Vol.1, Issue 2, July-Sep 2025, Page: 17-23 ISSN: 3107-4863

Global Journal of Science and Technology (GJST)

A Review on the Impact of Chemicals Used in Crops on Human Health

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Abstract

Modern crop production relies on a wide array of chemicals—including pesticides, synthetic fertilizers, plant growth regulators, and post-harvest treatments—to secure yields and reduce losses. While these inputs contribute to food security, accumulating evidence links both occupational and dietary exposures to a spectrum of human health outcomes. Documented risks range from acute poisonings and irritant effects to chronic neurobehavioral changes, endocrine disruption, reproductive toxicity, carcinogenicity, cardiometabolic disease, and perturbed gut microbiomes. Risk varies with compound class, formulation, dose, timing, and life stage, and is modified by regulatory controls, residues at consumption, and mitigation practices across the farm-to-fork chain. This review synthesizes epidemiological, toxicological, and risk-assessment evidence; highlights vulnerable populations; summarizes regulatory approaches (ADI, ARfD, MRLs); and identifies practical risk-reduction strategies such as integrated pest management (IPM), safer formulations, improved application practices, and consumer-level decontamination. We conclude with research and policy priorities to align agricultural productivity with human health protection.

Keywords: pesticides; fertilizers; agrochemicals; residues; human exposure; endocrine disruption; neurotoxicity; carcinogenicity; nitrates; cadmium; integrated pest management; food safety; maximum residue limits.

1.Introduction

Feeding a growing global population under climate variability has intensified reliance on agrochemicals to protect crops against insects, weeds, and pathogens and to supplement soil nutrients (Pretty & Bharucha, 2015). Over 2 million tons of active pesticide ingredients are applied annually worldwide, with usage concentrated in high-value fruit and vegetable systems but rising in low- and middle-income countries (LMICs) where regulatory and stewardship capacity may lag (Damalas & Koutroubas, 2016; PAN International, 2023). Synthetic nitrogen fertilizers underpinned the Green Revolution, driving yield gains but also elevating nitrate and nitrite contamination risks in water and produce, with potential methemoglobinemia in infants and longer-term cancer concerns (Ward et al., 2018).

Agrochemical benefits are clear—reduced pre- and post-harvest losses, improved market quality, and stabilized farmer income (Oerke, 2006). Yet the public-health literature documents a wide range of adverse human outcomes. Farmworkers experience the highest acute exposures, with organophosphate (OP),

carbamate, and pyrethroid poisonings still prevalent despite phase-outs of some compounds (Eddleston et al., 2002; Boedeker et al., 2020). Chronic, low-dose exposures via diet and drinking water raise additional concerns about developmental neurotoxicity, endocrine disruption, reproductive toxicity, and carcinogenicity (Mostafalou & Abdollahi, 2013;; Mnif et al., 2011; Landrigan et al., 2018). Separately, contaminants in phosphate fertilizers (e.g., cadmium) and by-products of pesticide formulations (solvents, surfactants) may add toxic burdens (Satarug et al., 2019; Mesnage & Antoniou, 2018).

Risk depends on intrinsic hazard and on exposure—which is shaped by application practices, personal protective equipment (PPE), environmental fate and transport, processing/cooking, and regulatory measures such as acceptable daily intakes (ADIs), acute reference doses (ARfDs), and maximum residue limits (MRLs) (FAO/WHO JMPR, 2022; EFSA, 2020). This review integrates current evidence on human health impacts of crop chemicals, focusing on: (i) exposure pathways; (ii) major health endpoints by chemical class; (iii) vulnerable populations and equity considerations; (iv) dietary risks and regulatory science; and (v) mitigation strategies spanning farm to household.

2. Scope and search strategy

We synthesized peer-reviewed epidemiology, toxicology, and risk-assessment literature (systematic reviews, meta-analyses, key cohort and case–control studies) and authoritative evaluations (WHO, IARC, FAO/WHO JMPR, EFSA, US EPA) published mainly since 2000. Emphasis was placed on pesticide classes (OPs, carbamates, pyrethroids, neonicotinoids, triazole and strobilurin fungicides, dithiocarbamates, glyphosate and other herbicides), fertilizer-related nitrate/nitrite, and trace metals (cadmium) from phosphate fertilizers. We prioritized high-quality evidence (e.g., Agricultural Health Study; large birth cohorts) and landmark mechanistic reports.

3. Chemicals used in crops: classes and uses

3.1 Pesticides. Insecticides (OPs like chlorpyrifos; pyrethroids like cypermethrin; neonicotinoids like imidacloprid), herbicides (glyphosate, paraquat, 2,4-D), fungicides (triazoles, strobilurins, dithiocarbamates), rodenticides, and fumigants (phosphine) vary in mode of action and toxicological profiles (Tomlin, 2012; WHO, 2020).

3.2 Fertilizers. Nitrogen (urea, ammonium nitrate), phosphorus (DAP, SSP), and potassium fertilizers support yields but can elevate nitrate in leafy vegetables and contaminate groundwater (Camargo & Alonso, 2006; Ward et al., 2018). Phosphate fertilizers may introduce cadmium to soils (Satarug et al., 2019).

3.3 Plant growth regulators and adjuvants. Ethephon, gibberellins, auxins, surfactants, and solvents can modify efficacy and residues (Mesnage & Antoniou, 2018).

3.4 Post-harvest treatments. Fungicides, waxes, and ripening agents (legal: ethylene; illegal in some contexts: calcium carbide) affect consumer exposures (FAO/WHO, 2019).

4. Human exposure pathways across the farm-to-fork chain

Occupational exposure arises during mixing/loading, application, equipment cleaning, and re-entry (Arcury & Quandt, 2003; Kamel & Hoppin, 2004). **Para-occupational exposure** affects family members via take-home contamination (Curwin et al., 2003). **Environmental drift and volatilization** can expose nearby communities (Lee et al., 2011). **Dietary exposure** stems from residues on/in foods and from

contaminated water (EFSA, 2020; US FDA TDS, 2019). **Early-life exposure** via placenta and breast milk is critical for neurodevelopmental toxicity (Rauh et al., 2011; Bouchard et al., 2010).

5. Acute health effects

Acute pesticide poisoning remains a major global burden, particularly in LMICs (Boedeker et al., 2020). OPs cause cholinergic syndrome (miosis, bronchorrhea, seizures) via acetylcholinesterase inhibition; carbamates act similarly but reversibly (Eddleston et al., 2002). Pyrethroids provoke paresthesia and, at higher doses, neuroexcitation (Ray & Fry, 2006). Paraquat ingestion leads to pulmonary fibrosis and high case fatality (Gawarammana & Buckley, 2011). Fumigants (phosphine) can cause cardiotoxicity and metabolic acidosis (Sidhu et al., 2015).

6. Chronic health effects

6.1 Neurodevelopment and cognition

Prospective birth cohort studies associate prenatal OP exposure (metabolites like DAPs; chlorpyrifos cord blood) with decreased IQ, working memory deficits, and increased ADHD behaviors in children (Bouchard et al., 2010; Rauh et al., 2011). Occupational OP exposure has been linked to chronic neurobehavioral impairment (Kamel & Hoppin, 2004).

6.2 Neurodegenerative disease

Epidemiologic studies report elevated Parkinson's disease risk with paraquat and rotenone exposure among applicators and rural residents (Tanner et al., 2011; Gao et al., 2012; Wang et al., 2011). Mechanistic data support mitochondrial complex I inhibition and oxidative stress (Betarbet et al., 2000).

6.3 Endocrine disruption and reproduction

Multiple pesticide classes (DDT/DDE legacy, certain azoles, OPs, pyrethroids) show endocrine-active properties affecting thyroid, androgen, and estrogen signaling, with associations to reduced semen quality, altered menstrual cycles, adverse birth outcomes, and earlier puberty (Mnif et al., 2011; Harley et al., 2016; Meeker et al., 2006; Windham et al., 2015).

6.4 Cancer

The Agricultural Health Study and meta-analyses report associations between specific pesticides and non-Hodgkin lymphoma, leukemia, multiple myeloma, and prostate cancer (Alavanja et al., 2003; Schinasi & Leon, 2014; Koutros et al., 2020). IARC classified glyphosate as "probably carcinogenic to humans" (Group 2A) in 2015 based on limited human evidence and sufficient animal evidence (IARC, 2015), whereas updated large cohort analyses did not observe overall increases in NHL in applicators (Andreotti et al., 2018). Divergent hazard vs risk interpretations highlight ongoing controversy (Mie et al., 2017).

6.5 Cardiometabolic outcomes

Epidemiologic evidence links certain pesticide exposures to type 2 diabetes and dyslipidemia, possibly through mitochondrial toxicity and endocrine mechanisms (Mostafalou & Abdollahi, 2013; Evangelou et al., 2016).

6.6 Respiratory and allergic disease

Fumigants and dusts can exacerbate asthma and cause reactive airways dysfunction syndrome among workers; organophosphate and pyrethroid exposure has been associated with wheeze and decreased lung function in children (Hoppin et al., 2006; Salam et al., 2004).

6.7 Microbiome and antimicrobial resistance

Emerging studies suggest herbicides and fungicides can alter gut microbial communities and may potentiate antibiotic resistance selection in environmental reservoirs (Mesnage et al., 2019; Kurenbach et al., 2015).

6.8 Nitrates/nitrites and water quality

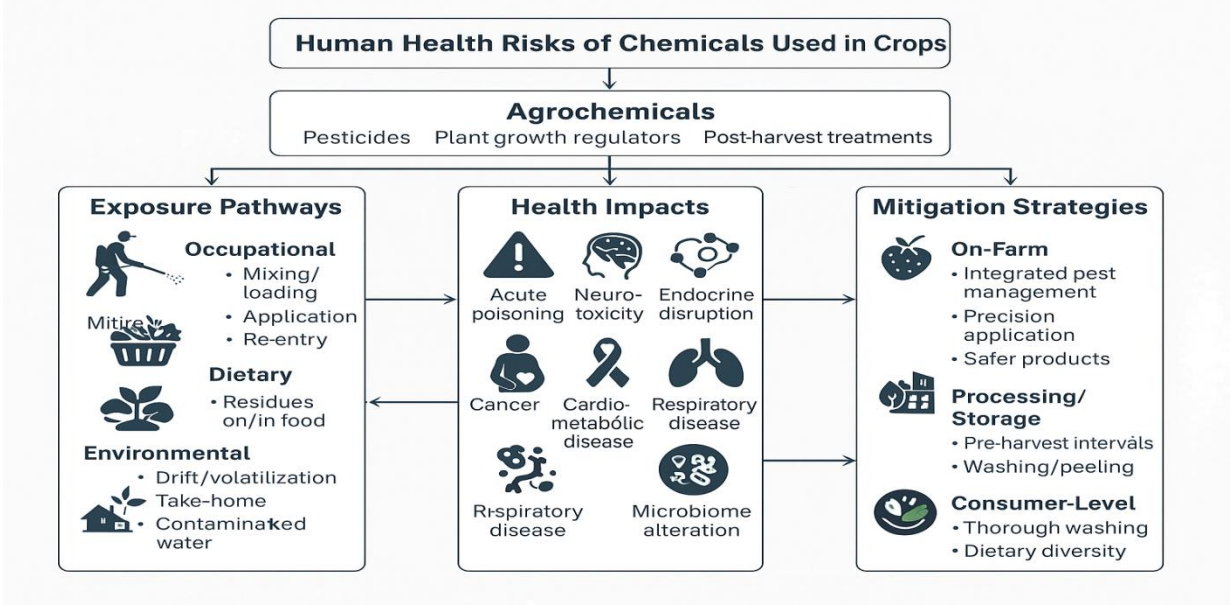
High nitrate in drinking water increases risk for infant methemoglobinemia ("blue baby syndrome") and is under investigation for associations with colorectal and other cancers (Greer et al., 2005; Temkin et al., 2019; Ward et al., 2018).

6.9 Metals from fertilizers (cadmium)

Cadmium accumulated from phosphate fertilizers can increase dietary cadmium, contributing to renal tubular dysfunction, bone demineralization, and cardiovascular risk (Satarug et al., 2019; EFSA, 2009).

7. Dietary residues, risk assessment, and regulation

Risk assessment integrates hazard identification, dose–response, exposure assessment, and risk characterization (EFSA, 2020; FAO/WHO JMPR, 2022). ADIs and ARfDs are derived from animal and human data with uncertainty factors; MRLs are set to ensure residues from approved uses remain below levels compatible with these health-based guidance values (Codex, EFSA). Total diet studies and probabilistic modeling evaluate population exposure distributions (US FDA TDS, 2019; EFSA, 2020). Notably, mixtures ("cocktails") and vulnerable subgroups (infants, pregnant people) challenge single-compound frameworks (Kortenkamp, 2007; Trasande et al., 2018).



8. Vulnerable populations and equity

Farmworkers and applicators face highest exposures; migrant and informal workers may lack training/PPE (Arcury & Quandt, 2003). **Children and fetuses** are more susceptible due to developmental windows and higher intake per body mass (Rauh et al., 2011). **Communities near fields** can be exposed

via drift and contaminated dust (Lee et al., 2011). **LMICs** bear disproportionate acute poisoning burdens and may have limited surveillance and enforcement (Boedeker et al., 2020; WHO, 2020).

9. Mitigation strategies across the supply chain

9.1 On-farm: IPM to reduce chemical reliance; resistant varieties and biological control; precision application; buffer zones; substitution of highly hazardous pesticides (HHPs); closed transfer systems; mandated re-entry intervals; and universal access to appropriate PPE (Pretty & Bharucha, 2015; FAO/WHO, 2019; PAN International, 2023).

9.2 Post-harvest and processing: Adherence to pre-harvest intervals; cold chain; approved fungicides; monitoring programs; and residue decline through washing, peeling, and cooking (Keikotlhaile et al., 2010).

9.3 Consumer-level: Thorough washing with running water, peeling where appropriate, trimming outer leaves, and diversified diets to minimize repeated exposure to a single residue profile (EFSA, 2020).

9.4 Policy levers: Phase-out of HHPs; stronger surveillance of poisonings; harmonization of MRLs; addressing mixture risks; and targeted education for smallholders (Landrigan et al., 2018; WHO, 2020).

10. Research gaps and priorities

1. Longitudinal cohort data in LMICs; 2) Mixture and low-dose non-monotonic effects; 3) Microbiome and metabolomics integration; 4) Real-world exposure biomarkers (including emerging fungicides and adjuvants); 5) Safer formulations and delivery technologies; 6) Climate-change interactions (e.g., pest range shifts increasing use intensity).

11. Conclusion

Agrochemicals remain indispensable tools for stabilizing crop yields and ensuring food security, but their benefits are inseparable from measurable human health risks that span acute poisonings to chronic, developmental, and endocrine outcomes. The weight of evidence shows that risk is neither static nor uniform—it is mediated by compound-specific hazards, timing and route of exposure, vulnerable life stages, and social determinants such as occupational protections and regulatory enforcement. The science also underscores a central tension: hazard classifications (e.g., IARC) and cohort-based risk estimates (e.g., Agricultural Health Study) can reach different conclusions when dose, timing, and co-exposures diverge. Productive policy must therefore integrate both hazard and exposure science, while recognizing uncertainty and mixtures.

A pragmatic path forward couples **risk prevention at source**—phasing out highly hazardous pesticides, prioritizing IPM/biological controls, and reformulating to minimize co-formulant toxicity—with **exposure minimization** through better application technology, PPE, buffer zones, re-entry intervals, and rigorous pre-harvest intervals. Regulatory programs should strengthen surveillance of poisonings, routinely reassess ADIs/ARfDs with contemporary mechanistic data, and explicitly account for mixture effects and early-life susceptibility. Food-safety systems should sustain residue monitoring and communicate practical, evidence-based guidance to consumers on washing, peeling, and dietary diversification.

Ultimately, aligning agricultural productivity with human health requires re-balancing incentives toward safer pest and nutrient management, building capacity for stewardship in LMICs, and closing knowledge

gaps on mixtures, microbiome, and long-term cardiometabolic outcomes. Such an agenda protects those who feed the world and those who are fed by it—delivering not only more food, but safer food.

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