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Review Article

Public Health Implications of Pesticide Use: Evaluating Acute, Long-Term, and Genetic Toxicity

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ABSTRACT

Pesticides remain indispensable in modern agriculture for safeguarding crops and ensuring global food security. However, their widespread and often indiscriminate use has raised significant concerns due to acute, chronic, and genetic toxicities in humans. This review synthesizes current evidence on the health implications of pesticide exposure, highlighting both immediate and long-term risks. Acute effects include dermatological irritation, respiratory distress, gastrointestinal disorders, and ocular toxicity, while chronic exposure has been associated with endocrine disruption, reproductive abnormalities, neurodegenerative diseases such as Parkinson's, cardiotoxicity, and carcinogenesis. Particular emphasis is given to genotoxic outcomes, with cytogenetic biomarkers—such as chromosomal aberrations, micronucleus frequency, and sister chromatid exchanges—serving as critical tools in assessing DNA damage among exposed populations. Genetic polymorphisms influencing metabolic detoxification further underscore individual susceptibility to pesticide-related harm. Additionally, the review discusses regulatory frameworks, maximum residue limits, and preventive strategies designed to minimize risks. Ultimately, this article emphasizes the urgent need for stricter regulatory enforcement, safer alternatives, and multidisciplinary collaboration among pharmacists, toxicologists, healthcare providers, and policymakers to mitigate pesticide-related health hazards and protect public health.

INTRODUCTION

Pesticides are widely used in agriculture to control pests and increase crop yields. Pesticides are made of a wide variety of substances, and their use is expanding through the world. Over 1378 active chemicals are listed in the EU Pesticides Database; 466 of them have received approval for usage in the EU, while 858 have not.[1] In the 1950s, organochlorine insecticides were widely utilised in agriculture.[2] It is currently prohibited in the majority of nations and categorised as a persistent

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organic pollutant (POP).[3] Consequently, organochlorine pesticides have been gradually replaced in recent years by safer, more efficient substitutes with quicker rates of biodegradation, such as neonicotinoids and organophosphorus insecticides.[4,5] Pesticides are also employed in a variety of fields, including forestry, aquaculture, food production, agriculture, wood processing, transportation, and storage, as well as other biological production.[6]

Historical Development and Agricultural Importance

Human civilisations have been attempting to cultivate and preserve their food resources using the most efficient and time-efficient methods from the beginning of time.Several millennia ago, they used elemental sulphur to get rid of pests. Additionally, primitive sulphides are used in traditional Chinese medicine. Early usage of the "para-pesticides," especially arsenic and mercury, began to appear in the 1500s. Originally employed to destroy food supplies during World War II, these poisons continued to be utilised until the advent of synthetic pesticides in 1940 and later. The first modern pesticide was discovered in 1939 by Paul Muller and is known as dichlorodiphenyl-trichloroethane (DDT). Years later, this discovery earned him the Nobel

Prize in Medicine.[7] The public debate surrounding Rachel Louise Carson's (1907–1964) writings strengthened the prevailing narrative about DDT and the US; her book Silent Spring sparked the alarm in the 1960s and eventually resulted in the US Environmental Protection Agency banning DDT.[8] In the years that followed, many jurisdictions outlawed the use of DDT in favour of less hazardous carbamates and organophosphates. In order to reduce the number of major illnesses in the population, producers must reduce the amount of pesticides they create, according to new EU legislation. The introduction of pesticide-resistant herbs, on the other hand, is currently receiving a lot of attention and will undoubtedly have a significant influence in the future. [7]

Classification of Pesticides

Pesticides are categorised according to a number of different criteria, including the following

1. Classification of pesticides based on Toxicity.
2. Classification of pesticides based on pest organism they kill and pesticide function.
3. Classification of pesticides based on its chemical composition.
4. Classification of pesticides according to the way in which they are administered.
5. Classification of pesticides based on how and when they work.
6. Classification of pesticides based on type of formulations. [6]

WHO Recommended Classification of Pesticides

The Global Health Service ((Globally Harmonised System) Acute Toxicity Hazard Categories are now used by WHO as the basis for classification. This modification is in line with the World Health Assembly Resolution from 1975, which called for countries, international organisations, and regional entities to be consulted as the WHO Classification was evolved throughout time. As a categorisation system that has gained worldwide recognition after much international deliberation, the GHS satisfies this condition. [9]

Table 1: WHO recommended classification of Pesticides. [9]

Class	Hazardous level	LD_{50} for Rats (Mg/kg body wt.)		Pesticides examples
		Oral	Dermal	
Ia	Extremely hazardous	< 5	< 50	Parathion, Endosulfan
Ib	Highly hazardous	5-50	50-200	Aldrin, Dichlorvas
II	Moderately hazardous	50-2000	200-2000	DDT, Chlordane
III	Slightly hazardous	Over 2000	Over 2000	Malathion
U	Unlikely to present acute hazard	5000 or higher		Carbetamide, Cycloprothrin

Acute toxicity of pesticides:

In times of food scarcity, agricultural products must be stored in order to be preserved for later use. In order to prevent pests and preserve agricultural products, pesticides are used during food storage, which exposes workers to them at work. The general public is exposed to pesticides through the presence of pesticide residues in agricultural products that are kept. Dichlorvos, zinc phosphide, aluminium phosphide, benomyl, permethrin, pirimiphos-methyl, gamalin, chlorpyriphos, DDT, and carbofuran are a few examples of pesticides that preserve agricultural products.[10,11] It's possible that some of these chemicals, which have been found to have lethal levels, could be added to agricultural goods directly or indirectly. If taken carelessly, they could be extremely dangerous to humans.[12,13] Following are some consequences of adding chemicals and pesticides to agricultural products during storage.

Acute effect on skin

Toxic chemicals can be absorbed by the skin as a result of spills and splashes during handling. Contact dermatitis, including irritant contact dermatitis (ICD) or allergic contact dermatitis (ACD), allergic urticaria, and asthma, are caused by dermal exposure to storage chemicals. Local inflammatory symptoms like redness, itching, swelling, discomfort, and rash are caused by acute contact dermatitis. Dermatological exposure to the pyrethroid insecticide deltamethrin is one way this shows up. An agricultural worker was found to experience diarrhoea and paraesthesia of the mouth, tongue, and legs after being exposed to approximately 5 g/L of deltamethrin.[14] Tartrazine and other food additives have also been connected to dermatitis, asthma, and urticaria.[15] Urticaria is also linked to antioxidants such sorbates, sulphites, butylated hydroxyanisole (BHA), and butylated hydroxytoluene (BHT). Sulphites can also cause flushing and rashes on the skin. [16]

Acute effect on gastrointestinal tract

The gastrointestinal tract (GIT) and gut microbes, often known as the gut microbiota, are the initial biological and physical barriers that prevent ingested food from being exposed to chemicals used in food preservation.[17] Toxins, antibiotics, and nutrition may all have an impact on the composition of the gut microbiota.[18] As evidenced by the storage pesticide DDT, nausea and vomiting are frequent signs of the acute effects of pesticides on the gastrointestinal tract.[19] Within 10 minutes to 1 hour after administration, pyrethroid poisoning causes epigastric discomfort, headache, nausea, vomiting, exhaustion, and increased stromal secretion.[14]

Acute effect on Respiratory tract

Pesticide inhalation is the main exposure linked to work. Inhaling pesticides can produce acute respiratory symptoms such as dryness, wheezing, airway irritation, dry throat, nose discharge, and dyspnoea.[20] Organophophorus causes alveolar oedema, bronchoconstriction, and bronchorrhea, which result in respiratory failure. [21] Due to their direct ability to modify the lining of the bronchi through irritation, inflammation, and immunosuppression, these pesticides are linked to asthma. [22] By influencing oxidative phosphorylation, pesticides derived from nitrophenols also have an impact on respiration. [23]

Acute effect on Eye

Numerous reports indicate that the use of organophosphate pesticides in agriculture has increased the occurrence of Saku disease, a visual disease syndrome, in human populations. Animal follow-up experiments conducted in Japan with agents including fenthion, ethylthiometon, and fenitrothion verified the harmful effects of organophosphates on the visual system. The effects of organophosphates on the visual system, such as myopia, astigmatism, visual field narrowing, decreased vision, and histopathological evidence of degeneration of extraocular muscle, ciliary muscle, retina, and other ocular tissues, are conclusively demonstrated by animal studies conducted by Japanese authors and some studies currently being submitted by registrants to The Office of Pesticide Programs (OPP).[24]

Chronic Toxicity of Pesticides

Even when these substances are exposed in trace amounts, they can have some long-term impacts in addition to the acute ones that occur after exposure. Despite being utilised for preservation, some of these chemicals have been shown to remain in ready-to-eat foods like fruit juice, prepared foods, water, and breast milk. These compounds have the potential to have either acute or chronic toxicological consequences when used for storing agricultural products. Chronic toxicity is typically caused by occupational poisoning or long-term ingestion, whereas acute poisoning might be accidental or the result of exposure over a brief period of time. Individuals' current health status, chemical type (particularly chemical structure), quantity, age, sex, time period, and exposure route are some of the elements that determine how harmful they are. The following are some chronic effects of introducing pesticides and chemical to agricultural products during storage.

Reproductive Effects

It has been demonstrated that both vertebrates and invertebrates' reproductive systems are negatively impacted by pesticide and organic chemical exposure. Exposure to certain pesticides used for food storage may cause congenital abnormalities and reproductive issues. According to a recent study, the combined effects of several exposures to different pesticides, including metalaxyl, mancozeb, and cypermethrin, can result in dosages that are more than 50% below their LD50 for eight weeks. Testicular and epididymal weight, as well as testosterone and cholesterol levels, were found to have drastically decreased, according to the researchers. Furthermore, there was a significant decrease in sperm concentration, motility, viability, and antioxidant content in comparison to the control. Another study revealed that acetamiprid had negative effects on reproduction, such as a dose-dependent decrease in sperm concentration and plasma testosterone levels. even at a modest exposure dose of 12.5 mg/kg for 90 days.

Endocrine Disruption

P-dichlorodiphenyldichloroethylene (DDE), a metabolite of dichlorodiphenyltrichloroethane (DDT), has been linked to endocrine disruption, raising some health concerns.[25] A study by Rini Ghosh and his associate assessed Lambda-cyhalothrin's endocrine-disrupting effects in female rats. Adrenal cholesterol significantly decreased with a corresponding decrease in ovarian 3β- and 17β-hydroxysteroid dehydrogenase activity in experimental animals given a dosage of 6.3–11.33 mg/kg BW daily for 14 days, while ovarian cholesterol increased with a corresponding increase in adrenal 3β- and 17β-HSD activity.[26] Pentachloronitro-benzene, pyrimethanil, amitrole, and other substances can block the synthesis of thyroid hormones. Aldicarb and carbofuran are carbamates that can interfere with endocrine function.[27]

Neurotoxicity

Long-term DDT exposure has raised concerns about a number of possible negative health impacts. Regretfully, very little research has been done on humans to examine DDT's possible neurotoxicity. Employees who sprayed DDT showed signs of cognitive impairment in two investigations. DDT was detected more frequently in the brains of Alzheimer's disease sufferers, according to tiny research. Serum DDE levels were linked to lower cognitive function in older US adults, according to a recent study that used data from the NHANES population. This suggests that non-occupational exposures to DDT can also result in cognitive abnormalities. There is growing evidence of possible neurotoxicity for a number of pesticides that were introduced more recently than those discussed here, such as neonicitinoids, pyrazoles, biopesticides, etc.[28]

Cardio toxicity

Studies on the cardiotoxicity of pesticide residues in honey, milk, and water are uncommon. The cardiotoxicity among workers exposed at work was clarified by certain epidemiological surveys. For example, human cardiotoxicity was caused by DDT and its analogues, which were most commonly found in honey samples. Patients with atherosclerosis had higher serum concentrations of DDT, DDE, DDD, lindane, dieldrin, heptachlorepoxide, and polychlorinated biphenyls than control groups, and occupational exposure to OC (Organochlorine) insecticides was linked to the development of both arterial hypertension and arteriosclerosis. Humans who were unintentionally poisoned by chlordane experienced tachycardia, but occupationally exposed people did not have cardiovascular effects. However, workers at an industrial company that manufactures chlordane showed signs of elevated risk. While occupational exposure to aldrin, DDT, and 2, 4, 5-T resulted in nonfatal myocardial infarction, occupational exposure to p,p′-DDE, Trans-Nonachlor, Oxychlordane, Dieldrin, and γ-HCH (lindane) produced severe peripheral artery disease, which ultimately led to worker mortality in the United States. [29]

Parkinsonism

PD (Parkinson's disease) is primarily caused by external pollutants. Air pollution, industrial solvents like trichloroethylene (TCE), and certain insecticides are the most common of these.

More toxicants may be linked to Parkinson's disease; these are not the only ones. Nonetheless, both epidemiological and preclinical research have connected them to Parkinson's disease (PD); many of them harm mitochondria, which are known to be compromised in PD; and humans are exposed to them through a variety of ways, including ingestion and inhalation, as well as occupational and environmental sources as well. There is evidence from numerous animal and epidemiological studies that some pesticides cause Parkinson's disease. The sub

acute development of end-stage parkinsonism in seven young adults following intravenous injection of the designer drug 1methyl-4-phenyl-l,2,3,6-tetrahydropyridine (MPTP) in the 1980s provided a crucial hint. MPTP is metabolised to its metabolite 1-methyl-4-phenylpyridinium (MPP+), which inhibits mitochondrial complex 1 and kills dopaminergic neurones, causing damage to the nigrostriatal dopaminergic neurones. The structure of MPP+ is noticeably similar to that of paraquat, one of the most popular weedkillers in the world that likewise disrupts mitochondrial activity. [30]

Genotoxicity

One of the main risk factors for long-term consequences including carcinogenic and reproductive toxicology is genotoxic potential. Most pesticides have undergone testing in a broad range of mutagenicity tests that include DNA damage, chromosomal disruption, and gene mutation.[31] Although there are several biomarkers to evaluate both temporary and permanent genotoxic reactions, biomonitoring research on human populations exposed to pesticides has mostly concentrated on cytogenetic end-points, such as sister-chromatid exchanges (SCE), micronuclei (MN) frequency, and chromosomal aberrations (CA).

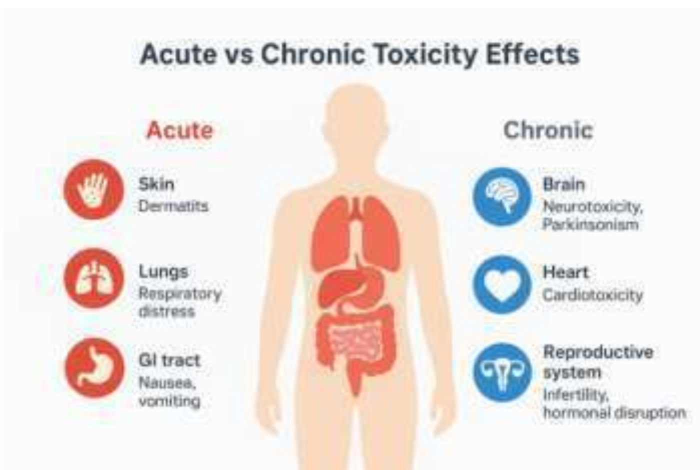
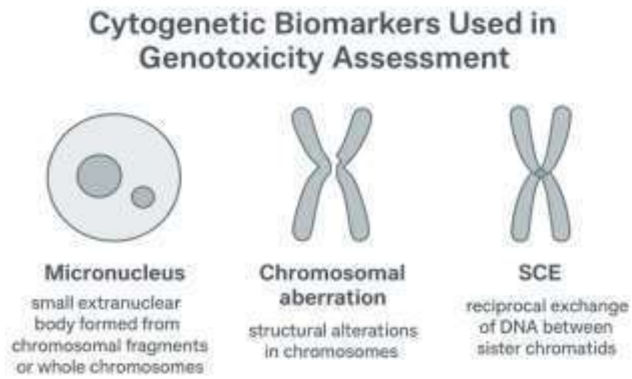


Figure 2 : Acute vs chronic toxicity effects

Cytogenetic Biomonitoring Studies

Chromosome-level genetic damage involves changes in the number or structure of chromosomes, and these changes can be quantified as CA or MN frequency. According to recent research, the nucleotide pool imbalance is crucial for the development of SCE and can have serious effects on DNA metabolism. The possibility that DNA alterations are the cause of SCE and mutations in mammalian cells is increased by the control of SCE by DNA precursors. [32,33] Although elevated CA levels have been linked to an increased risk of cancer. [34,35] Neither SCE nor MN have been found to have the same association. However, those at higher risk for cancer have been found to have high levels of SCE and MN frequency as a result of environmental or occupational exposure to a diverse range of carcinogens. [36]



Cytogenetic damage due to effect of genotypes

As biomarkers of vulnerability to mutations, cancer, and other disorders, genotypes that cause interindividual variations in the capacity to activate or detoxify genotoxic chemicals are known to exist. [37] The study examined how certain cytochrome P450 (CYP) gene polymorphisms, specifically cytochrome P450 2E1 (CYP2E1), glutathione S-transferase M1 (GSTM1), glutathione S-transferase theta 1 (GSTT1), N-acetyltransferase 2 (NAT2), and paraoxonase 1 (PON1), modulate cytogenetic effects in populations exposed to pesticides. These polymorphic genes were chosen because of their function in pesticide metabolism. [38] The metabolism of pesticides based on organophosphates is carried out by paraoxonases (PONs). The activity of serum paraoxonase (PON1) is crucial for organophosphate metabolism. Compared to those with higher PON1 activity individual, those with lower PON1 activity are more vulnerable to parathion poisoning. [39]

Regulatory Aspect of Pesticides

Maximum residue limits (MRLs): MRLs for pesticide and other agrochemical residues on food products are set by governments worldwide. The highest concentration of residues that can be present on or in food products is specified by these limitations. To guarantee food safety, MRLs must be regularly monitored and enforced.

Registration and licensing of pesticides: Before being sold or utilised, agrochemicals mustpass a strict registration and approval process. By considering possible hazards to the environment, human health, and non-target creatures, this procedure assesses the safety and effectiveness of these substances.

Labelling and safety data sheets: Agrochemical products are accompanied by comprehensive labelling and safety data sheets that include instructions on how to handle, apply, store, and dispose of them properly. In order to reduce hazards while in use, these publications also provide safety measures.

Certification and training: People who handle and apply agrochemicals must complete certification and training programs in several nations. The appropriate use of these substances, safe handling procedures, and protective equipment usage are all covered in this session.

Restricted application and buffer zones: To lower the chance of drift and contamination of neighbouring areas, regulations may require buffer zones surrounding treated fields. In order to reduce their negative effects on the environment, some chemicals may also be restricted in their use during particular periods or in particular weather conditions. [40]

CONCLUSION

Pesticides, while indispensable to modern agriculture for their role in enhancing crop yield and protecting food resources, pose substantial risks to human health due to their toxicological properties. This review highlights the extensive evidence of both acute and chronic toxic effects—including dermatological, gastrointestinal, respiratory, neurological, reproductive, endocrine, and cardiovascular impacts. Long-term exposure to even low levels of pesticide residues has been linked to serious conditions such as Parkinson's disease, endocrine disruption, genotoxicity, and various forms of cancer.Cytogenetic biomarkers such as chromosomal aberrations, micronuclei frequency, and sister chromatid exchanges provide valuable tools for monitoring genetic damage in exposed populations, especially those occupationally or environmentally at risk. Furthermore, genetic polymorphisms influencing metabolic detoxification pathways underline the importance of individual susceptibility in assessing health outcomes. From a pharmaceutical and public health perspective, there is a pressing need for increased toxicovigilance, routine biomonitoring, and community awareness to minimize health risks. In conclusion, a multidisciplinary and proactive approach—incorporating science, policy, healthcare, and sustainable agriculture—is essential to mitigate the adverse effects of pesticide exposure and protect human health for future generations.

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