

Pesticide Residues in Foods: Evaluating Human Health Hazards

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Abstract:

Food storage losses to insect and rodent pests are substantial worldwide, estimated at 10–30% in industrialized countries and up to 40–75% in developing regions and this has driven the widespread use of pesticides during post-harvest storage. Stored-product pesticides include fumigants such as phosphine, methyl bromide, and sulfuryl fluoride, as well as contact insecticides from the organophosphate, pyrethroid, carbamate, and (historically) organochlorine classes. While effective at reducing quantitative and qualitative losses, these compounds generate residues in stored commodities and may result in occupational and bystander exposure, with documented acute toxicity and growing evidence for chronic effects including cancer, neurotoxicity, endocrine disruption, reproductive toxicity, and metabolic disorders. This review consolidates recent literature on the principal pesticide classes employed in food storage, their application practices and residue behavior, routes of human exposure, and acute and chronic health outcomes, with emphasis on vulnerable groups in low- and middle-income countries. Regulatory status, residue monitoring data, and risk-assessment approaches are discussed alongside integrated pest management and safer alternatives. The evidence underscores the need to balance post-harvest protection with strengthened regulation, exposure reduction, and transition towards less hazardous technologies to safeguard human health.

Keywords—*Stored-product pests, Fumigants, Organophosphates, Pesticide residues, Food safety, Neurotoxicity*

1. Introduction

Post-harvest storage is essential to maintain year-round availability of cereals, legumes, and other staples, but unprotected stocks suffer major losses due to insects, mites, rodents, and fungi, particularly in tropical climates [1, 2]. To reduce these losses, chemical pesticides are widely used in on-farm stores, warehouses, and silos as grain protectants in the form of structural sprays and fumigants [3, 4]. These interventions have markedly improved grain preservation and food security, yet they also create multiple pathways for human exposure via contaminated food, occupational contact, and environmental contamination [5, 6]. Storage pesticides differ from field pesticides in their application context and exposure profile: they are often applied directly to dry commodities, used in enclosed or semi-enclosed spaces, and include volatile, highly acutely toxic fumigants [7,8]. Major groups include fumigants such as phosphine, methyl bromide, and sulfuryl fluoride; organophosphate insecticides like pirimiphos-methyl and chlorpyrifos-methyl; synthetic pyrethroids including deltamethrin and cypermethrin; carbamates; and, historically, organochlorines such as DDT and dieldrin [3, 9]. While these compounds are effective against key storage pests, their residues and toxicological profiles raise concerns about acute poisonings and long-term health effects in exposed populations, especially where regulatory oversight is weak [5,4]. This review provides an overview of: [1] major pesticides used in food storage globally, [2] residue behavior in stored and processed foods,

[3] human exposure pathways, and (4) acute and chronic health effects, alongside mitigation strategies and research gaps.

2. Major Pesticide Classes Used in Food Storage

2.1 Fumigants

2.1.1 Phosphine (PH₃)

Phosphine gas, generated from aluminum or magnesium phosphide formulations, is currently the most widely used fumigant for stored grains worldwide because of its broad spectrum, relatively low cost, and lack of persistent residues [7, 1]. Tablets or pellets are placed in sealed silos, bags, or stacks; phosphine diffuses through the grain mass, killing insects at all life stages, but requires adequate exposure time (often >48–72 h) and gas-tightness for efficacy [7]. Widespread and sometimes suboptimal use has led to phosphine resistance in pests such as *Sitophilus oryzae* and *Rhyzopertha dominica* in India, China, Brazil, Australia, and other countries, with resistance ratios reported up to 50-fold [10].

Phosphine is highly toxic to mammals; acute inhalation can cause non-specific early symptoms (nausea, dizziness, headache) progressing to pulmonary edema, cardiac arrhythmias, hepatic and renal damage, and death [1, 11, 12]. Chronic exposure has been associated with anemia, respiratory symptoms, and neurological disturbances in case reports and occupational cohorts [12].

2.1.2 Methyl Bromide (MB)

Methyl bromide was historically a dominant fumigant for grain, timber, and quarantine treatments, but it is a potent ozone-depleting substance and has been largely phased out under the Montreal Protocol, except for specific quarantine and pre-shipment (QPS) uses [13]. MB is rapidly absorbed by inhalation; acute poisoning causes nausea, dizziness, visual disturbances, ataxia, and can lead to delayed neurotoxicity, including peripheral neuropathy and cerebellar signs [14]. Numerous poisonings have been documented among fumigation workers, with several hundred cases and multiple fatalities reported in the literature [14]. Regulatory agencies in many countries, including Nigeria's NAFDAC, now prohibit its use in most agricultural fumigations, reinforcing the need for safer alternatives [15].

2.1.3 Dichlorvos (DDVP)

Dichlorvos is a volatile organophosphate used as a fumigant-type insecticide in storage and residential settings, typically via resin strips, aerosols, or sprays [16]. It acts by inhibiting acetylcholinesterase and is classified as "highly hazardous" (WHO Class 1B) and "possibly carcinogenic to humans" (Group 2B) by IARC [17, 18]. Regulatory agencies restrict DDVP use in areas with exposed food and have set low tolerance levels in commodities like milk [16].

2.1.4 Sulfuryl Fluoride

Sulfuryl fluoride is primarily used as a structural fumigant and as an MB alternative for wood packaging; it is effective against a range of wood-boring beetles and some stored-product pests when applied at sufficient concentration and exposure time (3). It leaves inorganic fluoride residues rather than organic pesticide residues, and concerns center on potential neurodevelopmental and skeletal effects from fluoride accumulation, particularly in children [5].

2.2 Contact and Residual Insecticides

2.2.1 Organophosphates (OPs)

Organophosphates such as malathion, chlorpyrifos-methyl, pirimiphos-methyl, and diazinon are widely used as grain protectants and structural treatments in stores (3,19). They inhibit acetylcholinesterase, causing accumulation of acetylcholine and cholinergic overstimulation, which explains both their insecticidal efficacy and neurotoxicity in humans (8,20). In Nigeria and other African countries, residues of malathion, parathion, ethion, and carbophenothion have been detected in cowpea and other stored products, often in mixtures (2).

2.2.2 Synthetic Pyrethroids

Pyrethroids, including deltamethrin, cypermethrin, fenvalerate, and permethrin, are used as grain protectants and on storage structures, often in combination with organophosphates to broaden pest spectrum and delay resistance [3, 4]. They act on voltage-gated sodium channels, prolonging depolarization of neuronal membranes [21]. Pyrethroids are generally less acutely toxic to mammals than organophosphates but are highly lipophilic and persistent on

grain surfaces, with residues concentrating in bran and outer layers [22, 4].

502.2.3 Carbamates and Insect Growth Regulators

Carbamates such as carbaryl and carbofuran share a mechanism of reversible acetylcholinesterase inhibition and are used less commonly in storage than in field crops but may appear in mixed pesticide regimes [8]. Insect growth regulators (e.g., methoprene, pyriproxyfen) and newer chemistries are being explored for stored-product pest management as part of integrated programs, often with lower mammalian toxicity [3].

2.3 Organochlorines (Legacy and Illegal Use)

Organochlorines such as DDT, aldrin, dieldrin, and endosulfan were formerly used as grain protectants and structural insecticides because of their persistence and effectiveness [9]. Due to bioaccumulation, endocrine disruption, and carcinogenicity, they are now banned or severely restricted under the Stockholm Convention [23]. Nonetheless, residues continue to be detected in grains, legumes, and animal products in some regions, reflecting legacy contamination or continued illegal use [9].

3. Pesticide Residues in Stored Foods

3.1 Distribution in Milled Products

Processing can redistribute pesticide residues between fractions. In wheat, pyrethroids such as cypermethrin and fenvalerate tend to concentrate in the bran, with lower levels in the endosperm; studies report that 79–88% of residues may remain in white flour, depending on the compound and milling conditions [22, 4]. Organophosphates like pirimiphos-methyl can have half-lives of several months in whole wheat, with detectable levels in flour and bread after storage and baking [4].

In rice, residues are higher in the husk and bran than in polished grain; polishing can reduce residues by 35–91%, but concentrates them in by-products often used for animal feed or oil extraction (24). Boiling or parboiling may further reduce some organophosphate residues, yet other compounds are more heat-stable and persist through cooking [25, 26].

3.2 Residue Levels Versus Maximum Residue Limits (MRLs)

Maximum residue limits are the highest legally allowed concentration of a pesticide residue in or on food or animal feed, expressed as mg/kg (ppm). Monitoring in African and Asian markets frequently reveals multiple pesticide residues in stored staples. In Tanzania, a survey reported that 40.7% of residues in vegetables and 38.9% in fruits exceeded Codex MRLs, highlighting risk from both field and storage use [6]. In Nigeria, Omoike et al. [2] documented widespread use of toxic agrochemicals for storage and reported organophosphate and carbamate residues in cowpea and grains at levels of concern, with chronic hazard indices for children exceeding 100% for some compounds. Naqvi et al. [9] investigated organochlorine residues in foods from Lagos and found that while most risk indices were below one for the measured compounds, the presence of multiple residues underscored the need to extend monitoring to organophosphates and pyrethroids that are

currently more heavily used. In China, multi-residue analyses in wheat and bran detected numerous pesticides, with concentrations up to 2.188 mg/kg, though mean chronic hazard quotients were below 100%, suggesting low risk for average consumers [4].

4. Human Exposure Pathways

4.1 Dietary Exposure

For the general population, diet is the main route of exposure to storage pesticides, primarily through consumption of treated grains, flours, and processed foods [4]. Residues in staples such as rice, wheat, maize, cowpea, and derived products like bread, pasta, and noodles contribute to cumulative daily intake, particularly in cereal-based diets typical of low-income populations [27, 28]. Washing, peeling, milling, and cooking can reduce but often do not eliminate residues, and certain compounds (e.g., some organophosphates, pyrethroids) are relatively heat-stable [26, 24].

4.2 Occupational Exposure

Fumigators, warehouse workers, millers, and farmers can be exposed to high levels of fumigants and insecticides during application, especially where PPE, training, and ventilation are inadequate [7, 29]. Inhalation of phosphine, methyl bromide, and DDVP vapors is the primary occupational route, complemented by dermal and oral exposure from handling treated grain or contaminated equipment [30, 16]. Many reported acute poisoning cases in Asia and Africa arise from misapplication, failure to seal fumigation sites, or presence of workers and residents during fumigation [11, 31].

4.3 Residential and Environmental Exposure

Domestic use of DDVP strips, household insect sprays, and contamination from nearby storage facilities can lead to residential exposure [16]. Environmental contamination occurs when pesticide containers are improperly disposed, wash-water from cleaning equipment enters surface waters, or legacy organochlorines persist in soils and sediments [9, 5]. Persistent compounds can bioaccumulate in aquatic and terrestrial food chains, extending exposure beyond directly treated commodities [32].

5. Acute Health Effects of Storage Pesticides

5.1 Organophosphates and carbamates

Acute organophosphate and carbamate poisoning results from acetylcholinesterase inhibition, leading to excessive cholinergic stimulation [8, 33]. Symptoms include miosis, salivation, bronchorrhea, bronchospasm, bradycardia, diarrhea, vomiting, muscle fasciculations, seizures, and, in severe cases, respiratory failure requiring intensive care [33]. Storage-related poisonings have been reported among grain handlers and fumigators using pirimiphos-methyl, chlorpyrifos-methyl, or DDVP without adequate PPE or in confined spaces [29]. DDVP, in particular, has been associated with genotoxic, neurological, immunological, hepatic, renal, and respiratory effects in animal and human studies, prompting regulatory restrictions [16, 17].

5.2 Phosphine

Phosphine is acutely toxic at very low concentrations. Early symptoms of inhalation include headache, dizziness, nausea, and chest tightness, progressing to dyspnea, cough, pulmonary edema, hypotension, arrhythmias, and central nervous system depression [1, 12]. A National Institute for Occupational Safety and Health (NIOSH) alert documented more than 200 cases of illness or injury linked to phosphine exposure during fumigation, including fatalities among workers and bystanders [30]. Mechanistically, phosphine disrupts mitochondrial oxidative phosphorylation, generates reactive oxygen species, and leads to metabolic collapse and multi-organ failure [11, 22].

5.3 Methyl bromide

Acute methyl bromide exposure causes irritation of the respiratory tract and eyes, followed by neurological symptoms such as ataxia, paresthesia, confusion, and seizures; severe poisoning may result in coma and death [14]. Case reports describe long-term neurological sequelae including cognitive deficits and peripheral neuropathy in survivors of acute grain-store fumigation incidents [14].

5.4 Pyrethroids

Pyrethroids, though less acutely toxic than many organophosphates, can cause paresthesia, skin and eye irritation, dizziness, and, at high doses, seizures and respiratory compromise [21]. Occupational poisonings have been reported among workers exposed to concentrated formulations or in poorly ventilated environments [5].

6. Chronic Health Effects of Storage-Pesticide Exposure

6.1 Cancer

Epidemiological studies have linked chronic pesticide exposure among applicators and agricultural workers to elevated risks of non-Hodgkin lymphoma, leukemia, and cancers of the prostate, lung, and other organs [20]. Organochlorines such as DDT and lindane are classified by the International Agency for Research on Cancer (IARC) as probable or possible human carcinogens; residues in stored food can contribute to cumulative lifetime exposure [9]. Organophosphates including malathion and diazinon are classified as probable carcinogens based on animal and limited human data, and DDVP is designated as a “probable human carcinogen” by the United States Environmental Protection Agency [18, 16].

6.2 Neurotoxicity and Neurodevelopmental Effects

Chronic low-level exposure to organophosphates has been associated with persistent neurobehavioral deficits in adults, including impaired memory, attention, and psychomotor function [27, 20]. In children, prenatal and early-life exposure to organophosphates has been linked to reduced IQ, attention-deficit/hyperactivity symptoms, and developmental delays [3]. MB fumigation work has been associated with chronic central nervous system effects, including cognitive impairment and motor dysfunction, in case series and occupational studies [14]. Pyrethroids, initially considered to be relatively safe, are increasingly implicated in potential developmental neurotoxicity, with cohort studies suggesting

associations between urinary pyrethroid metabolites and behavioral problems or cognitive deficits in children [35].

6.3 Endocrine Disruption and Reproductive Outcomes

Several pesticide classes (notably organochlorines and certain organophosphates) act as endocrine disruptors, interfering with estrogen, androgen, and thyroid signaling pathways [32]. Epidemiological studies have associated pesticide exposure with menstrual disorders, reduced fertility, adverse pregnancy outcomes, and altered semen quality in occupationally exposed men [36]. Organochlorine metabolites such as DDE have been linked to increased risk of obesity and insulin resistance, suggesting complex interactions between endocrine disruption and metabolic disease [32].

6.4 Metabolic and Immune Effects

Animal and human evidence indicates that chronic pesticide exposure can contribute to metabolic syndrome, insulin resistance, and type 2 diabetes, possibly via oxidative stress, mitochondrial dysfunction, and chronic low-grade inflammation [37, 38]. Organophosphates such as chlorpyrifos may alter hepatic adenylyl cyclase/cAMP signaling and promote a prediabetes-like phenotype in early-life exposure models [37]. Immune effects include altered cytokine profiles, increased susceptibility to infections, allergy and hypersensitivity, and potential modulation of autoimmunity [39].

7. Risk Assessment And Vulnerable Populations

Risk assessment typically compares estimated daily intake (EDI) of pesticide residues from food with acceptable daily intake (ADI) values. Hazard quotient (HQ) and chronic hazard index (CHI) metrics are used to gauge non-cancer risk [40, 4]. Many studies report that average exposures to individual pesticides are below ADI for the general population, yet these assessments often do not fully account for co-exposure to multiple residues with similar modes of action or for highly exposed subgroups [5, 9]. Children, pregnant women, the elderly, and informal-sector workers are particularly vulnerable. Children have higher food intake per body weight and immature detoxification systems, making them more susceptible to neurodevelopmental and endocrine effects [27, 34]. In one Nigerian assessment, chronic hazard indices for organophosphates in cowpea exceeded 100% for children but not adults, suggesting significant risk in this age group [2]. Women engaged in small-scale storage, processing, and marketing may experience combined dietary and occupational exposure with implications for maternal and fetal health [29, 36].

8. Mitigation Strategies and Alternatives

8.1 Effects of Processing

Processing techniques such as washing, peeling, milling, polishing, cooking, parboiling, and fermenting can reduce pesticide residues in many plant foods [26, 24]. For example, polishing rice can remove 40–90% of residues into bran, and parboiling reduces surface organophosphates such as malathion [25]. However, reductions are compound-specific, and some pesticide residues persist in edible fractions, including flour and bread [4]. Processing therefore mitigates but does not eliminate risk.

8.2 Integrated Pest Management (IPM) in Storage

Integrated pest management for stored products seeks to minimize reliance on synthetic pesticides by combining preventive and non-chemical measures with judicious pesticide use [3]. Key elements include improved storage structures (hermetic bags, metal silos) and optimized aeration; good hygiene and sanitation; physical controls such as temperature and humidity management or controlled atmospheres; and monitoring-based, threshold-triggered pesticide application. Successful implementation in smallholder settings can substantially reduce both losses and pesticide use [41].

8.3 Botanical and Biological Alternatives

Botanical insecticides (e.g., neem extracts, essential oils) and inert dusts such as diatomaceous earth are being developed as lower-toxicity alternatives for stored-grain pest control [42, 43]. They often have shorter persistence and lower mammalian toxicity, but challenges include variability in composition and efficacy, narrower pest spectrum, and the need for standardized formulations and regulatory approval [42].

8.4 Policy, Regulation, and Education

Strengthening pesticide regulation, implementing robust residue-monitoring programs, and enforcing bans or restrictions on highly hazardous and obsolete pesticides are critical steps [40, 23]. Regulatory actions such as phasing out methyl bromide for most uses and restricting DDVP illustrate how policy can reduce high-risk exposures [13, 16]. Farmer and worker training on correct dosing, safe handling, PPE use, and adherence to withholding periods can significantly lower occupational and dietary risks [29, 5].

9. Knowledge Gaps and Future Directions

Important knowledge gaps remain regarding the long-term impacts of chronic low-level exposures, especially mixture effects and gene–environment interactions in susceptible subgroups [5, 28]. There is a need for longitudinal cohort studies in children, pregnant women, and elderly populations in regions with heavy reliance on storage pesticides, as well as for improved exposure assessment integrating biomarkers, residue data, and modeling [9]. Research on the comparative effectiveness, cost-benefit, and adoption barriers of integrated pest management (IPM), hermetic storage, and botanical alternatives will be essential to guide policy and practice [42, 41].

10. CONCLUSION

Pesticides used for food storage are indispensable tools for controlling stored-product pests and reducing post-harvest losses, but they also create significant opportunities for human exposure through residues in food, occupational contact, and environmental contamination. Organophosphates, pyrethroids, phosphine fumigants, and legacy organochlorines are of particular concern given their acute toxicity and associations with chronic health outcomes, including cancer, neurodevelopmental and neurodegenerative disorders, endocrine and reproductive dysfunction, and metabolic and immune dysregulation. The risks are greatest in settings with

weak regulation, poorly controlled storage environments, and limited awareness of safe-use practices. Balancing food security and public health requires a multipronged strategy: stricter regulation and monitoring of storage pesticides, enhanced residue surveillance, promotion of integrated pest management and hermetic technologies, development and deployment of safer alternatives, and targeted education for farmers, storage workers, and consumers. With such measures, it is possible to maintain effective post-harvest pest control while substantially reducing the burden of pesticide-related diseases.

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