is a consequence of the search for tools and methodologies that maximize food production. They also

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generated widespread acceptance among the population due to their contribution to disease reduction by eliminating their vectors (crop pests) [2].

The evolution of pesticides can be categorized into three general phases. The first involved the use of natural pesticides, such as sulfur, silicon dioxide, and rotenone, which have been known since 1870 for their membrane-damaging effect on pathogens [3]. The second phase involved the development of inorganic pesticides, such as arsenicals and sulfides, which were commonly used between 1870 and 1945. The third phase emerged with the synthesis of organic synthetic pesticides, such as organophosphates and carbamates, which are still widely used today [2]. These developments gave rise to a wide range of classifications based on the concentration of active ingredients, chemical composition, mechanism of action, persistence, and target pest [4]. Pesticides demonstrate great versatility due to their structural diversity and specific mechanism of action. These compounds can be tailored to control a wide variety of organisms, including insects, fungi, bacteria, mammals, weeds, or herbs.

Pesticides have played a crucial role in modern agriculture by enhancing crop yields and ensuring food security. However, the intensive and often indiscriminate use of conventional pesticides has led to serious concerns regarding environmental pollution and adverse health effects in humans. Pesticide residues are frequently detected in soil, water bodies, and food products, contributing to ecological imbalance, biodiversity loss, and the bioaccumulation of toxic compounds across trophic levels [5]. Human exposure to pesticides, particularly in agricultural communities, has been associated with a wide range of chronic health conditions, including endocrine disruption, neurotoxicity, reproductive disorders, and carcinogenesis [6].

Environmental impacts are also significant. Pesticide runoff can contaminate aquatic ecosystems, impairing microbial communities, affecting non-target organisms such as pollinators and beneficial insects, and disrupting natural pest control mechanisms [7]. The persistence of certain pesticides in the environment contributes to long-term contamination, undermining efforts to achieve sustainable agriculture and conserve soil fertility [8]. While their regulated use has led to increased crop and feed production, concerns remain regarding unregulated use and human and environmental exposure. Regulatory frameworks differ considerably between countries and regions, further complicating the management of their risks [9].

Considering these challenges, nanotechnology has emerged as a promising and sustainable solution for enhancing crop protection and productivity. Nanopesticides, nanoformulated fertilizers, and nanobiosensors offer improved stability, targeted delivery, and reduced dosage of active ingredients, potentially lowering environmental burden while maintaining agricultural efficacy. Additionally, nano-enabled systems facilitate controlled release and site-specific action, minimizing off-target effects and the development of resistance in pests [10,11].

Therefore, this review aims to provide a comprehensive overview of the environmental and human health impacts associated with the use of conventional pesticides while critically evaluating the emerging role of nanotechnology as a sustainable and safer alternative in modern agriculture.

2. Environmental Impact of Conventional Pesticides

The use of pesticides entails various risks, which can be grouped into two main factors: environmental damage and health damage. The environmental damage caused by these compounds results from their interaction with the environment, whether through contact during application, degradation, or the production of specific metabolites (Figure 1). The most significant environmental risks associated with pesticides include their physicochemical properties, persistence, and resistance to natural degradation, which can result in long-term contamination of soil, water, and air, depending on their molecular weight, ionizability, volatility, solubility, and polarity [7]. Pesticides can bioaccumulate and biomagnify through the food chain, posing threats to biodiversity and ecosystem stability [12]. Access to these fractions of the environment can culminate in the accumulation of potentially toxic compounds in considerable quantities in the food environment, for example, in vegetables, which is around 0.1 to 100 mg/kg depending on the nature of the food and the pesticide,

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which is why the environment to which each food is exposed must be studied; the response to these components will depend on the exposure, species, and susceptibility [13]. The harm caused to the environment is related to the alteration of the ecosystem's balance, primarily affecting flora and fauna [14]. Poor practices with pesticides and inadequate regulation of compounds generate waste that ends up in channels or aquifers, affecting marine species [15].

The most significant risk associated with pesticides in soil is their potential to accumulate due to their persistence and chemical stability, which can lead to long-term soil contamination [7]. This accumulation can affect soil microbial communities and reduce soil fertility, thereby disrupting soil health and ecosystem functions. For example, Streletskii et al. [16] found that the changes in the relative abundance of the genera T*errabacter*, *Kitasatospora*, *Streptomyces*, *Sphingomonas*, *Apiotrichum*, *Solicoccozyma*, *Gamsia*, and *Humicola* can be proposed as an indicator of pesticide contamination. Additionally, pesticides can leach and run off, transferring pollutants to water bodies and further contaminating groundwater and surface water systems [17]. The persistence and mobility of pesticides in the soil pose a long-term environmental risk by transforming soil into a secondary source of pollution, which can negatively impact plant growth, microbial diversity, and overall soil quality.

This contamination poses serious threats to aquatic ecosystems by affecting the health and diversity of marine organisms. Pesticides entering water bodies can lead to the mortality of fish and invertebrates, cause sub-lethal effects such as behavioral changes, and impair physiological functions [18]. Additionally, pesticides can affect algae and aquatic plants, which are crucial to the food web, resulting in decreased primary productivity and altered habitat structures. For example, a study found that the pesticides acetochlor, dicofol, and chlorpyrifos significantly inhibit the growth and photosynthesis (measured by chlorophyll-a) of the microalga *Skeletonema costatum* [19]. The bioaccumulation of pesticides in aquatic organisms can also biomagnify through the food chain, impacting higher trophic levels, including fish consumed by humans [12]. Water contamination by pesticides represents a critical environmental and public health concern due to their toxicity, persistence, and ability to enter human drinking water supplies.

Moreover, pesticides can have a significant impact on the ecosystem due to their toxic effects on non-target organisms and disruption of ecological balance [20]. Of the total amount of pesticides applied, less than 1% effectively target the intended pests, while most of the remaining pesticides disperse into the surrounding environment, posing risks to human health and harming non-target organisms within the agricultural ecosystem [21]. Pesticides can damage or eliminate beneficial species by disrupting essential biological functions. In earthworms, they compromise growth, reproduction, DNA integrity, and enzymatic activity. In pollinators such as honeybees, pesticides impair memory, learning, and alter foraging behavior. Additionally, they affect critical life-history traits in predators and parasitoids, including reproduction, development, longevity, and feeding patterns [21]. Widespread pesticide application often leads to resistance among target pests, necessitating the use of higher doses or the development of new chemical agents. This approach simultaneously suppresses natural enemies, such as predators and pollinators, which can lead to pest resurgence or secondary outbreaks that may be even more severe than the initial infestation. These impacts collectively threaten ecosystem stability, reduce biodiversity, and impair essential ecological services, including pollination, nutrient cycling, and water purification. The nature of many pesticides makes their environmental impact a long-lasting and widespread concern, emphasizing the need for effective management and remediation strategies.

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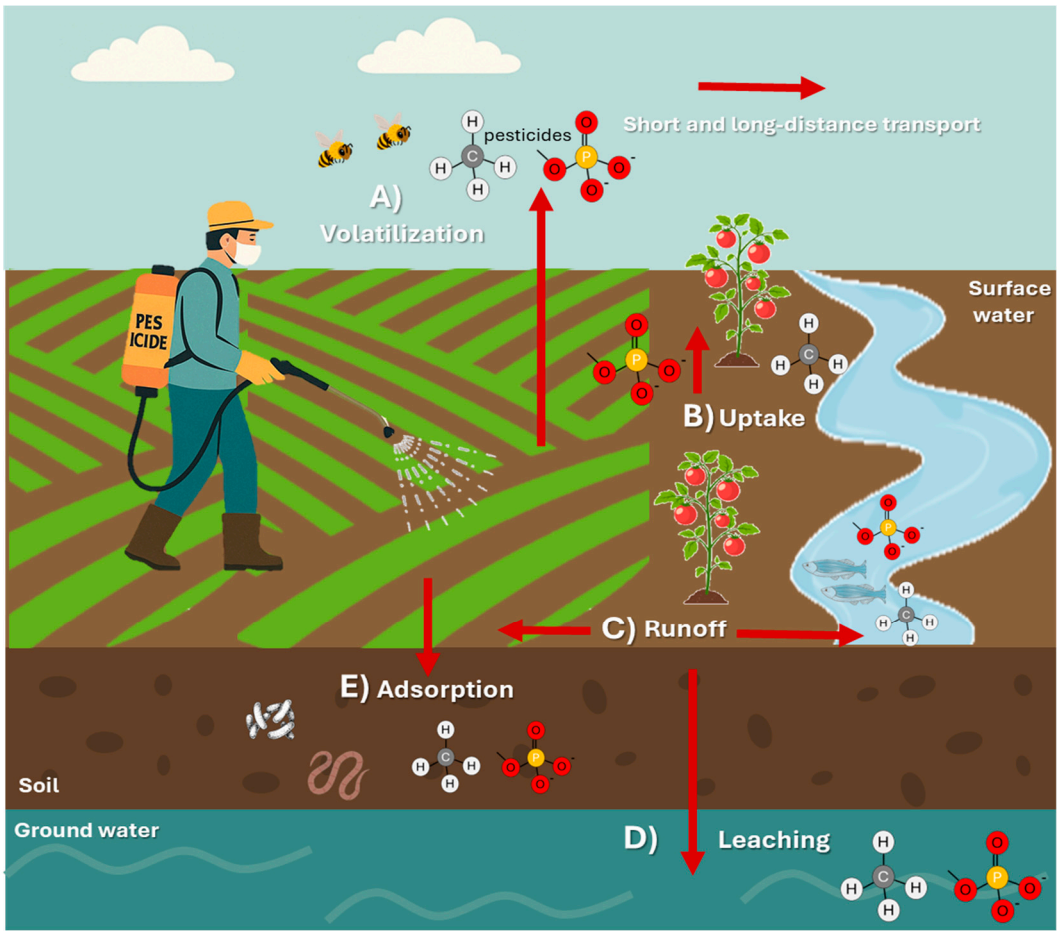

Figure 1. Main routes of environmental dispersion of pesticides applied in agriculture. A) After application in the field, pesticides can volatilize and be transported through the air over short and long distances, affecting non-target organisms such as pollinators. B) Part of the pesticide is absorbed by plants (uptake), generating residues in crops. C) The excess can run off over the soil surface into nearby water bodies (rivers, lakes), contaminating surface water and affecting aquatic fauna, or D) leach through the soil profile until it reaches groundwater. E) Furthermore, pesticides can absorb soil particles, altering the microbiota and beneficial organisms such as earthworms. These multiple dispersion routes represent a potential risk to the environment, food safety, and human health.

3. Oxidative Stress Risk Associated with Pesticide Exposure

Exposure to pesticides represents a significant risk to human health, both through immediate acute effects and through chronic consequences that can manifest years after exposure. In the short term, the main routes of absorption are through the skin, especially in agricultural workers, as well as inhalation and ingestion of residues in food and water [22]. This can cause immediate symptoms such as eye and skin irritation, nausea, vomiting, headaches, dizziness, respiratory distress, acute bronchitis, and even seizures. Long-term, chronic exposure to pesticides has been associated with a significantly increased risk of various types of cancer, neurological disorders (such as Parkinson's, Alzheimer's, and autism), endocrine and reproductive disruptions, immunotoxicity, autoimmune disorders, and premature aging [22–26]. These effects are mediated by multiple toxicity mechanisms, including oxidative stress, mitochondrial damage, genetic and epigenetic alterations, interference with key neurotransmitters such as acetylcholinesterase, and disruption of the gut microbiota. The most vulnerable populations include agricultural workers, children, pregnant women, and rural communities, who are at greater risk due to constant exposure or at sensitive stages of development [22]. These findings underscore the urgent need to implement stricter regulatory measures and promote sustainable agricultural alternatives.

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The extent of damage to human health varies and is influenced by the chemical structure of the compound, which dictates its mechanism of action. Its physicochemical properties determine its affinity for specific biological systems. The dose and duration of exposure are critical factors that determine both the severity and nature of the resulting health effects [27]. In this context, regulatory oversight of pesticide use is essential. One of the critical parameters for evaluating the safety of these compounds in international trade is the determination of their median lethal dose (LD_{50}), which identifies the concentration at which a pesticide becomes toxic or poses a potential risk to human health through ingestion or exposure. The LD_{50} represents the dose that causes mortality in 50% of a test population, typically determined using animal models such as mice or rats via oral or dermal administration. Normally, the lower the LD_{50}, the more toxic the pesticide is, and therefore, the more dangerous it is.

The potential harm associated with pesticide use is so broad that addressing it effectively requires classifying risks by compound type. Among the most studied pesticides are organophosphates, organochlorines, carbamates, pyrethroids, and their derivatives. Table 1 presents commonly used pesticides and the associated health risks resulting from exposure to them.

Organophosphates are highly lipophilic pesticides derived primarily from phosphoric acid and widely used as insecticides, fungicides, acaricides, and nematicides. Their primary mechanism of toxicity is their potent inhibition of acetylcholinesterase, which is attributed to the presence of a quaternary nitrogen group in their structure. Due to their high reactivity, some organophosphates have historically been employed as chemical warfare agents. It is estimated that approximately 40% of global crops are treated with this class of pesticide. Their lipid solubility and elevated vapor pressure at ambient temperatures enable rapid absorption through oral, dermal, and respiratory routes. Once inside the body, they distribute efficiently into lipid-rich tissues and can readily cross the blood-brain barrier. Despite this, they do not typically bioaccumulate due to their rapid and effective biotransformation [28]. Some examples of organophosphate pesticides are chlorpyrifos, diazinon, malathion, and parathion, among others.

Organochlorine pesticides are halogenated organic compounds widely recognized for their high chemical stability and environmental persistence. Approximately 40% of all pesticides used were organochlorines [29]. Their mechanism of action is based on altering nerve transmission, acting as modulators of sodium channels in neuronal membranes that remain abnormally open, thereby generating continuous hyperexcitation of the nervous system. Furthermore, some organochlorines interfere with GABA-regulated chloride channels, which exacerbate their neurotoxic effects. One of the main problems associated with their use is their capacity for bioaccumulation, as they are stored in the fatty tissues of organisms, including humans, and biomagnify throughout the food chain, increasing their concentration at higher trophic levels and posing a significant ecological and toxicological risk. Some types of organochlorines are dichlorodiphenyltrichloroethane (DDT), hexachlorobenzene, polychlorinated biphenyls, lindane, endosulfan, dieldrin, methoxychlor, dieldrin, chlordane, taxophene, and dicofol [30].

Carbamates are chemical compounds belonging to the family of esters derived from N-methyl or dimethyl carbamic acids, widely used as insecticides, nematicides, herbicides, and fungicides [31]. Their mechanism of action consists of the reversible inhibition of the enzyme acetylcholinesterase through a carbamylation process, which generates the accumulation of acetylcholine in neuronal synapses [32]. Although this mechanism is similar to that of organophosphates, the neurotoxic effects caused by carbamates are usually less prolonged and severe due to the reversible nature of the inhibition. In cases of acute toxicity, symptoms typically include manifestations of cholinergic syndrome, such as excessive salivation, muscle spasm, and respiratory distress, although with less duration and intensity. Unlike other more persistent pesticides, carbamates have a low capacity for bioaccumulation due to their short half-life and rapid metabolism and elimination by the body, which reduces their risk of long-term systemic accumulation [31].

Pyrethroids, such as permethrin, deltamethrin, resmethrin, tetramethrin, γ-cyhalothrin, and cypermethrin, are synthetic compounds designed to mimic the structure and function of natural

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pyrethrins and are widely used as insecticides in agriculture, public health, and domestic pest control. Pyrethroids are categorized as neurotoxins targeting the peripheral and central nervous system axons by modulating sodium channels in neurons, causing them to remain open longer than usual, which leads to hyperexcitation, paralysis, and ultimately, death in insects [33]. These compounds are more toxic to insects and fish than to mammals due to differences in metabolism rates and neuronal sensitivity. However, toxicity has been documented in aquatic organisms; in humans, exposure to high doses can cause paresthesia, skin irritation, seizures, and even severe neurological effects in extreme cases [34]. Although they present lower bioaccumulation and carcinogenic potential compared to organochlorines, their intensive use has favored the emergence of resistance in various insect species, posing a growing challenge for effective pest management.

Table 1. Highly used pesticides and the harm associated with their exposure in *in vivo* studies.

Pesticide	Type	Target Organism	Associate damage	DL_{50}	Reference
Parathion /Methyl parathion	Organophosphate	Herbicide	*In vivo* studies have linked its use to the development of heart disease, an increase in CAT, TBARS, and GPx biomarkers, and a decrease in SOD, resulting in an overload of oxidative stress, alterations in acetylcholinesterase levels, and overstimulation of the central nervous system.	6-14 mg/kg /2-30 mg/kg	[35,36]
Rotencidal	Coumarin	Bromadiolone	At low concentrations it has been linked to the appearance of oxidative stress in short exposures and the destabilization of biomolecules, at acute exposures bromadiolone has been linked to the inhibition of the carboxylation of vitamin K-dependent coagulation factors (II, VII, IX and X) making an anticoagulant effect, it is also widely related to the deterioration of the intestinal mucosa and bleeding in the digestive and urinary tract. There have been cases related to exposure to bromadiolone and the development of diseases of the central nervous system or conditions affecting the brain mass, such as leukoencephalopathy.	1.125 mg/kg	[37–40]
Carbofuran	Carbamates	Herbicide and insecticide	After exposure to humans, a considerable increase in oxidative stress has been reported in several organs, including the liver, brain, kidney, and heart, which leads to the propagation of necrosis in hepatic and nephrotic cells.	8-14 mg/kg	[41–43]
2,4-D	Phenoxyacetic Acid	Herbicide	It is a widely used compound that causes significant damage to the	639-764 mg/kg	[44,45]

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			environment and humans. In addition to the increase in oxidative stress and destabilization of biomolecules, it has been highly related to the inhibition of growth in cells and tissues; its effects have been studied in different in vivo models where they found a behavioral pattern in terms of neurotoxicity, a decrease in motor skills was observed, biochemically it showed a decrease in serotonin levels or a decrease in dopamine levels and its metabolites depending on the brain area analyzed.		
Cypermethrin	Pyrethroid	Acaracide	Often used in mixtures, its acute and subacute exposure causes clinical symptoms such as pneumonia, acute kidney injury, tearing, acute respiratory failure, and diarrhea. Cypermethrin primarily acts by delaying the closure of voltage-sensitive sodium channels. Most of the effects caused by poisoning with this pesticide are neurotoxic, particularly in the respiratory and gastrointestinal tracts. Cases of cardiotoxic conditions have been reported, but these are insufficient to associate them with cypermethrin poisoning.	240-4123 mg/kg	[46–49]
Imidacloprid	Neonicotinoid	Insecticide	The most widely used neonicotinoid in the world is known to produce oxidative stress upon exposure. It has also been observed that, in the case of oral ingestion, the main symptoms and associated damage are gastrointestinal without corrosive lesions and neurological effects, such as dyspnea, coma, and diaphoresis. There is a particular relationship between imidacloprid poisoning and the development of various types of liver damage, which sometimes occurs late.	450-650 mg/kg	[50–53]
Benomyl	Carbamates	Fungicide	Linked to the generation of systemic oxidative stress. In vitro studies in rat cardiomyoblasts (H9c2) demonstrated a 2-fold increase in ROS and glutathione levels measured in cells exposed to benomyl	>10000 mg/kg	[54,55]

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			compared to controls. Exposure to benomyl has been shown to induce apoptosis, oxidative stress, and DNA damage.		
Acetamiprid	Neonicotinoid	Insecticide	After the severe oxidative stress generated by this pesticide is linked to genotoxic damage and the formation of cleavages in tRNA due to the changes it generates in biomolecules, isolated cases have been reported where poisoning with acetamiprid triggered lactic acidosis, hyperglycemia, and intestinal obstruction.	217 mg/kg	[56–58]
Glyphosate	Organophosphate	Herbicide	Exposure to pesticides during the early stages of development can severely disrupt normal cell growth by interfering with several critical signaling pathways, leading to significant changes in cell differentiation, neuronal development, and myelination. Furthermore, glyphosate appears to have a notable toxic effect on neurotransmission, generating oxidative stress, neuroinflammation, and mitochondrial dysfunction, which can result in neuronal death through mechanisms such as autophagy, necrosis, or apoptosis. These neurotoxic effects are also associated with the development of behavioral disorders and impaired motor skills.	4320 mg/kg	[59,60]

CAT: catalase, TBARS: thiobarbituric acid reactive substances, GPx: glutathione peroxidase, SOD: superoxide dismutase, ROS: reactive oxygen species.

There are various mechanisms and pathways of pesticide damage; however, one mechanism stands out as common to different types of pesticides: oxidative stress. This concept was coined by Helmut Sies, who defined it as an imbalance between the production of oxidizing agents and antioxidant defenses. Oxidative stress leads to the generation of free radicals and reactive oxygen species (ROS), which are highly reactive and capable of damaging lipids, proteins, and DNA [61].]. Among free radicals and ROS, the most studied include hydroxyl, peroxyl, alkoxyl, hydroperoxyl radicals, as well as nitric oxide, all of which directly contribute to oxidative damage. In addition to ROS, there are reactive nitrogen species (RNS), unstable, nitrogen-containing molecules, that also participate in redox imbalance and contribute to cellular dysfunction and inflammation. [62].

ROS are primarily produced during oxidative metabolism, such as in the mitochondrial electron transport chain, where molecular oxygen (O_2) serves as the final electron acceptor and is ultimately reduced to water (H_2O) (Figure 2). If only a single electron is transferred to oxygen, a superoxide anion (O_2^-) is formed. This unstable species is converted into less reactive molecules by the action of the enzyme superoxide dismutase (SOD), which catalyzes its dismutation into hydrogen peroxide

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(H_2O_2) and oxygen. These intermediate products, in turn, can give rise to other reactive oxygen species. At the end of the electron transport chain, after the transfer of protons (H^+) and electrons (e^-), the complete reduction of molecular oxygen to water requires the addition of four electrons and four protons, highlighting the delicate balance in redox homeostasis [63].

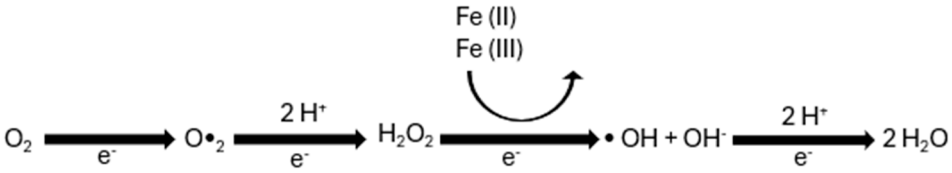

Figure 2. Production of reactive oxygen species (ROS) from molecular oxygen.

ROS are essential to the body at very low concentrations, serving as a cellular signaling system and providing protection against infectious agents. However, if this balance is disrupted, ROS, given their limited stability, can generate highly reactive free radicals. Multiple factors, including poor diet, stress conditions, exposure to UV radiation, and metal ions, among others, can trigger the overproduction of these radicals. Once generated, free radicals interact with cellular components, causing damage proportional to their concentration and persistence. ROS can overcome the endogenous antioxidant system; given their reactivity, they are capable of oxidizing biomolecules. When oxidized, there is a change in their structure, resulting in the total or partial inhibition of their normal function. The most susceptible biomolecules are proteins, lipids, and DNA (nucleic acids). In search of stability, the free radical can steal an electron from any available biomolecule, generating a chain reaction called oxidative stress [64,65].

Oxidative stress is associated with the development of diseases, which can be classified as those generated by pro-oxidants that modify the redox state and alter glucose tolerance, promoting mitochondrial oxidative stress in diseases such as cancer and diabetes mellitus. The second group includes inflammatory oxidative stress and increased activity of the enzyme nicotinamide adenine dinucleotide phosphate oxidase (NADPH-ox), leading to atherosclerosis and chronic inflammation. The third group is derived from the xanthine oxidase system, generating ROS that are implicated in ischemic reperfusion injury. Furthermore, the aging process is linked to the damaging effects of free radicals, which cause the oxidation of biomolecules such as lipids, DNA, and proteins, thereby directly impacting the aging process [66]

Pesticides, being a widely used product in agriculture, are an important factor to consider when studying the means of oxidative stress as a generator of chronic degenerative diseases. Exposure to pesticides significantly increases the production of ROS, which are responsible for altering the oxidation-reduction state of cells. Faced with these stimuli, the first line of defense against oxidative imbalance is the endogenous antioxidant system, which neutralizes reactive species, thereby correcting the imbalance. Figure 3 illustrates how pesticides induce oxidative DNA damage, resulting in numerical and structural chromosomal alterations, single- and double-strand DNA breaks, and epigenetic changes. These alterations result in changes in the expression of genes involved in maintaining cellular homeostasis and in the progression of diseases. Direct interaction with the pesticide can destabilize the cell membrane, resulting in the loss of essential membrane functions. Constant exposure to pesticides generates imbalances in antioxidant enzymes (depending on the nature of the pesticide), which results in a deficient antioxidant system.

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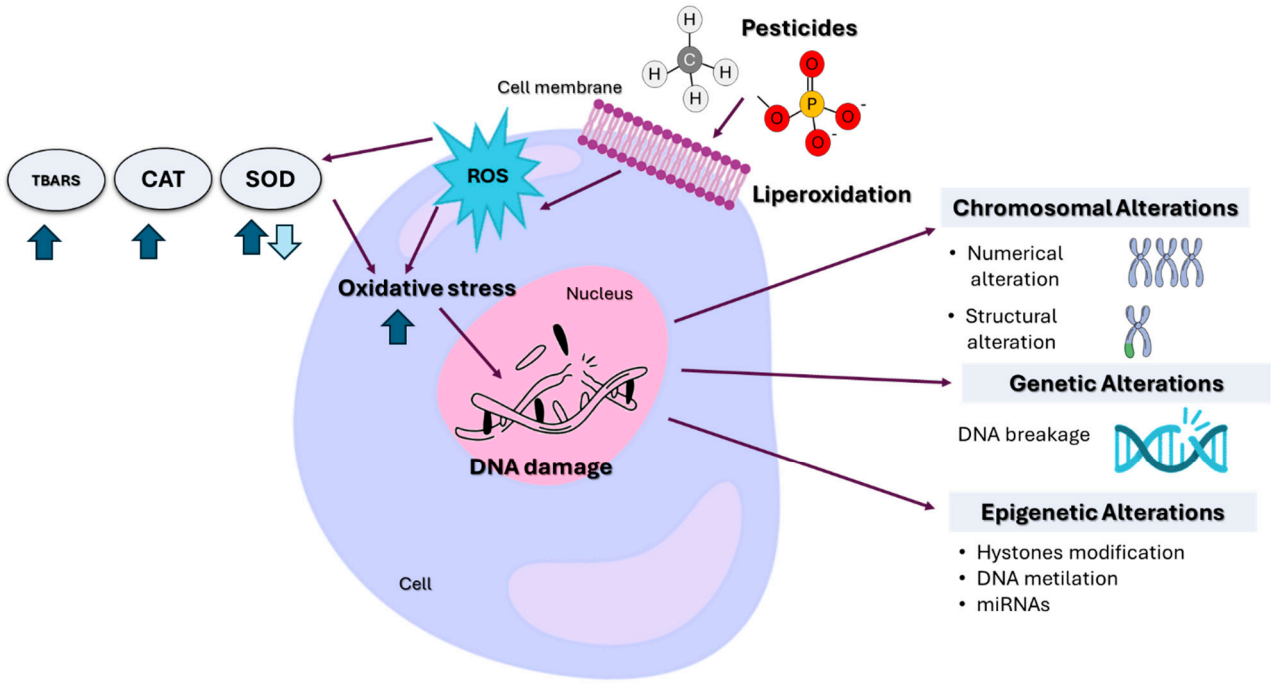

Figure 3. Mechanisms of pesticide damage at the cellular level. Adapted from [67].

SOD is an endogenous antioxidant enzyme that is the first line of defense against oxidative imbalances. It is a catalyst in the conversion of the superoxide radical into hydrogen peroxide and molecular oxygen. The resulting products are much less toxic and more stable, which contributes to cellular homeostasis. SOD activation due to pesticide exposure varies, depending on nature and duration of exposure. It has been observed that SOD levels differ depending on the stimulated areas. In the brain, they decrease during exposure, while erythrocyte SOD increases, mainly with exposure to organophosphates and organochlorines [68].

On the other hand, the primary function of CAT is to protect cells from the harmful effects of hydrogen peroxide generated by metabolism and various external factors such as pesticide exposure. It is essential when it comes to tolerance to oxidative stress, and its action is crucial to combat hemoglobin peroxidation. Their behavior upon exposure to pesticides varies widely depending on the concentration to which they are exposed and the nature of the pesticide. Glutathione S-transferase is a dimeric enzyme responsible for phase 2 biotransformation of various electrophilic compounds, a mediator of tyrosine catabolism and prostaglandin biosynthesis, as well as cell apoptosis [69]. They are divided into three groups: cytosolic, mitochondrial, and membrane-associated proteins. When exposed to pesticides, they can be considered an inducer of enzyme activation.

Several studies have demonstrated that pesticides can significantly alter the antioxidant systems of non-target organisms, thereby affecting their ability to neutralize oxidative stress. In *Labeo rohita* fish exposed to the insecticide pyriproxyfen, a significant increase in oxidative stress biomarkers (such as ROS and thiobarbituric acid-reactive substances (TBARS)) was observed, along with a decrease in the activity of antioxidant enzymes such as SOD, glutathione peroxidase (GPx), peroxidase (POD), and CAT [70]. Similarly, in prepubertal mice treated with malathion, a depletion of SOD, CAT, and GPx was reported, along with a specific reduction in the expression of GPx-4 in the liver and GPx-3 in the kidney, indicating severe damage to liver and kidney function [71]. In honeybees (*Apis mellifera*), the combination of the pesticides imidacloprid and amitraz with the fungal compound zymosan A resulted in synergistic inhibition of SOD and glutathione-S-transferase (GST), although CAT activity remained unchanged. These findings demonstrate that pesticides from different classes converge to affect redox balance, weakening key antioxidant defenses such as SOD, GPx, CAT, and GST, and increasing the organism's vulnerability to oxidative damage and disease [72].

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Genotoxic damage is a significant risk factor in evaluating potential diseases resulting from repeated exposure to pesticides over several years. Genotoxicity-related damage is of various kinds (carcinogenic, neurological, and reproductive processes). Genetic alterations occur through mutagenic and non-mutagenic processes, with the first signs being the appearance of micronuclei, chromosomal aberrations, and nuclear abnormalities, which are the most frequently observed. Research in the field confirms a strong relationship between occupational exposure to pesticides and the development of these types of damage. The different kinds of pesticides produce this genotoxic damage, in addition to their effects on the immune, nervous, and endocrine systems [73].

Various investigations have examined the health risks associated with pesticide exposure from different perspectives, considering the conditions under which they interact with xenobiotics. Singh et al. [74] determined the relationship between polymorphisms and susceptibility to genotoxic damage from organophosphate pesticides in farm workers. This point escalates further when addressing the possibility that various diseases may be triggered by prenatal exposure. A relationship was found between prenatal exposure in children and the development of a polymorphism in the paraoxonase-1 (PON1) gene, based on a substitution of glutamine for arginine at position 192, which modifies normal antioxidant properties [75].

Children exposed prenatally had similar characteristics to unexposed children, such as a larger waist circumference, a higher percentage of body fat, higher blood pressure, serum concentrations, and metabolic biomarkers, compared to children not exposed to pesticides [76]. These antecedents have made a significant contribution to linking pesticide exposure with the development of type 2 diabetes mellitus and cardiovascular diseases due to the genotoxic damage and mutations it can cause.

Hilgert Jacobsen-Pereira et al. [73]found that metabolic biomarkers, such as CAT and TBARs, were modified in exposed individuals compared to unexposed individuals, indicating consistently greater oxidative stress. There was also a greater number of micronuclei in the cells analyzed compared to those of unexposed individuals, indicating genotoxic damage. The damage caused by pesticide exposure or poisoning covers a wide range of effects, which is why there are different approaches to studying their toxicity. Among the most common tests evaluating the toxicological potential of pesticides are the following, as shown in Table 2.

Table 2. Main toxicological tests for pesticides.

Test name	Evaluated focus	Basis	Reference
Acute Toxicity Evaluation (Oral, dermal, inhalation) • LD_{50} • LC_{50} • Skin irritation test (Draize Skin Test) • Eye irritation test (Draize Eye Test) • Acute inhalation test (Exposure of animal models in chambers)	Acute toxicity tests.	Designed to assess the immediate effects of exposure to different pesticides. Tests are classified by exposure routes and evaluated within 24 to 96 hours.	[77–79]
Chronic Toxicity Evaluation • Carcinogenicity studies (OECD TG 451) • Prenatal developmental toxicity study (OECD TG 414) • Reproductive toxicity study (OECD TG 416)	Chronic toxicity tests.	Chronic toxicity tests evaluate the effects of prolonged and repeated low-dose exposures.	[77–79]

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Genotoxicity Tests • Ames test • Micronucleus test (OECD TG 487) • Comet assay • Chromosomal aberration test (OECD TG 473)	Toxicological studies based on the pesticide's ability to damage DNA and cause point mutations.	Due to the high reactivity of pesticides, they can induce mutations, chromosomal aberrations, or DNA strand breaks. These tests encompass the main mechanisms of DNA damage caused by pesticides.	[80–83]
Neurotoxicity Studies • Behavioral tests • Measurement of cholinesterase inhibition • Functional tests in rats or mice (Functional Observational Battery)	Evaluation of pesticide effects on the central nervous system, especially those caused by organophosphates and carbamates.	By detecting inhibition of key enzymes in the central nervous system, it is possible to identify motor or behavioral alterations in animal models and relate them to cognitive impairment.	[84–86]
Toxicokinetic Assays • ADME tests (Absorption, Distribution, Metabolism, and Excretion) • Radio-labeled isotopes • In vitro models (Cell cultures simulating liver metabolism)	General evaluation of the pesticide.	Analyzing ADME helps understand how long a pesticide can remain reactive in the body and where it might accumulate.	[87–89]
Biochemical Tests • Cholinesterase inhibition (Ellman test) • Alterations in liver enzymes (Alanine transaminase and aspartate transaminase)	Evaluation of alterations in enzymatic systems based on the central nervous system.	These tests assess the pesticide's effects on specific metabolic and enzymatic systems, usually in the liver or nervous system, depending on the pesticide's nature.	[90–92]

4. Pesticides in Food and Their Effect on Human Health

The presence of pesticide residues in food is an undeniable reality. Numerous studies have reported that fruits and vegetables frequently contain trace amounts of these chemicals, in some cases exceeding the maximum residue limits (MRLs) established for consumer safety [93,94]. The leading causes of these residues are often related to improper pesticide use, such as applications made outside the recommended pre-harvest intervals or at concentrations higher than those approved to preserve crop yield and quality. These poor agricultural practices increase the likelihood of detecting pesticide residues above legal limits, thereby posing a potential health risk to consumers [95]. The problem posed by this situation escalates significantly when evidence of contamination is found outside of agricultural fields where direct pesticide treatment is carried out. Jia et al. [96] noted that the migration of pesticide residues into foods of animal origin and during the breeding of these species could lead to accumulation, migration, and the formation of secondary metabolites, which pose a potential risk to human health. Among the foods identified are meat, eggs, and milk [96,97].

The risks associated with consuming foods containing pesticide residues are varied; in general, they are attributed to an increased risk of developing chronic degenerative diseases, such as various types of cancer or endocrine and reproductive disorders [95]. Pesticides are known as potential mutagens due to the reactivity of their active ingredients. Although these vary depending on the nature of the chemical, the main damages are usually chromosomal aberrations and DNA damage. This mechanism has been linked to accelerated telomere shortening thanks to exposure to and consumption of pesticides, mainly organochlorines. Five groups of occupational exposure associated

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with this phenomenon have been identified: pesticides, organic solvents, dust and particles, metals, and ionizing radiation [98,99].

The damage mentioned above is focused explicitly on modifications resulting from interactions with pesticides. However, consuming foods with food residues produces various health effects. Table 3 presents various health effects associated with the consumption of foods containing residues and exposure to pesticides.

Table 3. Health effects of pesticide consumption and exposure.

Pesticide	Study	Conclusion	Reference
Mixtures of organochlorine and organophosphate pesticides, most notably 2,4-DDE, 4,4-DDE, g-BHC, and b-BHC.	A group of 29 adolescents was studied, with 75% of them belonging to families of agricultural day laborers. Additionally, 43.7% had gardens at home, and 64.28% used pesticides. The study linked interactions with pesticides to menstrual cycle disruption.	In serum levels of sexual hormones, more than 40% of adolescents presented alterations in their hormonal profile, and 96.9% of adolescents had detectable plasma levels of pesticides. However, some indications suggest a relationship between 4,4-DDE in plasma and alterations in the menstrual cycle; no statistically significant differences were found. This may be due to the group chosen and the time designated for the study.	[100]
More than 100 pesticides classified as carcinogenic by the EPA	Meta-analysis of the presence of pesticides in different fruits and vegetables	Within the study, various pesticides found in fruits and vegetables, including grapes, mangoes, tomatoes, strawberries, apples, and peppers, were compiled. These pesticides are widely linked to the development of chronic degenerative diseases, alterations in the endocrine system, and disruptions in reproductive health in both adults and children.	[101]
91 samples were identified as exceeding the permitted MRLs in Korea, including Chlorfenapyr, Procymidone, Etofenprox, Pendimethalin and Fluopyram	1,146 fruits and vegetables were collected from the Korean market and tested for 15 pesticides of interest.	Although the identified pesticides are related to damage to the central nervous system, endocrine system, and liver conditions, it is necessary to note that it was only 8.9% of the total samples, compared to other countries, where this percentage is lower.	[102]
Pesticides such as DEE, DDT, dieldrin, and HCB	The factors influencing the presence of organochlorine pesticides in breast milk and the resulting damage to children were addressed.	Organochlorine pesticides can act as endocrine disruptors, and estrogen-inducing pesticides can accumulate with exposure to water, soil, environmental exposure, or food. Levels of HCB or DDT residues have been linked to decreased birth weight and head circumference. The opposite effect can occur with certain OGC pesticides, thanks to lipogenesis.	[103]

2,4-DDE: 2,4-Dichlorodiphenyldichloroethylene; 4,4-DDE: 4,4-Dichlorodiphenyldichloroethylene; b-BHC: Beta-hexachlorocyclohexane; DEE: Diethyl ether; DDT: Dichlorodiphenyltrichloroethane; EPA: Environmental Protection Agency; HCB: Hexachlorobenzene; g-BHC: Gamma-hexachlorocyclohexane (Lindane); MRLs: Maximum Residue Limits.

5. Nanotechnology as an Alternative to Pesticides in Agriculture

Nanotechnology is emerging as a promising alternative to alleviate the environmental and health impacts of conventional pesticides, offering more precise, biodegradable, and efficient

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solutions. Nanotechnology holds significant promise for addressing prevalent agricultural challenges, particularly through the development of nanofertilizers, nanopesticides, and nanobiosensors utilizing nanomaterials. These innovations aim to increase crop yield and mitigate adverse environmental impacts [104,105]. This review will focus on nanotechnology as an alternative to pesticides in agriculture (Figure 4).

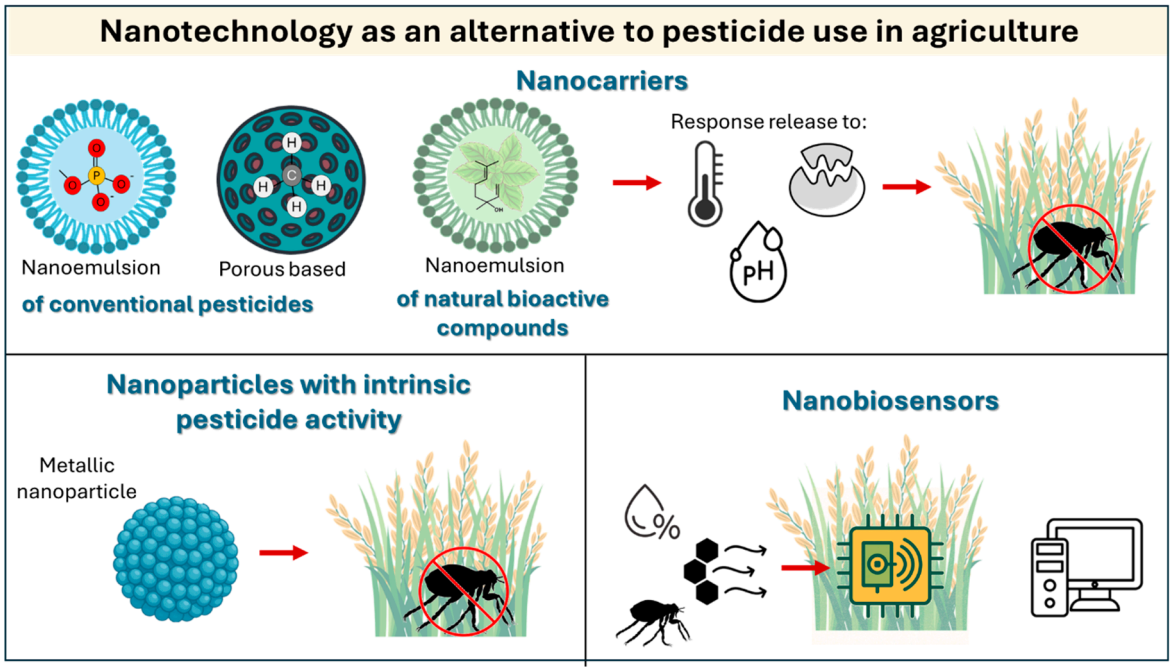

Figure 4. Applications of nanotechnology as an alternative to the use of pesticides in agriculture.

It is essential to highlight that pesticides play a crucial role in agriculture, enabling the production of a greater number of crops without wasting and providing food for the population. Furthermore, without them, the loss of human lives could be significant due to vector-borne diseases and crop losses [106]. However, as mentioned above, they have adverse effects on the environment, ecosystem, and human health because only a small amount of the applied pesticides reaches the target pests, and the rest is dispersed into the environment due to their volatility and leaching. In this sense, nanotechnology, through the development of different materials, is a viable option to overcome these limitations. Thanks to their slow degradation and the controlled release of active ingredients through the use of appropriate nanomaterials, these products enable effective pest control over extended periods.

Unlike conventional pesticides, nanopesticides exhibit distinct behaviors that improve their effectiveness [107]. Nanopesticides are nanoscale materials (measuring between 1 and 100 nanometers, or even larger, depending on the application), where the physical and chemical properties of the compounds are significantly modified, giving them unique advantages. One of the primary characteristics of these nanomaterials is their high surface-to-volume ratio, which increases their contact area and enhances adherence to plant surfaces or the bodies of pest insects, also facilitating penetration through biological barriers [105,108,109]. Furthermore, they enable the encapsulation of compounds and the creation of systems for the prolonged, targeted, and specific release of the active ingredient, which responds to specific environmental stimuli, such as changes in pH, temperature, or the presence of enzymes specific to the pest organism [110,111]. Furthermore, these nanomaterials can be functionalized with molecules that bind to specific receptors of the biological target, thereby increasing their precision and reducing side effects.

Nanoencapsulation also protects the active ingredient from adverse environmental factors, such as ultraviolet radiation, humidity, and volatilization, thereby increasing its stability and prolonging its activity in the field, which allows for reduced application frequency. Nanoformulations also

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improve the solubility of many poorly water-soluble pesticides, increasing their bioavailability and, consequently, their efficacy. Furthermore, by increasing the solubility of the active ingredient, these systems facilitate its mobility and biodegradation by microorganisms present in the soil. For these reasons, nanoparticle-based pesticides are considered to have a lower environmental impact compared to their conventional counterparts [106]. Moreover, some nanomaterials, especially metallic nanoparticles such as silver, zinc, or copper, possess intrinsic pesticidal effects. These materials can act by ROS, releasing metal ions that are toxic to pest cells or disrupting fundamental cellular processes, thereby providing new modes of action that complement or replace traditional pesticides [112].

Nanopesticides are developed from a wide range of materials and formulations. One of the most common applications is the nanoencapsulation of conventional pesticides. It involves encapsulating the active ingredients of synthetic pesticides, such as insecticides, fungicides, and herbicides, in nanovesicles (e.g., liposomes and micelles), polymeric nanocapsules (e.g., chitosan, starch, and polylactic acid), or inorganic matrices (e.g., silica and zeolite). This improves their stability, reduces leaching and volatilization, and allows for gradual and targeted release [111]. For example, azoxystrobin, a fungicide, was encapsulated in porous silica and compared with the unencapsulated pesticide during application to agricultural soils under control conditions with *Solanum lycopersicum* (tomato) plants [113]. The results showed that the conventional pesticide had a severe impact on plant growth, reducing biomass by 3.85 times, whereas the nanoencapsulated formulation allowed for healthy plant growth. At the level of soil microbiota, no significant changes were observed with either formulation, indicating low environmental toxicity. Initial absorption of the pesticide was higher with the conventional form (2.7 times more per unit of biomass in 10 days), but this led to phytotoxicity due to its high bioavailability. In contrast, nanoencapsulation enabled a slow and controlled release, which prevented toxic effects and, after 20 days, resulted in greater total absorption and absorption by dry biomass (3 and 10 times more, respectively) [113].

Another application of nanotechnology is the use of nanoparticles with intrinsic pesticide activity. Specific nanomaterials, such as metal oxide nanoparticles (ZnO, CuO, AgNPs, TiO_2) or carbon nanoparticles (carbon nanotubes, graphene), have demonstrated insecticidal, fungicidal, and bactericidal properties on their own, even at low concentrations [106]. This opens the door to green pesticides that do not contain synthetic chemical active ingredients. In this context, suspensions of ZnO-NPs at three concentrations (300, 1000, and 3000 ppm) were tested on controlled populations of *Puto barberi (mealybug) [114]*. The results showed a mortality rate of approximately 55%, demonstrating its potential as a nanopesticide. However, the authors emphasize that additional studies, particularly focused on surface characteristics and other physicochemical parameters, are needed to optimize the effectiveness and safety of the nanomaterial in agricultural applications [114]. Similarly, green-synthesized CuO nanoparticles exhibited dual functionality, demonstrating high insecticidal efficacy against storage pests and stimulating plant growth in wheat. Specifically, CuO-NPs were tested against *Sitophilus granarius* and *Rhyzopertha dominica,* observing mortality rates of 55–94.4% for *S. granarius* and 70–90% for *R. dominica* [115]. In the same approach, ZnO-NPs biosynthesized with the alga *Ulva fasciata* demonstrated superior efficacy as nematicides compared to their bulk form or to oxamyl alone [116]. Their ability to adhere to nematodes and potentially cause structural disruption represents a promising and environmentally friendly strategy for nematode control in plantain crops. The treatment with ZnO-NPs + oxamyl (chemical nematicide) produced the highest mortality of second-stage juveniles (J2s) with 98.91% after 72 hours, while ZnO-NPs alone reached 72.86% mortality. Under *in vivo* conditions, the same treatment significantly reduced the J2 population in the soil (82.77%) and the number of root galls (81.87%). However, treatments with ZnO-bulk + oxamyl and oxamyl alone promoted the most significant plant growth in terms of shoot height and weight [116].

Nanoemulsions and nanosuspensions are another approach to applying nanotechnology in the delivery of conventional pesticides. These formulations enable the active ingredient to be presented in nanometer-sized droplets or particles, thereby increasing its efficacy and improving the dispersion

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and penetration of poorly soluble pesticides [117]. Zhang et al. [118] developed an efficient and environmentally safe pyraclostrobin nanoemulsion, with improved retention and lower surface tension, which allows for better plant adhesion and dispersion. In addition, the formulation showed high bactericidal activity and lower toxicity in zebrafish, representing a sustainable alternative to conventional pesticide formulations. Similarly, a nanoemulsion of fenpropathrin, a lipophilic insecticide, presented an LC_{50} of 50.01 mg/L against *Helicoverpa armigera* larvae, a lower value than the commercial formulation, indicating greater insecticidal efficacy [119]. Treated larvae showed darkening, shrinkage, and rapid immobilization. Regarding toxicity to the earthworm *Eisenia fetida*, the LC_{50} values were 96.60 mg/kg at 7 days and 47.99 mg/kg at 14 days, suggesting low toxicity in soil. Regarding cellular cytotoxicity, cell viability in human L02 cells was 85.32% and 68.95% after 24 and 48 hours of exposure, respectively. Furthermore, the apoptosis rate was significantly lower with the nanoemulsion (8.52%) compared to the commercial formulation (63.18%), demonstrating lower cytotoxicity associated with the use of the nanoemulsion [119].

On the other hand, nanotechnology has also been used to encapsulate bioactive compounds, such as natural compounds extracted from plants, other than pesticides. In this regard, biodegradable nanoparticles of zein, a corn protein, were prepared for the encapsulation of limonene and carvacrol [110]. This system proved to be a targeted release system, activated by insect intestinal enzymes, allowing for controlled and specific release. The formulations were stable over time and nontoxic to bean plants (*Phaseolus vulgaris*). Furthermore, in *in vivo* tests with *Spodoptera frugiperda* larvae, the nanoparticles showed higher mortality compared to controls, indicating high insecticidal efficacy [110]. In a similar approach, the efficacy of nanoemulsions formulated with essential oils of basil, cumin, marjoram, and chamomile as botanical insecticides against *Aphis craccivora* was evaluated, compared to chemical insecticides such as dinotefuran and pymetrozine [120]. These nanoemulsions exhibited high toxicity against both laboratory and field strains of the cowpea aphid A. craccivora, with basil having an LC_{50} of 45 mg/L, compared to 992 mg/L for the unencapsulated essential oil and the synthetic insecticides. This was attributed to the small size of the nanoemulsion and its ability to penetrate the body of the pest. Furthermore, significant enzymatic alterations were detected in treated insects, suggesting toxicity mechanisms associated with the inhibition of key enzymes such as acetylcholine esterase [120]. These studies indicate that nanotechnology can be utilized to promote more sustainable and environmentally safe pest management.

One of the primary applications of nanotechnology is the development of nanobiosensors, which are now being utilized as a crucial tool for rapid and sensitive detection of pathogens or pests, enabling earlier and more localized intervention and reducing the need for widespread pesticide applications. Additionally, nanosensors are used to identify soil moisture levels, pesticide residues, and nutrient requirements [121]. Nanobiosensors possess essential properties, including smaller detection limits, robustness, selectivity, sensitivity, rapid response times, high surface-to-volume ratios, and cost-effectiveness, that contribute to their overall effectiveness. Detection limits reported in studies are at the parts per trillion level for atrazine, ranging from nanomoles to micromoles for acetamiprid, and at nanogram levels for glyphosate and glufosinate [122].

Biosensors function by detecting a specific stimulus and converting it into a measurable signal, such as an electrical wave, heat, or another quantifiable response. This conversion typically occurs through chemical or enzymatic reactions or via light absorption. These sensors are capable of detecting changes in wavelength, intensity, polarity, light phase, and, in some cases, fluorescence [123]. One of the most common applications of biosensors is the use of colorimetric methods. For example, Ahmed et al. [124] proposed the use of red-colored selenium particles, a product of bioreduction with heavy metals, for identification and toxicity testing in the field. Similarly, a colloidal gold immunochromatographic assay using nanobodies as recognition elements was developed to detect parathion, an organophosphate pesticide banned due to its high toxicity in agricultural products [125]. Under optimal conditions, the assay demonstrated good recoveries in detecting parathion in real samples of cabbage, cucumber, and orange, with a high correlation to UPLC-MS/MS analysis, thereby validating its accuracy [125].

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Additionally, the electronic nose (e-nose) and electronic tongue (e-tongue) are examples of biosensors used in agriculture and food analysis [126,127]. These gadgets replicate the sensory roles of the human tongue and nose, respectively. With its combination of chemical sensors and pattern recognition software, the e-nose can detect and distinguish volatile organic compounds with great precision, making it a powerful tool for identifying particular odors or alterations in aroma profiles [128,129]. In a like manner, the e-tongue employs a collection of non-specific, cross-reactive sensors to identify dissolved substances associated with taste (such as bitterness, sweetness, sourness, saltiness, and umami) [127]. These sensors produce electrical signals when they detect chemical stimuli, and these signals are analyzed using statistical or machine-learning models. Both tools have extensive applications in the quality control of food products, monitoring crop freshness, detecting contamination or spoilage, and assessing the impact of various agricultural treatments on flavor and aroma. Because they are highly sensitive, respond quickly, and perform non-destructive analyses, they serve as valuable substitutes for traditional sensory panels and chromatographic techniques [105,127].

The adoption of nanotechnology in pest management offers multiple benefits, including reduced pesticide use, as increased efficiency and direct delivery can result in lower amounts of active ingredients, thereby reducing costs for farmers and minimizing environmental impact. The use of nanotechnology in agriculture helps reduce soil and water contamination by lowering the risks of volatilization and leaching, thereby decreasing the accumulation of toxic residues in the environment and throughout the food chain. Furthermore, nanotechnology, by offering controlled and even targeted release, could present lower toxicity to non-target organisms, control pesticide resistance in pests, and increase safety for pesticide applicators in the field. Moreover, nanoencapsulated pesticides or active ingredients can offer greater stability and a longer shelf life.

However, despite their many advantages, the adoption of nanopesticides in agriculture faces several challenges. Further studies are needed to assess the toxicity of these technologies to different organisms, as well as their persistence in the environment, bioaccumulation in the food chain, and long-term effects on non-target organisms and human health. Production costs and the lack of clear regulatory frameworks for commercializing these nanopesticides also need to be addressed. Public education and acceptance of these emerging technologies are crucial components for their practical application. With rigorous research, appropriate regulatory frameworks, and effective dissemination strategies, nanopesticides have the potential to become a vital tool for more efficient, sustainable, and environmentally responsible agriculture.

6. Regulations

The increasing awareness of the potential harm associated with pesticide use has led to the implementation of extensive legislation regulating these chemicals. However, such regulations vary significantly across countries, depending on specific crop requirements and legal thresholds for plant protection [130]. Typically, developed nations tend to enforce stricter regulations than developing countries, mainly due to differences in available resources and experience in implementing and monitoring pesticide legislation [131]. These disparities often pose challenges to international trade, prompting efforts to harmonize standards at the global level. To address this, organizations such as the Codex Alimentarius Commission, the North American Free Trade Agreement (NAFTA), and the European Union have sought to harmonize standards by establishing Maximum Residue Limits (MRLs). Nonetheless, these limits still differ across regions, influenced by economic interests [132].

Among the bodies responsible for legislating, the Food and Agriculture Organization of the United Nations (FAO) establishes international standards through the Codex Alimentarius, which are not mandatory. In the United States, pesticide regulation is overseen by the Environmental Protection Agency (EPA) through the Federal Insecticide, Fungicide, and Rodenticide Act. The EPA, in collaboration with the U.S. Food and Drug Administration (FDA) and the U.S. Department of Agriculture (USDA), is responsible for setting MRLs and ensuring the safe use of pesticides, including the sale, distribution, storage, waste disposal, and usage records [133].

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The European Union is also recognized as having the most stringent regulatory system, as outlined in Regulation (EC) No. 1107/2009, which governs the marketing and authorization of plant protection products. This regulation prioritizes human and environmental safety, requiring comprehensive testing to verify the efficacy and safety of pesticides before they are authorized [134,135]. Despite these advances, regulatory gaps remain. For instance, in Mexico, approximately 141 pesticides are currently in use that are banned in other countries. As of 2017, many of these substances were still classified as highly hazardous pesticides by the FAO, underscoring the need for greater international alignment and enforcement in pesticide regulation [136].

7. Conclusions

Pesticides are undoubtedly effective at controlling pests when used appropriately and in accordance with regulatory guidelines, but their widespread and sometimes indiscriminate application has been linked to a range of adverse effects. Notably, long-term and chronic exposure has been linked to the development of chronic-degenerative diseases, including cancer, due to the oxidative stress generated. Oxidative stress and genotoxic damage caused by pesticide exposure have been documented from prenatal development through old age, highlighting the urgency of addressing this public health concern. Given the entrenched use of pesticides in global agriculture, a preventive and proactive approach is necessary.

Nanotechnology offers a promising and sustainable alternative to traditional pesticides, providing innovative solutions that improve effectiveness while reducing environmental and health risks. Controlled, targeted, and prolonged release of active ingredients can be achieved through nanocarriers, nanoencapsulation, and the intrinsic pesticidal activity of diverse nanomaterials. This reduces off-target effects and pesticide overuse. Moreover, incorporating nanobiosensors into agricultural frameworks facilitates real-time surveillance of pests, pathogens, and agrochemical residues. This capability enables more targeted actions and reduces dependence on the use of broad-spectrum pesticides.

Importantly, nanotechnology can play a significant role in minimizing environmental contamination by reducing pesticide runoff, leaching into soil and water systems, and volatilization into the atmosphere. This reduces the buildup of harmful residues in ecosystems and the food chain. Nanoformulations may reduce exposure risks for farmers and consumers from a human health standpoint due to their enhanced delivery mechanisms and reduced dosage needs. Nevertheless, the long-term impact of nanopesticides on non-target organisms and the potential for bioaccumulation are still crucial subjects for research.

Even with these advancements, obstacles persist regarding the long-term toxicity, environmental durability, and regulatory oversight of agricultural nanomaterials. To ensure their safe deployment, understand their interactions with ecosystems and food chains, and aid in developing standardized risk assessment protocols, further interdisciplinary research is crucial. With suitable regulatory frameworks and public acceptance, nanotechnology has the potential to transform pest management practices, leading to a more efficient, resilient, and environmentally responsible agricultural future.

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Abbreviations

The following abbreviations are used in this manuscript:

2,4-D	2,4-Dichlorophenoxyacetic acid
2,4-DDE	2,4-Dichlorodiphenyldichloroethylene
4,4-DDE	4,4-Dichlorodiphenyldichloroethylene
b-BHC	Beta-hexachlorocyclohexane
CAT	catalase
DDT	dichlorodiphenyltrichloroethane
DDT	Dichlorodiphenyltrichloroethane
DEE	Diethyl ether
e-nose	Electronic nose
EPA	Environmental Protection Agency
FAO	Food and Agriculture Organization of the United Nations
FDA	Food and Drug Administration
e-tongue	Electronic tongue
g-BHC	Gamma-hexachlorocyclohexane (Lindane);
GPx	glutathione peroxidase
GST	glutathione-S-transferase
HCB	Hexachlorobenzene
LD_{50}	Median lethal dose
MRLs	Maximum Residue Limits.
MRLs	Maximum Residue Limits
RNS	Reactive nitrogen species
ROS	Reactive oxygen species
SOD	Superoxide dismutase
TBARS	Thiobarbituric acid reactive substances
UPLC-MS/MS	Ultra Performance Liquid Chromatography coupled with Tandem Mass Spectrometry
USDA	U.S. Department of Agriculture

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