International Journal of Advanced Research and Multidisciplinary Trends (IJARMT)
An International Open Access, Peer-Reviewed Refereed Journal
Impact Factor: 6.4 Website: https://ijarmt.com ISSN No.: 3048-9458

Evaluating The Hazards Impacts of Monocrotophos on the Environmental Contamination & Animal Health

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Abstract
The synthetic pesticide monocrotophos (MCP) serves as a widely utilised chemical of its category. The organophosphate pesticide retains its important status because it effectively controls pests within rice and maize crops as well as sugarcane and cotton and vegetable cultivation. The global application of this pesticide has established serious ecological risks that also trigger health problems. The review paper executes a risk assessment of MCP by analysing environmental pollutants which affect animals alongside their health issues. This review draws all its content from peer-reviewed PubMed publications to investigate MCP distribution within environmental elements from the land and sky elements while evaluating MCP dispersal mechanisms and longevity and accumulation behaviour. The scientific research assesses toxicological damage caused by MCP throughout separate animal populations starting from livestock animals until wild species and terminating with aquatic organisms. The review provides numerical data regarding MCP exposure levels while documenting its biological and biochemical effects and confirmed mechanisms of toxicology that involve cholinesterase blockage and oxidative stress effects leading to mitochondrial breakdown. The research evidence shows that MCP reaches extensive ecological zones by disrupting animal wellness which causes biodiversity losses and food contamination. The document presents educational data to lawmakers and experts in research and industry about why proper regulatory oversight and sustainable options need scientific evidence for advancement. The implementation of MCP risk reduction requires complete awareness about these dangers for developing safety protocols regarding agricultural biological and environmental standards.

Keywords*:* Monocrotophos, Animal toxicity, Ecotoxicity, Environmental contamination

Introduction:

Organophosphate pesticides represent a significant class of synthetic insecticides widely employed in agriculture to protect crops from a variety of pests.1 Among these, monocrotophos (MCP) has been extensively utilized globally due to its effectiveness and broad-spectrum properties against numerous agricultural pests.[1] This organophosphate insecticide has been crucial in enhancing agricultural yields across various crops, including rice, maize, sugarcane, cotton, and vegetables.6 However, the escalating global concern regarding the environmental and health impacts of pesticide use, including MCP, necessitates a comprehensive evaluation of its hazards.1 The widespread application of MCP, coupled with its known toxicity, highlights the critical need for a thorough understanding of its environmental fate and the associated health risks to inform effective management and regulatory strategies. Assessment of monocrotophos hazards depends on constructing a complete scientific method which

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examines soil and water and air contamination due to its toxicity and its impacts on livestock health and wildlife health and aquatic life health. Scientific peer-reviewed articles discovered through PubMed databases enable the environmental assessment because these articles provide quantifiable data supported by proven toxicological mechanisms. A comprehensive review of toxic risks exists in all environmental areas and multiple animal species providing a complete knowledge of MCP's diverse hazards.

2. Monocrotophos in the Environment: Contamination and Persistence

2.1. Soil: Quantification, Persistence, and Factors Influencing Levels

The insecticide monocrotophos becomes widely detected in agricultural soil because farmers use it directly for crop protection.[1] Numerous studies found significant levels of MCP in different agricultural zones where researchers collected soil measurements that confirmed its prevalent distribution in these environmental settings.[16] Soils act as main determinants of exposure times and water source leaching risks due to monocrotophos persistence. Research indicates that the half-life of MCP in soil can vary significantly, ranging from days to months, depending on prevailing environmental conditions.[5] For instance, a study conducted on specific Indian agricultural soils, namely black vertisol and red alfisol, demonstrated rapid degradation of MCP at both low and high concentrations under aerobic conditions, with half-lives of 9.2 and 11.4 days, respectively.[16] This rapid degradation in certain soil types contrasts with the potential for much longer persistence under stable storage conditions, where technical grade MCP can remain stable for extended periods.[5]

Several reports jointly determine the duration of MCP remains present within soil. Temperature and pH are key determinants, affecting the rate of chemical degradation.5 Soil type and composition, including organic matter content and clay content, also play a significant role in the sorption and degradation of MCP.[16] Microbial activity is another crucial factor, as various microorganisms in the soil can biodegrade MCP.[17] Water content and the depth of the water table can further influence the movement and degradation of MCP in the soil profile.[16] Research has elucidated the degradation pathways of MCP in soil, primarily involving hydrolysis, where the molecule is broken down by reaction with water, and microbial breakdown, where microorganisms utilize MCP as a carbon and energy source.[1] One of the identified metabolites during this degradation process is N-methylacetoacetamide.[16] Furthermore, studies have explored the potential of bioremediation techniques using specific bacterial consortia to enhance the degradation of MCP in contaminated soils.[17] The variability in MCP's persistence in soil is not a fixed characteristic but rather a dynamic property shaped by the complex interaction of these environmental factors and biological activity. Soil organism exposure duration along with MCP transfer into groundwater depend heavily on the various environmental conditions which produce significant impacts on toxicity assessment.

2.2. Water: Quantification, Persistence, and Implications for Aquatic Ecosystems

Agricultural runoff and leaching from contaminated soils contribute significantly to the contamination of both surface water and groundwater with monocrotophos.[1] Studies have detected varying levels of MCP in diverse water bodies, highlighting the extent of this contamination.[15] The persistence of MCP in aquatic environments is influenced by factors such

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as pH and temperature, which affect its hydrolysis rate.[5] For instance, the half-life of MCP in natural water with a pH of 7.6 has been reported as 147 days at 25°C and 29 days at 35°C, indicating that higher temperatures accelerate its degradation.[15]

The application of MCP, which has been very useful due to its water solubility, also renders the aquatic environment particularly vulnerable to contamination and the duration of exposure of aquatic life and the possibilities for downstream contamination are dictated by its persistence in water.[4] Microbial degradation also plays a role in the removal of MCP from water, and specific microorganisms capable of degrading MCP have been identified in water and wastewater treatment systems.[28] Additionally, research has explored the use of photocatalytic degradation as an alternative method for the removal of MCP from contaminated water.[6] To mitigate water contamination from water-soluble pesticides like MCP, the development of encapsulated formulations that provide a controlled release of the pesticide is being investigated.[26]

2.3. Air: Evidence of Contamination and Potential Pathways

Monocrotophos can potentially contaminate the air through spray drift during its application in agricultural fields.[7] While direct quantification of MCP in air samples is not detailed in the provided snippets,[30] the practice of aerial application inherently suggests a pathway for atmospheric contamination.[30] Volatilization from soil and water surfaces also represents a potential route for MCP to enter the air.[37] A study on the toxicological effects of aerial application of monocrotophos on cattle, chickens, buffaloes, and human volunteers found no significant effect on cholinesterase levels in these exposed subjects.[30] However, it is important to note that MCP has been identified as an agent of concern in air pollution in relation to the increased risk of Parkinson's disease.[39] Generally, it is understood that pesticides, including MCP, can contaminate air, soil, and water, with only a fraction of the applied amount reaching the intended target pests.[7] Strategies for reducing airborne pesticide pollution, such as modifying pulverization habits and banning products with high vapor pressure, are being considered.[37] Although specific measurements of MCP in air are limited within the provided information, the evidence indicates that aerial application and volatilization are plausible pathways for air contamination. This is concerning to realise that human and animal inhalation exposure may occur in agricultural and surrounding areas.

3. Impact of Monocrotophos on Animal Health

3.1. Livestock: Toxicity, Physiological Effects, and Residue Accumulation

Monocrotophos can be introduced to livestock through several routes, namely, through consumption of contaminated feed and water and through direct contact during pesticide application.[2] Research has been made to ascertain the acute and chronic toxicity of MCP in different livestock species. For instance, aerial application of MCP in cattle, chickens and buffaloes does not greatly influence cholinesterase levels;[30] however, repeated oral administration of MCP to buffalo calves' results in significant reduction of the total number of protozoa in the buffalo rumen probably by affecting the rumen micro biota.[44]

Different physiological effects can be induced in livestock by exposure to MCP. These include neurological symptoms such as confusion, agitation, hypersalivation, and convulsions.[5]

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Research on broiler chicks revealed that chronic toxicity from MCP can affect bone health, leading to destructive changes in the femur.[43] Furthermore, the general effects of organophosphate pesticides, including MCP, can potentially lead to aberrations in embryonic development and defects in neurocognition.[40] Biochemical modifications and adverse clinical outcomes, such as alterations in haematological parameters and increased activities of liver enzymes, have also been associated with pesticide exposure in general.[42] Notably, MCP residues can accumulate in animal tissues, milk, and eggs, posing direct risks to the health of the animals and potentially entering the human food chain through the consumption of these products.[4] The livestock industry utilizes MCP and other organophosphates for pest control to protect animals from various infestations.[45] While some studies suggest no immediate significant effects of aerial MCP application on cholinesterase levels in certain livestock, other research indicates that sublethal chronic exposure can lead to various physiological disruptions, including impacts on bone health and rumen function. Residue accumulation in animal products have the potential for food safety and long-term health risks for animal's and human's consuming animal products.

3.2. Wildlife: Effects on Different Species, Reproductive Impacts, and Behavioral Changes

Monocrotophos can be introduced into wildlife species in the contaminated environment, including soil, water, air and vegetation especially in agricultural landscape with MCP is being used widely.[4] Many studies have addressed the extent of acute toxicity of the MCP to various wildlife species. These include fish, such as guppies and goldfish, which exhibit feminization/demasculinization of reproductive traits and altered sex steroid levels upon exposure.[32] Mussels exposed to MCP show genotoxic effects and retardation of somatic growth.15 Rats exposed to MCP have demonstrated cardiac oxidative stress, myocardial damage, neurotoxicity, and induction of insulin resistance.[49] Mice exposed to MCP have shown impacts on the striatal dopaminergic system and bone loss.[53] Earthworms also exhibit adverse effects, including increased mortality and reproductive abnormalities, upon exposure to MCP.[21]

Significant reproductive impacts of MCP on wildlife are also encountered. Male guppies exposed to MCP during sexual development exhibit feminization, characterized by increased oestradiol levels and vitellogenin synthesis, and demasculinization, indicated by decreased testosterone levels, reduced sperm count, and smaller sexually attractive spots.[48] Similarly, male goldfish exposed to MCP show elevated oestradiol and reduced testosterone levels, along with altered expression of genes involved in steroidogenesis.[32] Earthworms exposed to increasing concentrations of MCP show abnormal sperm and defective cocoons, indicating reproductive toxicity.[62] Behavioural changes have also been observed in wildlife exposed to MCP. Zebrafish larvae exposed to MCP show altered locomotor activity and increased behavioural irregularities.[40] Studies on nematodes have indicated that MCP exposure can affect motor activity.[64] Other physiological effects in wildlife include the development of cataracts in fish,[50] cardiac oxidative stress and myocardial damage in rats,[51] genotoxic effects in mussels,[52] and impacts on the dopaminergic system in mice.[53] Furthermore, MCP exposure in rats has

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been linked to oxidative stress, neuronal loss, insulin resistance, and intestinal dysfunction,[49] and in mice to cortical and trabecular bone loss.[59] Additionally, thyroid disruption has been linked to the developmental neurotoxicity of MCP.[65] The diverse toxic effects of MCP on a wide range of wildlife species, impacting reproduction, behaviour, and various physiological systems, highlight the potential for significant ecological consequences.

3.3. Aquatic Organisms: Toxicity Levels, Physiological Disruptions, and Ecological Consequences

Aquatic organisms are particularly vulnerable to the toxic effects of monocrotophos due to its presence in water bodies through agricultural runoff and leaching.[4] Studies have reported various LC50 values and other toxicity endpoints for different aquatic species. For example, the 96-hour LC50 value of monocrotophos for the Nile tilapia fish was found to be 4.9 mg/l.[33] For *Daphnia magna*, the 48-hour LC50 was reported as 388 µg/L.[34] In freshwater bivalves (*Lamellidens marginalis*), exposure to 52.36 mg/l of monocrotophos for four days resulted in significant toxic effects.[15]

Monocrotophos causes a range of physiological disruptions in aquatic organisms. A primary effect is the inhibition of acetylcholinesterase (AChE) activity in various tissues, including the brain, gills, and muscles of fish and mussels.[15] Exposure to MCP also induces oxidative stress, characterized by increased lipid peroxidation and alterations in the activities of antioxidant enzymes such as superoxide dismutase, catalase, glutathione S-transferase, and glutathione reductase.[15] Genotoxicity, indicated by the formation of micronuclei, has been observed in the fish *Catla catla* and the mussel *Meretrix ovum* upon exposure to MCP.[27] Endocrine disruption is another significant consequence, with MCP affecting sex steroid levels and inducing vitellogenin production in male goldfish and guppies, suggesting estrogenic properties.[32] In sea urchins, MCP has been shown to cause developmental neurotoxicity by disrupting cholinergic and dopaminergic neurotransmitter systems.[66] Histopathological changes, such as fibrosis in gill filaments and hypertrophy in mucous cells, have been observed in mussels exposed to MCP.[15] Furthermore, MCP can impact the swimming behavior and other motor functions of aquatic organisms.[40] The ecological consequences of MCP contamination in aquatic environments can be far-reaching, potentially affecting food webs and reducing biodiversity.[4] Aquatic organisms are often used as indicators of environmental quality due to their sensitivity to pollutants like MCP.[15]

4. Mechanisms of Monocrotophos Toxicity in Animals

4.1. Biochemical Mechanisms: Inhibition of Acetylcholinesterase and Other Enzymatic Effects

The primary biochemical mechanism underlying the toxicity of monocrotophos in animals is the irreversible inhibition of the enzyme acetylcholinesterase (AChE).[1] MCP, being an organophosphate compound, binds to the esteratic site of AChE, an enzyme crucial for the hydrolysis of the neurotransmitter acetylcholine (ACh) in the synaptic cleft.[49] This binding leads to the phosphorylation of the serine residue at the active site of AChE, resulting in its inactivation and preventing the breakdown of ACh.[49] Consequently, ACh accumulates at the neuromuscular junctions and in the central nervous system, leading to the continuous

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stimulation of cholinergic receptors and disruption of normal neurotransmission.[5] This overstimulation manifests as a range of toxic symptoms observed in animals exposed to MCP. In addition to AChE inhibition, MCP has been shown to inhibit other enzymes, including monoamine oxidase.[38] Furthermore, studies indicate that MCP can interfere with enzymes involved in steroidogenesis, affecting the synthesis and conversion of sex hormones in fish.[55] This interaction with steroidogenic enzymes contributes to the endocrine-disrupting effects of MCP. The significant role of AChE inhibition in MCP toxicity has led to the development of AChE-based biosensors for the detection of MCP in environmental samples.[69] It is also noteworthy that some insect species have developed resistance to MCP through mutations in the gene encoding AChE, resulting in an enzyme with reduced sensitivity to the insecticide.[68]

4.2. Physiological Mechanisms: Neurotoxicity, Endocrine Disruption, Oxidative Stress, and Organ-Specific Damage

The biochemical inhibition of AChE by monocrotophos translates into significant neurotoxic effects in animals. The accumulation of acetylcholine at synapses leads to cholinergic overactivity, which can manifest as a variety of neurological symptoms, including confusion, agitation, hypersalivation, convulsions, and ultimately, potential neuronal death.[1] These neurotoxic effects can also impact behavior and motor function in exposed animals. Beyond its neurotoxic effects, MCP acts as an endocrine disruptor, interfering with the normal functioning of the endocrine system. Studies have shown that MCP can alter the levels of sex hormones, such as estradiol and testosterone, and affect reproductive development in various animal species, potentially leading to feminization or demasculinization.[10]

Oxidative stress is another critical physiological mechanism through which MCP exerts its toxicity. Exposure to MCP induces the generation of reactive oxygen species, leading to increased lipid peroxidation and protein oxidation, and can deplete antioxidant defense systems in a wide range of animal species.[11] This imbalance between pro-oxidant and antioxidant mechanisms can cause cellular damage and contribute to various pathological conditions. Furthermore, MCP exposure has been associated with organ-specific damage in different animal models. For instance, it can induce cataract development in the lens of fish [50], cause cardiac oxidative stress and myocardial damage in rats [51], and lead to liver and kidney injury, as generally observed with organophosphate pesticides.[41] Intestinal dysfunction has been reported in rats following oral exposure to MCP [21], and studies in mice have shown that MCP can induce cortical and trabecular bone loss.[43] Notably, research suggests that MCP exposure can exacerbate the progression of existing neurological conditions, such as Parkinson's disease, in animal models.[53]

5. Environmental Fate and Degradation of Monocrotophos

5.1. Degradation Pathways in Soil and Water (Hydrolysis, Microbial Degradation, Photodegradation)

Monocrotophos undergoes degradation in the environment through several pathways, including hydrolysis, microbial degradation, and photodegradation. Hydrolysis, the chemical breakdown of MCP by reaction with water, is influenced by the pH and temperature of the environment.[5] Higher temperatures and alkaline conditions generally accelerate the rate of

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hydrolysis. Microbial degradation represents a significant pathway for the breakdown of MCP in both soil and water.[1] Numerous bacterial and fungal species isolated from contaminated environments have demonstrated the ability to degrade MCP, utilizing it as a source of carbon and nitrogen. Some of the identified microorganisms include *Bacillus subtilis*, *Brucella intermedia*, *Starkeya novella*, *Paracoccus sp.*, *Aspergillus flavus*, *Fusarium pallidoroseum*, *Macrophomina sp.*, and *Proteus myxofaciens*.[1] These microorganisms employ various enzymes, such as organophosphate hydrolase (opdA), phosphatase, and esterase, to catalyze the degradation of MCP.[17] The potential for utilizing microbial consortia and implementing bioremediation strategies to enhance the removal of MCP from contaminated sites is also being explored.[1]

Photodegradation, the breakdown of MCP through exposure to sunlight, is another environmental fate pathway. Studies have shown that the photodegradation of MCP can be accelerated in the presence of certain nanocomposites, which enhance the efficiency of light harvesting and the generation of reactive species involved in the degradation process.[25] Computational investigations have also explored the reaction mechanisms of MCP degradation by hydroxyl radicals in both atmospheric and aqueous environments.[78] The relative importance of these different degradation pathways is likely contingent on the specific environmental conditions, such as the availability of light, the composition of the microbial community, and the pH of the soil or water. Understanding these pathways is essential for developing effective strategies to remediate environments contaminated with MCP.

5.2. Factors Influencing Persistence: Temperature, pH, Soil Composition, and Microbial Activity

The persistence of monocrotophos in the environment is not a fixed characteristic but is significantly influenced by a multitude of interacting factors. Temperature and pH are critical determinants of MCP's persistence in both soil and water, primarily affecting the rate of hydrolysis.[4] Generally, higher temperatures and more alkaline pH values tend to decrease the persistence of MCP by accelerating its breakdown. Soil composition also plays a vital role; for instance, the organic matter and clay content of soil can influence the sorption and subsequent degradation of MCP.[16] Soils with higher organic matter content may sorb more MCP, potentially affecting its availability for degradation.

Microbial activity exerts a substantial impact on the biodegradation rate of MCP across different environmental matrices.[3] The presence of specific microorganisms capable of metabolizing MCP can significantly reduce its persistence in soil and water. Conversely, conditions that inhibit microbial growth or activity can lead to a longer persistence of the insecticide. Other environmental factors, such as rainfall and the depth of the water table, can affect the movement of MCP through soil and its potential to leach into groundwater, thereby influencing its persistence in the topsoil layers.[4] The persistence of MCP residues can also vary in different crops. Studies on cabbage have shown that the persistence of MCP residues is influenced by the application dose and frequency.[79] Notably, the persistence of technical grade MCP under controlled storage conditions can be considerably longer compared to its persistence in the open environment, highlighting the role of environmental factors in its

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degradation.[5] The context-dependent nature of MCP's persistence underscores the need to consider the specific environmental conditions of a contaminated site when assessing the long-term presence and potential hazards of this insecticide.

6. Bioaccumulation and Dietary Exposure Risks

6.1. Bioaccumulation in Terrestrial and Aquatic Food Chains

Monocrotophos has the potential to bioaccumulate in both terrestrial and aquatic food chains, posing risks to the health of organisms at higher trophic levels.[1] Biomagnification, a process where the concentration of a substance like MCP increases as it moves up the food chain, is a significant concern.[4] In aquatic ecosystems, MCP can be absorbed by smaller organisms, such as algae and invertebrates, which are then consumed by larger predators like fish. This can lead to a progressive increase in MCP concentrations in the tissues of organisms at each trophic level. For example, studies have shown bioaccumulation of MCP in aquatic organisms like fish and mussels.[4]

The accumulation of MCP residues in the edible tissues of animals is particularly concerning as it represents a direct pathway for human exposure through the consumption of contaminated food sources.[4] Research has detected the presence of MCP in various agricultural products, indicating its entry into the food chain.[11] The general understanding is that pesticides like MCP can biomagnify along food chains, potentially leading to harmful concentrations in top predators, including humans, who consume these contaminated organisms.[4] The persistence of MCP in the environment, coupled with its ability to accumulate in the tissues of living organisms, makes it a long-term concern for both food safety and the health of ecosystems.

6.2. Potential Risks to Animal Health from Dietary Intake of Contaminated Food Sources

Dietary exposure to monocrotophos residues in contaminated feed and water poses potential risks to animal health.[42] Studies have reported the presence of MCP residues in fruits and vegetables at levels exceeding the maximum residue limits, indicating a significant source of dietary exposure.[42] Chronic exposure to even low doses of MCP through the diet can lead to adverse health effects in animals. For instance, research in rats has shown that long-term dietary exposure to MCP can induce insulin resistance and metabolic dyshomeostasis.[58] Furthermore, oral exposure to MCP has been found to cause intestinal dysfunction in rats, highlighting the potential for digestive system impairment through dietary intake.[61]

The calculation of dietary exposure to organophosphate residues, including MCP, from various food sources is an important aspect of risk assessment.[81] General concerns about dietary pesticide exposure and its potential long-term health impacts are well-documented.[42] The widespread presence of MCP in agricultural products and the potential for continuous, low-level exposure through the diet underscore the necessity for rigorous monitoring and regulation of MCP use in agriculture. This is crucial for safeguarding both animal and human health from the potential adverse effects associated with the dietary intake of MCP residues.

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7. Comparative Assessment of Monocrotophos Toxicity

7.1. Comparison with Other Organophosphate Pesticides Regarding Environmental Persistence

The environmental persistence of organophosphate pesticides can vary depending on their specific chemical structures and the environmental conditions they encounter. While the provided snippets do not offer direct comparative data on the environmental persistence of monocrotophos with other specific organophosphates, they establish that MCP's persistence in soil and water is influenced by factors such as temperature, pH, soil composition, and microbial activity.[4] Generally, organophosphates are known to have shorter environmental persistence compared to organochlorine pesticides, as they are more prone to degradation through hydrolysis and microbial action. However, within the class of organophosphates, the degree of persistence can differ. For example, some organophosphates might be more water-soluble, leading to faster leaching and potentially different degradation rates in soil compared to MCP. Similarly, the rate at which different organophosphates are broken down by microorganisms can vary depending on the specific enzymes they possess. A comprehensive comparison would necessitate a broader review of the literature on the environmental fate of various organophosphate pesticides under comparable conditions.

7.2. Comparison with Other Organophosphate Pesticides Regarding Animal Health Risks (Acute and Chronic Effects)

Monocrotophos exhibits varying degrees of toxicity compared to other organophosphate pesticides in terms of animal health risks, depending on the species affected, the specific toxicological endpoint measured, and the potential for interactions with other substances.[6] Studies comparing the toxicity of MCP with its analogs in fish have shown that some analogs can be significantly more toxic than MCP.[67] Research comparing MCP with chlorpyrifos in lung cancer cells indicated that both induce oxidative stress and DNA damage, but they differ in their effects on apoptosis-related factors.[86] A study comparing the acute neurotoxicity of MCP with mevinphos, dicrotophos, and phosphamidon in rats found that mevinphos was clearly more toxic to young rats, while the other three were only moderately more toxic.[85] The common mechanism of neurotoxicity among organophosphates is the inhibition of AChE, which can lead to a range of acute and chronic effects depending on the specific pesticide and the level of exposure.[36] Notably, the potential for synergistic effects when MCP is combined with other pesticides or environmental contaminants, such as other organophosphates or arsenic, can enhance its toxicity.[34]

Table 1: Comparative Acute Toxicity (LC50/EC50) of Monocrotophos and Other Organophosphate Pesticides in Aquatic Organisms (selected examples from snippets).

Pesticide	Organism	Endpoint	Value (mg/L)	Snippet ID(s)
Monocrotophos	Nile Tilapia	96-hour LC50	4.9	[33]
Monocrotophos	*Daphnia magna*	48-hour LC50	0.388	[34]

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RPR II (analog)	*Oreochromis mossambicus*	96-hour LC50	0.167	[67]
RPR V (analog)	*Oreochromis mossambicus*	96-hour LC50	0.174	[67]
Monocrotophos	*Lamellidens marginalis*	96-hour LC50	52.36	[15]

This table provides a limited comparison of the acute toxicity of monocrotophos with some of its analogs and highlights the variability in toxicity even within closely related compounds. Monocrotophos generally exhibits significant toxicity to aquatic life, but its potency can be lower than some other organophosphate compounds.

8. Synthesis of Existing Review Literature on Monocrotophos Hazards

Existing review literature consistently emphasizes the significant hazards associated with monocrotophos, encompassing its environmental persistence, toxicity to a wide range of animal species, and potential for bioaccumulation.[2] Several reviews specifically focus on the toxicity of MCP to aquatic organisms, detailing its adverse effects on survival, behavior, reproduction, and genetic material.[6] Other reviews concentrate on the environmental fate of MCP, discussing its degradation pathways, persistence in different environmental matrices, and various detoxification strategies, including biodegradation and photocatalysis.[5] The metabolism and toxicology of MCP in animals, including livestock and wildlife, are also covered in several review articles, highlighting its absorption, distribution, metabolism, and excretion, as well as its acute and chronic toxic effects.[41] Additionally, broader reviews on pesticide hazards often include monocrotophos as a significant example of an organophosphate insecticide with substantial environmental and health risks.[1] These reviews collectively underscore the need for continued research to fully elucidate the long-term effects of MCP, the importance of stringent regulatory measures to control its use, and the necessity of developing and promoting safer alternatives to mitigate the risks posed by this insecticide to the environment and animal health.

Conclusion

This literature review has synthesized the extensive research on the hazards of monocrotophos concerning environmental contamination and animal health risks. Key findings indicate that MCP is a persistent insecticide that can contaminate soil, water, and potentially air, primarily through agricultural application. Its persistence in these environmental matrices is influenced by a complex interplay of factors, including temperature, pH, soil composition, and microbial activity. MCP exhibits significant toxicity across a wide range of animal species, including livestock, wildlife, and aquatic organisms. The primary mechanism of toxicity is the irreversible inhibition of acetylcholinesterase, leading to neurotoxic effects. However, MCP also induces oxidative stress, disrupts endocrine systems, and can cause organ-specific damage. Furthermore, MCP has the potential to bioaccumulate in food chains, particularly in aquatic ecosystems, posing risks to animal health through dietary exposure, including potential long-

term chronic effects from low-level exposure. Comparative assessments suggest that MCP's toxicity varies relative to other organophosphate pesticides depending on the species and the specific endpoint. Existing review literature consistently highlights the substantial hazards associated with MCP, emphasizing the need for careful management and regulation of its use. Several research gaps warrant further investigation. The long-term impacts of chronic, low-level exposure to MCP on various animal species and ecosystems require more comprehensive studies. Further research is also needed to fully understand the synergistic effects of MCP in combination with other environmental contaminants. Additionally, continued efforts to develop and implement effective bioremediation strategies for MCP-contaminated environments are crucial.

The implications of these findings for environmental and animal health management are significant. Regulatory policies should consider the environmental persistence and bioaccumulative potential of MCP, along with its broad toxicity profile. Agricultural practices should aim to minimize the release of MCP into the environment through integrated pest management strategies and the adoption of safer alternatives where feasible. Future research should focus on addressing the identified gaps to provide a more complete understanding of the hazards posed by MCP and to inform evidence-based risk management decisions. A balanced approach is essential, considering both the agricultural benefits of MCP and the potential environmental and health costs associated with its use.

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