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Pesticide Resistance in Nigeria: Emerging Threats to Public Health, Food Security, and Ecosystem Services

Nsikak J. Akpan[1], Isaac T. Akinwumi[2] and Olusegun O. Awotoye[3]

[1]Department of Environmental Health Science, Federal University of Health Sciences, Ila-Oragun, Nigeria. [2]Department of Public Health Science, Atiba University, Oyo, Nigeria. [3]Institute of Ecology and Environmental Studies, Obafemi Awolowo University, Ile-Ife, Nigeria.

Abstract

Pesticide use in Nigeria has increased steadily in response to agricultural intensification, climate-driven pest pressure, and vector-borne disease control. However, inconsistent regulation, misuse, and heavy reliance on chemical pesticides have accelerated the emergence of resistance across agricultural pests, disease vectors, and weeds. This review synthesizes current evidence on pesticide use patterns, resistance mechanisms, environmental contamination, public-health risks, and implications for food security in Nigeria. A structured literature review was conducted using major academic databases and government reports published between 2000 and 2025. Findings show widespread detection of pesticide residues in cereals, legumes, water bodies, and fish, with several exceeding international maximum residue limits. Resistance in Anopheles, Aedes, Culex, Helicoverpa, Spodoptera, and stored-product beetles is now documented across multiple ecological zones, driven by metabolic detoxification, target-site mutations, behavioural avoidance, and cuticular barriers. Environmental impacts include biodiversity loss, soil degradation, aquatic toxicity, and bioaccumulation. Public-health consequences range from acute poisoning to chronic disorders affecting neurological, reproductive, endocrine, and metabolic systems. The review highlights regulatory gaps, economic losses from export bans, and the urgent need for integrated pest management, biopesticides, farmer training, residue monitoring, and national resistance-management programs. Coordinated, multi-sectoral interventions are essential to safeguard food systems, ecosystems, and public health.

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Keywords: pesticide resistance, residues, Nigeria, food security, ecosystem services, public health, Anopheles, IPM.

1. Introduction

Pesticides are widely used in agriculture and public health to suppress organisms that threaten crop production, food storage, and human well-being. According to the Food and Agriculture Organization (FAO), pesticides include chemical or biological agents applied to prevent, repel, or control pests — including disease vectors and unwanted plants — as well as substances used as growth regulators or post-harvest protectants (Daraban *et al*., 2023; Hlihor *et al*., 2018). Over the past seven decades, global demand for pesticides has expanded steadily, driven by the intensification of commercial agriculture and the need to reduce transmission of vector-borne diseases. Their contribution to increased crop yields, preservation of commodity quality, and reductions in malaria, dengue, and other vector-borne illnesses is well documented (Tudi *et al*., 2021; Ahmad *et al*., 2024).

However, the rapid and often indiscriminate expansion of pesticide use has raised significant environmental and public-health concerns. Residues are now frequently detected in soil, water, air, food crops, and processed commodities (Cech *et al*., 2023). Acute and chronic exposures have been linked to carcinogenic, neurotoxic, mutagenic, endocrine, and reproductive effects (Fang *et al*., 2020; Ahmad *et al*., 2024). Globally, approximately three million cases of pesticide poisoning and over 200,000 related deaths occur annually, with low- and middle-income countries bearing a disproportionate burden (Boedeker *et al*., 2020).

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*Corresponding author *Email address:* ecologistakpan@gmail.com (Nsikak J. Akpan)

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Pesticides vary widely in chemical composition and toxicological behaviour. Major classes include organochlorines, organophosphates, carbamates, pyrethroids, triazines, phenoxyacetic acids, benzoic acids, and dithiocarbamates (Bortoli *et al*., 2018). Many of these compounds are persistent, bioaccumulative, and capable of dispersing far from application sites through volatilization, leaching, and runoff. It is estimated that less than 1% of applied pesticides reach their intended targets, with the remainder infiltrating surrounding ecosystems (Daraban *et al*., 2023). This inefficiency contributes to widespread non-target toxicity and ecosystem disruptions, including reductions in soil microbial activity, pollinator decline, diminished insect biomass, loss of farmland birds, and contamination of aquatic systems (Ali *et al*., 2021).

Despite increasing global restrictions, highly hazardous pesticides — such as dichlorvos, methomyl, carbofuran, paraquat, aldicarb, and DDT — remain in circulation in many developing countries due to affordability and availability (Kehinde & Tijani, 2021). Their continued use heightens environmental burdens and intensifies human exposure. Compounding this challenge is the accelerating emergence of pesticide resistance across multiple pest species, driven by sustained dependence on chemical control. Resistance develops when individuals with naturally occurring tolerance survive repeated applications and pass on resistant genes, resulting in populations that are no longer controlled by previously effective compounds (Satyanarayana *et al*., 2024; Raj *et al*., 2023).

Pesticide resistance now represents a major threat to agricultural productivity, public health, and ecosystem stability. In agriculture, resistant weeds, insects, and pathogens undermine crop-protection efforts, elevate production costs, and contribute to yield losses that jeopardize food security. In public health, resistance among mosquitoes, ticks, and other vectors compromises control of malaria, dengue, and other vector-borne diseases, reversing gains achieved through chemical interventions. The phenomenon spans sectors — crop production, veterinary medicine, and disease-vector management — highlighting its complexity and far-reaching implications (Khokhar *et al*., 2024; Tiwari, 2023).

The environmental consequences of rising pesticide resistance are equally concerning. As pesticide efficacy declines, farmers frequently respond by increasing application rates or switching to more toxic alternatives, intensifying contamination and long-term ecological damage. Toxicity profiles differ across chemical classes, but many compounds exert lethal and sublethal effects on beneficial insects, soil organisms, birds, mammals, and aquatic life. Persistent pesticides such as organochlorines may remain in the environment for decades, while organophosphates and carbamates, though less persistent, exhibit potent neurotoxicity. Sublethal effects — including impaired reproduction, behavioural changes, immune suppression, and altered development — can destabilize population dynamics and compromise ecosystem functioning (Nehul, 2025).

In Nigeria, pesticide dependence has risen in response to agricultural intensification, climate-driven pest pressures, and limitations in integrated pest management (IPM). Weak regulatory enforcement, limited farmer awareness, and unrestricted access to banned or counterfeit products further exacerbate misuse. Consequently, reports of resistance across agricultural pests and disease vectors are increasing, posing urgent challenges for food security, public health, and ecosystem resilience. Understanding the drivers, mechanisms, and consequences of pesticide resistance is essential for guiding sustainable pest-management strategies.

Therefore, this study aims to critically examine the current status of pesticide resistance in Nigeria and evaluate its environmental, food-safety, and public-health implications.

2. Methodology

This study adopted a systematic narrative-review design supported by thematic analysis to examine pesticide resistance and its environmental, food-safety, and public-health implications in Nigeria. The methodology combined evidence from peer-reviewed literature, national surveillance data, international policy documents, and case studies from Nigeria and comparable low- and middle-income countries. This approach ensured that both scientific evidence and regulatory perspectives were incorporated into the synthesis.

2.1. *Data sources and search strategy*

A comprehensive literature search was conducted across multiple databases, including PubMed, Scopus, Web of Science, ScienceDirect, Google Scholar, and African Journals Online (AJOL). Grey literature was obtained from recognised international agencies such as the FAO, the World Health Organization (WHO), the United Nations Environment Programme (UNEP), and the International Code of Conduct on Pesticide Management (FAO/WHO).

Keywords and Boolean operators included: “pesticide resistance”, “pesticide residues”, “insecticide resistance”, “Nigeria”, “public-health pesticides”, “food contamination”, “environmental impacts”, “agricultural pesticides”, “vector resistance”, and “FAO/WHO pesticide policy.” The search covered publications between 2000 and 2025, with

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selected foundational works prior to 2000 included to provide historical context for resistance development.

2.2. *Eligibility criteria*

Inclusion criteria

Studies reporting pesticide use, resistance, residues, or health/environmental impacts in Nigeria.

- Articles describing resistance mechanisms in agricultural pests or disease vectors.
- Laboratory, field, epidemiological, toxicological, and environmental-monitoring studies.
- WHO, FAO, and national policy documents relevant to pesticide governance.
- Case studies from sub-Saharan Africa with conditions comparable to Nigeria.
- Peer-reviewed journal articles, conference papers, theses, and technical reports.

Exclusion criteria

- Studies without relevance to pesticide use or resistance.
- Articles lacking methodological clarity or empirical evidence.
- Duplicates, editorials, short commentaries, and non-scholarly blog-style publications.

3. Result

3.1. Pesticide Use and Resistance Trends in Nigeria

Pesticide use in Nigeria has risen sharply with expanding agricultural production, reflecting broader increases in pesticide consumption across developing countries. National assessments indicate intensive application of insecticides, herbicides, and fumigants in both staple and horticultural systems, while market surveillance—particularly in Southwest Nigeria—has detected organochlorine, organophosphate, carbamate, and pyrethroid residues in grains and fresh produce, in some cases approaching or exceeding international maximum residue limits.

Vector-control data show widespread and intensifying pyrethroid resistance, the primary class used in long-lasting insecticidal nets. A synthesis of entomological studies (2007–2025) confirms resistance in *Anopheles*, *Culex*, and *Aedes* species across multiple ecological zones, with notable variation in resistance intensity and mechanisms. Low mortalities to permethrin and deltamethrin, high frequencies of knock down resistance (kdr**)** mutations, and elevated cytochrome P450 activity point to combined target-site and metabolic resistance.

In agriculture, resistance is increasingly documented in stored-product beetles and major lepidopteran pests such as *Helicoverpa* spp. and *Spodoptera frugiperda*. Studies attribute reduced susceptibility to prolonged chemical use and P450-mediated detoxification, with recent fall armyworm outbreaks further intensifying selection pressure.

3.2. Concept and Mechanisms of Pesticide Resistance

3.2.1 Concept of pesticide resistance

Pesticides are widely used in agriculture, public health, and veterinary practice to control insects, weeds, fungi, rodents, and other pests. However, repeated and intensive application of synthetic chemicals has accelerated the emergence of resistance across multiple taxa, including insects, mites, fungi, weeds, bacteria, and rodents. This trend undermines the effectiveness of major pesticide classes and threatens the sustainability of pest-management programmes globally.

The Insecticide Resistance Action Committee (IRAC) defines resistance as *a heritable decrease in a pest population's sensitivity to a pesticide, resulting in repeated failure of a product when used according to recommended guidelines* (Al Naggar *et al*., 2025).

Resistance develops when pesticide exposure eliminates susceptible individuals, allowing those with resistance-conferring traits to survive and reproduce. These traits—whether dominant, recessive, or polygenic—are passed to offspring, gradually shifting the population's genetic composition. In the absence of natural enemies, resistant individuals proliferate rapidly, eventually replacing susceptible populations. This evolutionary process follows classical Darwinian selection, where rare alleles providing survival advantages increase in frequency under sustained chemical pressure.

Historically, resistance has emerged shortly after the introduction of major pesticide groups. The earliest recorded case dates to 1908, when *Quadraspidiotus perniciosus* failed to respond to sulfur treatments. By 1914, Melander observed sulfur and sulfur–lime resistance in additional scale insects. Following the commercialization of organochlorines and other synthetic insecticides in the 1940s, resistance cases expanded rapidly. *Musca domestica* developed resistance to DDT within a few years of its introduction, and similar patterns followed with organophosphates, carbamates,

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pyrethroids, formamidines, *Bacillus thuringiensis* toxins, avermectins, spinosyns, insect growth regulators, and neonicotinoids.

Today, more than 500 globally important pest species exhibit resistance to one or more pesticide classes. Talebi *et al*. (2011) reported resistance in 520 insect and mite species**,** 150 plant pathogens**,** and 273 weed species, illustrating the scale of this challenge. The consequences of pesticide resistance are extensive. Reduced susceptibility forces farmers and public-health practitioners to apply higher doses or switch to more toxic or expensive alternatives, increasing production costs and ecological risks. In the United States alone, economic losses attributed to pesticide misuse and resistance exceed several billion dollars annually, including costs related to public-health impacts, crop damage, wildlife mortality, and groundwater contamination (Talebi *et al*., 2011).

Mechanistically, resistance can arise through several pathways. Talebi *et al*. (2011) and Van Leeuwen *et al*. (2009) broadly classify these mechanisms into two categories:

1. processes that reduce the effective concentration of pesticides at the target site, and
2. changes that decrease the sensitivity of the target site itself.

The first group includes behavioural avoidance, reduced cuticular penetration, enhanced metabolic detoxification through enzyme systems such as cytochrome P450 monooxygenases, glutathione S-transferases, and esterases, as well as sequestration mechanisms that bind or isolate toxic compounds. The second group involves modifications to target molecules—such as mutations in voltage-gated sodium channels, acetylcholinesterase, or ryanodine receptors—that lower pesticide binding affinity.

Comparable mechanisms occur across pest groups. In mosquitoes, resistance commonly involves kdr mutations, overexpression of detoxification enzymes, and behavioural shifts that reduce contact with treated surfaces (Douglas & Anderson, 2020). In fungal pathogens, resistance may arise through target-site alterations, efflux pumps, and metabolic changes that degrade fungicidal compounds (Lai & Dik, 2020).

3.2.2 Mechanisms of pesticide resistance

Pesticide resistance arises through several well-established biological and evolutionary pathways that reduce a pest population's susceptibility to chemical control. These mechanisms often occur simultaneously and may interact to produce complex resistance phenotypes. The major mechanisms include:

- Target-site resistance
- Metabolic (detoxification) resistance
- Reduced penetration (cuticular) resistance
- Behavioural resistance
- Cross-resistance and multiple resistance

i. Target-site resistance

Target-site resistance occurs when mutations alter the molecular site where a pesticide binds, reducing the compound's affinity and diminishing toxicity (Washim *et al*., 2024). Point mutations in crucial proteins—including voltage-gated sodium channels, acetylcholinesterase, and GABA-gated chloride channels—are among the most well-documented. Examples include:

- L1014F kdr mutation in sodium channels, reducing sensitivity to pyrethroids and DDT.
- G119S (ace-1) mutation conferring resistance to organophosphates and carbamates.
- Mutations in GABA receptors reducing susceptibility to cyclodiene insecticides.

These mechanisms are widespread in mosquito vectors (*Anopheles*, *Aedes*, *Culex* spp.) and are increasingly recognized in agricultural pests. Target-site resistance is one of the most significant and well-characterized pathways and often acts synergistically with metabolic resistance.

ii. Metabolic (detoxification) resistance

Metabolic resistance is the most common and versatile mechanism. It enables pests to detoxify, degrade, or sequester pesticides before they reach their intended targets. It is mediated primarily by three enzyme families:

- Cytochrome P450 monooxygenases (P450s)
- Esterases
- Glutathione S-transferases (GSTs)

Overexpression or structural modification of these enzymes increases detoxification rates, often leading to cross-

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resistance. Synergist bioassays using compounds such as piperonyl butoxide (PBO), which inhibits P450s, frequently restore susceptibility (Vontas *et al*., 2020; Fotso-Toguem *et al*., 2022). Metabolic resistance is well documented in *Anopheles*, *Aedes*, and *Culex* mosquitoes, and in many agricultural insects. Because it is often polygenic and inducible, it presents a major challenge for resistance management.

iii. Reduced penetration (cuticular) resistance

Reduced penetration results from structural or compositional changes in the insect cuticle that limit pesticide absorption. A thicker or chemically modified cuticle slows pesticide entry, reducing internal concentrations.

This mechanism often acts synergistically with metabolic and target-site resistance. For example, *Cimex lectularius* (bed bugs) with thickened cuticles exhibit reduced susceptibility to pyrethroids.

iv. Behavioural resistance

Behavioural resistance involves changes in pest activity, feeding, or resting patterns that reduce contact with pesticides. Examples include:

- Mosquitoes shifting from indoor to outdoor biting, or feeding earlier in the evening.
- Crop pests avoiding treated surfaces or relocating to unsprayed plant parts.
- *Blattella germanica* developing glucose aversion, reducing bait efficacy.

Behavioural changes can significantly weaken the performance of otherwise potent insecticides.

v. Cross-resistance and multiple resistance

Cross-resistance occurs when one mechanism confers tolerance to several pesticides, often within related chemical classes (e.g., P450-mediated metabolism of both pyrethroids and organophosphates; **kdr** affecting both DDT and pyrethroids).

Multiple resistance occurs when a population accumulates several independent mechanisms simultaneously, resulting in broad-spectrum resistance. This situation is increasingly common in both vector and agricultural pests and complicates rotation-based resistance-management strategies.

3.2.3 Drivers of pesticide resistance

Pesticide resistance arises from a combination of biological, operational, and environmental drivers that enhance the selection and spread of resistant genotypes within pest populations. These factors influence the speed, intensity, and geographical distribution of resistance, thereby undermining the long-term effectiveness of pesticides. Resistance develops when pests survive exposure to a pesticide due to heritable traits and subsequently pass these traits to their offspring. Over time, these resistant individuals dominate the population, reducing the efficacy of control measures (Al Naggar *et al*., 2025). The major drivers are discussed below.

i. Over-reliance and continual use of pesticides

Excessive dependence on pesticides—particularly repeated application of chemicals with similar modes of action—is one of the strongest drivers of resistance. Continuous selection pressure eliminates susceptible individuals while allowing rare resistant phenotypes to survive and reproduce. When the same pesticide or chemical group is used season after season, the probability of resistance allele fixation rises significantly. This sustained directional selection gradually reduces overall sensitivity within the pest population. The problem is magnified in systems where pesticides are applied prophylactically rather than based on monitoring thresholds, making resistance evolution almost inevitable. Over-reliance is especially problematic in monoculture systems and high-input agriculture, where pesticide use is intense and frequent.

ii. Sub-lethal dosing, incorrect application rates, and poor application methods

Resistance may accelerate when pests are exposed to sub-lethal concentrations of pesticides due to misapplication, equipment malfunction, poor timing, or failure to follow recommended guidelines. Sub-lethal exposure does not kill pests but stresses them enough to favour individuals with partial resistance, thereby acting as a “training dose.” Such exposure enables heterozygous individuals—those carrying a single resistance allele—to survive and reproduce, making the resistance gene functionally dominant in the population.

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Table 1: Mechanisms of Pesticide Resistance with Nigeria-Specific Case Studies

Mechanism of Resistance	Description	Nigeria-Specific Case Studies	Implications	References
1. Target-Site Resistance (TSR)	Genetic mutations alter the pesticide binding site, reducing effectiveness.	• High frequency of *kdr* L1014F mutations in *Anopheles coluzzii* from northern Nigeria. • *kdr* mutations detected in *A. gambiae s.l.* populations in Lagos, Ogun, and Osun States. • Ache-mediated organophosphate resistance reported in *Helicoverpa armigera* on Nigerian tomato farms..	Resistance to pyrethroids and DDT; reduced effectiveness of long-lasting insecticidal nets (LLINs) and indoor residual spraying (IRS).	• Ibrahim *et al*. (2014) • Olayinka *et al*. (2024) • Mohammed *et al*. (2015)
2. Metabolic (Enzymatic) Resistance	Increased detoxification through elevated cytochrome P450s, esterases, and GSTs.	• Upregulated P450 enzymes in *A. funestus* from southwest Nigeria showing strong pyrethroid resistance (supported by patterns similar to West/Central Africa). • Enhanced esterase activity in *Aedes aegypti* populations from Lagos, enabling breakdown of organophosphates and pyrethroids. • Detoxification-enzyme mediated resistance in *Maruca vitrata* (cowpea pest) in northern Nigeria.	Cross-resistance to multiple chemical classes; increased pesticide use by farmers as efficacy declines.	•Ibrahim *et al*.(2019) • Fagbohun *et al*.(2020). • Kalua *et al*. (2023)
3. Reduced Penetration Resistance	Thickened cuticle or cuticular modifications reduce pesticide absorption.	• Thickened cuticle layers observed in *Anopheles* species from Sokoto and Kano, associated with declining pyrethroid susceptibility. • Reduced penetration suspected in *C. quinquefasciatus* populations in southwestern Nigeria with diminished response to pyrethroids.	Extended survival after exposure; "partial resistance" that escalates into full resistance over time.	•Habibu *et al*. (2017). • Ikpesu & Ariyo, (2013).
4. Behavioral Resistance	Pests alter behaviour to avoid pesticide exposure.	• Outdoor feeding and resting behaviour documented among *A. gambiae s.l.* in coastal and forest zones after prolonged LLIN use. • Early-evening biting patterns reported in mosquito populations in Cross River and Oyo States, reducing contact with insecticides. • Nocturnal feeding shifts in armyworm populations on maize fields in Kaduna.	Chemical interventions lose effectiveness even when pesticides remain potent; need for behaviour-targeting vector control tools.	• Ranson *et al*. (2011) • Awolola *et al*. (2003) • Tongo *et al*. (2022).
5. Cross-Resistance	Resistance to one pesticide confers resistance to others with similar modes of action.	• Pyrethroid-resistant Anopheles in northern Nigeria also resistant to DDT due to shared voltage-gated sodium channel target-site mutations. • Cross-resistance between carbamates and organophosphates in stored-product beetles (*Sitophilus* spp.) in Nigerian grain markets. • Cotton bollworms in Kano and Katsina exhibiting cross-resistance between synthetic pyrethroids and older organochlorines.	Limited available pesticide options; rapid spread of resistance across chemical classes.	• Hemingway *et al*., 2004). • Egbecho *et al*. (2019). • Akan *et al*., 2019).
6. Multiple (Multifactorial) Resistance	More than one mechanism occurs simultaneously in the same population.	• *A. funestus* from northern Nigeria showing combined metabolic detoxification + *kdr*-type mutations (consistent with multi-resistance profiles in Sahelian belt). • Multiple resistance in fall armyworm populations in Nigeria to pyrethroids, carbamates, and organophosphates. • Mixed modes of resistance in *A. gambiae* across south-west Nigeria involving target-site and metabolic pathways.	Rotation of pesticides becomes ineffective; chemical control reliability collapses; increased pressure on farmers and health systems.	•Djouaka *et al*., (2011). • Lengai *et al*., (2020). • Yusuf *et al*., (2021).
7. Sequestration	Pests compartmentalize pesticides into tissues where they cannot reach target	• Sequestration of pyrethroids suspected in *A. coluzzii* from Lagos during bioassays where mosquitoes survived despite high knockdown rates. • Stored-product pests such as Rhyzopertha dominica in northern grain markets showing delayed mortality	Reduced toxicity and delayed lethal action; dosing becomes unreliable; encourages overuse and higher	•Ibrahim *et al*., 2014). • Fadina *et al*. (2021).

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	sites.	patterns suggestive of sequestration.	concentration applications.	

Applying pesticides below recommended rates allows partially resistant individuals to persist, while excessively high doses can paradoxically hasten resistance by disproportionately killing susceptible and heterozygous individuals while leaving highly resistant homozygotes. Both under-dosing and over-dosing therefore create conditions that favour rapid selection of resistance. Inconsistent spray coverage—where parts of a field receive insufficient pesticide—also mimics sub-lethal exposure and promotes resistance development.

iii. Environmental factors and pest migration

Environmental factors—particularly temperature, humidity, and seasonal variability—influence pest reproduction, survival, and susceptibility to pesticides. Warmer temperatures often enhance pest metabolism and reproductive capacity, resulting in more generations per year and faster evolution of resistance. Climate change, through prolonged warm seasons and altered precipitation patterns, has intensified these dynamics.

Pest migration further complicates resistance management. Resistant individuals can move from treated to untreated areas or vice versa, spreading resistance genes across landscapes. Long-distance migration, wind dispersal (in aphids, fungal spores, and weed seeds), and human-mediated movement through trade, farm equipment, or seed exchange enable resistant populations to establish in previously unaffected regions. Conversely, migration of susceptible individuals into treated fields can dilute the frequency of resistance alleles—a principle used in the establishment of "refugia" for resistance management.

iv. Activity spectrum of the pesticide

Broad-spectrum pesticides increase the risk of resistance because they are applied more frequently and affect multiple pest species within a cropping system. This elevates overall selection pressure and may inadvertently select for resistance in non-target species present at low but persistent densities. Narrow-spectrum pesticides, though less frequently used, exert selective pressure on fewer species.

For example, broad-spectrum fungicides such as DMI and QoI compounds used in cereals can drive resistance in multiple fungal pathogens simultaneously, while highly specific fungicides like carboxylic acid amides pose lower cross-species pressure. When a secondary pest requires a higher application rate for control than the primary target, broad-spectrum use may disproportionately favour resistance development in the secondary species.

v. Monoculture-based cropping systems

Monocultures create uniform environments that favour rapid pest proliferation and often necessitate repeated pesticide use. Reduced biodiversity eliminates natural pest suppression mechanisms, increasing pest pressure and pesticide dependence. Simplified agroecosystems also intensify selection pressure for resistance, as pests encounter identical chemical environments across wide areas. The lack of crop rotation or diversification reduces ecological barriers to pest establishment and accelerates resistance evolution. Diversified farming systems, in contrast, create heterogeneous environments that slow resistance development by reducing pest population stability and exposure frequency.

vi. Genetic factors (presence and frequency of resistance genes)

For resistance to develop, resistance alleles must exist within the pest population. These alleles may arise through spontaneous mutation, genetic recombination, or migration from other populations. The speed at which resistance spreads depends on allele dominance, allele frequency, and associated fitness costs. Alleles that confer high resistance with minimal fitness penalties tend to spread rapidly. If resistant individuals suffer little reproductive disadvantage in the absence of pesticides, the alleles persist even when pesticide pressure is reduced. Conversely, resistance genes that impose high fitness costs may decline when pesticide exposure decreases.

vii. Pest dispersal and mobility

Pest mobility strongly influences the spread and persistence of resistance. Low-mobility pests tend to evolve resistance more rapidly within localized populations due to limited gene flow that would otherwise introduce susceptible individuals. In contrast, high-mobility species may spread resistance across wide regions.

Seed dispersal (in weeds), spore movement (in fungal pathogens), and insect flight can introduce resistant genotypes into previously susceptible populations. This is particularly problematic when resistant pests escape greenhouse environments and colonize surrounding fields. Adequate dispersal of susceptible individuals into treated zones,

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however, can help dilute resistance allele frequencies—an approach central to insect resistance management strategies using refugia.

viii. High reproductive rate of pests

Pests with short life cycles and high reproductive potential—such as aphids, whiteflies, and many lepidopterans—can evolve resistance rapidly. High fecundity increases genetic diversity and ensures that each generation contains individuals capable of surviving pesticide exposure. For example, *Aphis gossypii* produces many generations annually through parthenogenesis, enabling swift propagation of resistance traits once selected. Fast reproduction also amplifies the effect of repeated pesticide exposure within a single growing season, accelerating allele fixation.

ix. Behavioral changes

Behavioral resistance arises when pests modify their actions to avoid exposure to pesticides. Examples include avoiding treated surfaces, shifting feeding sites, altering feeding times, or developing aversions to baits. Such behavioural shifts can emerge rapidly and may represent early stages of resistance development before physiological mechanisms evolve. For instance, some insect species move deeper into crop canopies or change their diel activity patterns to avoid pesticide sprays. Behavioral avoidance reduces exposure, allowing pests to survive without physiological changes, but can later combine synergistically with metabolic or target-site resistance.

x. Metabolic adaptations

Pests may increase the production or efficiency of detoxification enzymes—such as esterases, cytochrome P450 monooxygenases, and glutathione S-transferases—that degrade or neutralize pesticides before they reach lethal concentrations. Gene amplification, upregulation, or point mutations in detoxification genes enhance metabolic detoxification capacity. This mechanism often confers cross-resistance to multiple pesticide classes with similar metabolic pathways. Metabolic resistance is especially common in insects exposed to organophosphates, carbamates, and synthetic pyrethroids.

xi. Type of reproduction

Both sexual and asexual reproduction influence resistance dynamics. Sexual reproduction increases genetic variability, potentially giving rise to resistance alleles through recombination. Once resistance emerges, asexual reproduction enables rapid clonal expansion of resistant individuals. Pathogens and insects that reproduce asexually—such as many aphids and fungal pathogens—can spread resistance extremely quickly because all offspring inherit identical resistant traits. In weeds, cross-pollinating species are more likely to disseminate resistance than self-pollinating or vegetatively reproducing species.

xii. Number of target sites

Pesticides with a single biochemical target site are more vulnerable to resistance than those with multiple targets. A single mutation can confer high-level resistance when only one target site is involved, as seen with many herbicides (e.g., ALS inhibitors) and insecticides (e.g., pyrethroids that target sodium channels). Compounds with multiple target sites require simultaneous mutations across several genes, which is far less likely. Therefore, pests exposed to single-site pesticides evolve resistance more quickly and consistently.

xiii. Pest host range

Generalist pests that feed on multiple crops face more frequent pesticide exposure than specialist pests. Because they encounter various treated fields in different seasons, their cumulative exposure per year is higher, accelerating resistance development. For instance, insect pests receiving several treatments on cotton may encounter additional applications on nearby vegetable crops, resulting in far more total selections than anticipated by crop-specific management plans. Area-wide resistance management is therefore critical for pests with broad host ranges.

xiv. Spray coverage

Effective spray coverage ensures that pests receive lethal doses across the entire treated area. Poor coverage results in uneven distribution, creating pockets of pests exposed only to sub-lethal doses. Such inconsistent exposure mirrors under-dosing and leads to the survival of partially resistant individuals. Good spray technology, calibrated sprayers, and

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adherence to recommended spray volumes are essential to minimize this risk.

xv. Treatment frequency

Excessive treatment frequency, particularly when combined with sub-optimal dosing, intensifies selection pressure and accelerates resistance. Frequent applications kill primarily susceptible individuals while allowing resistant phenotypes to recover and dominate. In areas where pest pressure or migration necessitates repeated treatments, rotating pesticides with different modes of action is critical. Over-application often leads to rapid resistance followed by pest resurgence—sometimes at levels exceeding those before pesticide use.

xvi. Life stage treated

The life stage at which pests are targeted influences resistance development. Some life stages—such as neonate larvae or newly emerged adults—are more vulnerable to pesticides and have lower detoxification capacity. Treating pests at these vulnerable stages reduces the probability of resistant individuals surviving and passing on their genes. However, field populations often consist of mixed life stages, making stage-targeted treatments challenging. Using different pesticides against different life stages may reduce the likelihood of individuals surviving across stages, thereby slowing resistance evolution.

3.3. Impacts of Pesticide Resistance

Pesticide resistance initiates a cascade of agricultural and public health challenges. As resistance emerges, farmers often attempt to compensate for reduced efficacy by increasing pesticide doses or application frequency. Such practices amplify environmental contamination, elevate pesticide residues in food, and intensify health and ecological risks. Resistance and pesticide residues are therefore inextricably linked, creating a cycle that undermines sustainable agriculture and public safety.

3.3.1 Impacts of pesticide resistance in agriculture

Global population growth, projected to reach 10 billion by 2050, exerts immense pressure on agricultural systems to increase food production by approximately 70% (FAO; Raimi *et al*., 2019; Olalekan *et al*., 2019; Oshatunberu *et al*., 2023). To meet this demand, farmers rely heavily on agrochemicals—including insecticides, herbicides, fungicides, nematicides, and fertilizers—to protect crops and enhance yields (Asiegbu *et al*., 2022; Isah *et al*., 2020).

Since the 1940s, synthetic organic pesticides—initially organochlorines—have dominated agriculture. Subsequent resistance and international regulations prompted shifts to organophosphates, carbamates, and pyrethroids (Raimi *et al*., 2020). Despite restrictions, persistent organic pollutants (POPs), such as OCPs, are still illicitly used in West African agriculture due to efficacy and long residual activity. These chemicals are hydrophobic, bioaccumulative, and resistant to degradation, posing severe ecological and human health risks (Asiegbu *et al*., 2022).

Pesticides are ideally selective, biodegradable, and environmentally benign. However, most are broad-spectrum, affecting non-target organisms and contaminating soils, water, and crops. Estimates indicate only 0.1% of applied pesticides reach target pests, with the remainder entering ecosystems (Suleiman *et al*., 2019; Asiegbu *et al*., 2022). Pest infestations alone can reduce crop yields by up to 45%, highlighting the need for effective control, yet repeated pesticide use accelerates resistance evolution. Integrated pest management (IPM) is thus essential to balance crop protection with environmental and human health (FAOSTAT, 2021; Singh *et al*., 2024).

The primary consequence of resistance is diminished pest control effectiveness, reducing yields. Resistant populations survive treatments, causing persistent infestations. For instance, the Colorado potato beetle (*Leptinotarsa decemlineata*) has developed resistance to multiple insecticides, leading to marked yield losses (Cohen *et al*., 2008). Documented cases in West Africa include *Helicoverpa armigera* and fall armyworm (*Spodoptera frugiperda*), which exhibit pyrethroid tolerance linked to P450 overexpression (Tossou *et al*., 2019). Herbicide resistance is also escalating, with *Amaranthus palmeri* in Argentina serving as a cautionary example (Liu *et al*., 2020), while fungicide resistance threatens crop protection globally (Lai & Dik, 2020).

Herbicides constitute over 72% of pesticides used in agriculture (Sharma *et al.*, 2019; Sahoo *et al*., 2024 Oyetunji *et al*., 2024), but residues frequently accumulate in topsoil, affecting microbial activity (Ghazi *et al*., 2023).

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Overreliance on single pesticides can disrupt pest populations, causing secondary pest outbreaks, as observed in cotton farms where aphid populations surged after broad-spectrum insecticide use. Economic implications are substantial: increased doses, frequent applications, and switching to more potent chemicals raise input costs, eroding profitability—particularly for smallholder farmers.

3.3.2 Pesticides and food insecurity

i. Global trends

Pesticides enhance crop yields and protect food quality, but misuse has raised concerns regarding food safety, human health, and ecological sustainability. Developing nations rely heavily on agrochemicals due to limited access to advanced technologies (Asiah *et al*., 2019; Tudi *et al*., 2022). Global pesticide use has increased sharply; annual consumption reached an estimated 3.5 million tonnes by 2020, with low- and middle-income countries experiencing up to 153% growth (Shattuck *et al*., 2023; Salceanu *et al*., 2022). Despite regulations, monitoring gaps in many developing countries allow residues to exceed Maximum Residue Limits (MRLs) in food commodities (Benbrook, 2023; Yang *et al*., 2024). Continuous exposure can disrupt endocrine function, cause neurotoxicity, and compromise reproductive health. Herbicides make up 47.5% of global pesticide use, followed by insecticides (29.5%) and fungicides (17.5%), with 33% of agricultural products involving pesticides in production (Oyetunji *et al*., 2024).

ii. Pesticide residues in food in Nigeria

Nigeria exemplifies the interplay of pesticide use, food production, and safety. Multiple studies across agro-ecological zones reveal pesticide residues in cereals and legumes exceeding international limits:

- Rivers State: 23 pesticide residues detected in rice, maize, beans, and groundnuts, mainly organochlorines and organophosphates (Egbecho *et al*., 2019).
- Kwara, Oyo, Osun, Ekiti, Ondo: >70% of grain samples exceeded EU/FAO limits (Sosan *et al*., 2018; Oshatunberu *et al*., 2023).
- Southwest Nigeria: Organochlorines (β-HCH, heptachlor, endosulfan, aldrin, lindane, dieldrin, DDT), organophosphates (dichlorvos, carbofuran, carbaryl, diazinon, pirimiphos-methyl, malathion, chlorpyrifos), and pyrethroids (cypermethrin, flumioxazin) detected in maize, beans, millet, and rice (Oshatunberu *et al*., 2023).
- Makurdi (Benue State): Dichloran, heptachlor epoxide, propyzamide, chlorpyrifos, diazinon, endosulfan II, methoxychlor, and mirex prevalent in rice samples (Adah *et al*., 2020).
- Borno State: Herbicide residues in rice exceeded MRLs (Mohammed *et al*., 2020; Oyetunji *et al*., 2024).
- North-Central Nigeria: Cowpea and maize contained 23 residues (DDT, DDE, atrazine, simazine) at 72.67–159.67 mg/kg, exceeding 20 mg/kg thresholds (Ekhuemelo, 2023).
- Taraba State: Excess heptachlor, aldrin, and endosulfan detected (Abare-Jen, 2023; Barau *et al*., 2023).
- Anambra and Oyo States: Aldrin, endrin, and endosulfan above recommended limits (Omokpariola *et al*., 2023; Olutona & Aderemi, 2019).
- Enugu, Plateau, Lagos, Ibadan: DDT derivatives, methoxychlor, glyphosate detected in cowpea, millet, legumes, and rice (Omeje *et al*., 2021; Oladapo *et al*., 2022; Atsen *et al*., 2021).
- Anambra, Rivers, Kogi: Beans contained dichlorvos, glyphosate, carbofuran above EU/WHO limits (Kalu *et al*., 2023; Olajide *et al*., 2024; Dike-Iheanyi *et al*., 2024).
- Borno State: DDT, lindane, diazinon residues in pre- and post-harvest beans (Obida *et al*., 2012).
- Taraba and Ibadan: Similar contamination patterns (Arowora *et al*., 2020; Fadina *et al*., 2021).

Pesticide misuse limits food system functionality by reducing availability, accessibility, utilization, and stability. Pre- and post-harvest contamination diminishes nutritional safety, reduces market access, and undermines both domestic and export food security.

3.3.3 Economic implications and regulatory failures

The persistence of pesticide residues imposes significant economic costs. In 2015, the EU banned Nigerian bean imports due to excessive dichlorvos (0.03–4.60 mg/kg), exceeding the 0.01 mg/kg MRL. Chlorpyrifos and omethoate were also detected at toxic levels, reflecting inadequate national monitoring and regulatory enforcement. Such bans disrupt livelihoods, depress farmer income, and undermine food security.

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Weak enforcement, insufficient farmer education, and limited residue monitoring perpetuate unsafe pesticide use. Banned or restricted chemicals such as DDT and endosulfan remain accessible in local markets due to poor border control and limited consumer awareness. These systemic failures hinder Nigeria's progress toward SDG 2 (Zero Hunger) and SDG 3 (Good Health and Well-Being).

3.3.4 Pesticide resistance and public health implications

Pesticide resistance elevates exposure risks for humans. Extensive use in agriculture, vector control, and domestic settings increases acute and chronic toxicity. Epidemiological studies correlate pesticide exposure with neurological, reproductive, metabolic, and carcinogenic effects (Ahmad *et al*., 2024; Tudi *et al*., 2022). Farmers and applicators experience higher exposure than consumers, compromising workforce productivity, agricultural sustainability, and food system safety.

3.4. Pathways of Exposure and Mechanisms of Toxicity

In Pesticides enter humans via inhalation, ingestion, or dermal absorption. Toxicity severity depends on chemical class, dose, exposure frequency, and individual susceptibility (Ansari *et al*., 2024). Organophosphates, carbamates, and organochlorines inhibit acetylcholinesterase, disrupting neural transmission and causing paralysis or death in severe cases (Grewal *et al*., 2017). Chronic low-dose exposure is linked to neurological, reproductive, metabolic, and carcinogenic outcomes (Tudi *et al*., 2022; Ahmad *et al*., 2024).

3.4.1 Acute toxicity

Acute pesticide poisoning remains a significant occupational hazard. Symptoms include dizziness, salivation, diarrhea, convulsions, coma, and death. Outbreaks in Anambra and Benue (2018) from organophosphate-contaminated beans and yam flour resulted in fatalities, revealing regulatory and educational gaps (Shaibu, 2008; Okon, 2018).

3.4.2 Chronic toxicity and systemic health disorders

Chronic exposure affects neurological, endocrine, renal, cardiovascular, and hepatic systems. Persistent organochlorines bioaccumulate in fatty tissues, disrupting hormones and immunity (Panis *et al*., 2022b). Women and children are particularly vulnerable during mixing, spraying, and storage.

i. *Carcinogenic and genetic effects*

Pesticides induce oxidative stress, DNA damage, epigenetic alterations, and endocrine disruption, contributing to cancers. Glyphosate, chlorpyrifos, and mancozeb alter DNA methylation and histone acetylation, affecting cell proliferation, apoptosis, and tumor suppression (Benbrook, 2023; Tudi *et al*., 2022). Organochlorines act as xenoestrogens, promoting hormone-dependent cancers (PAN-Germany, 2012; Ahmad *et al*., 2024). Epidemiological studies link pesticide exposure to leukemia, lymphoma, multiple myeloma, breast, ovarian, prostate, liver, kidney, pancreatic, and lung cancers (Alrashdi *et al*., 2023; Gerken *et al*., 2024).

ii*. Genetic and reproductive consequences*

Mutagenic pesticides induce sperm DNA fragmentation, meiotic disruptions, and miscarriages. Maternal exposure increases risks of neural tube defects, autism, and cognitive impairments in offspring (Tudi *et al*., 2022; Sosan *et al*., 2018). Pesticides detected in breast milk indicate intergenerational transfer of toxins, threatening human capital and food security.

iii. Respiratory disorders

Exposure is linked to asthma, COPD, bronchitis, and lung cancer. Residential proximity to treated areas correlates with increased wheezing, allergic responses, and lower respiratory tract infections in children (Horne *et al*., 2024; Elsiwi *et al*., 2024; Islam *et al*., 2023). Occupational exposure also elevates COPD risk (De Matteis *et al*., 2021; Tarmure *et al*., 2020).

iv. Neurotoxicity

Pesticides disrupt neurotransmission, mitochondrial function, and oxidative balance, contributing to neurodegenerative diseases such as Parkinson's, Alzheimer's, and cognitive decline (Ratner *et al*., 2014; Marina *et al*., 2020; Dorsey &

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Bloem, 2024). Paraquat, organophosphates, dieldrin, and benomyl impair dopaminergic neurons and detoxification processes, increasing PD risk (Doorn *et al*., 2014; Boyd *et al*., 2020).

v. Kidney, liver, and metabolic disorders

Herbicides such as paraquat, glyphosate, and atrazine are nephrotoxic and hepatotoxic, leading to chronic kidney disease, hepatic fibrosis, diabetes, and obesity (Oshatunberu *et al*., 2023). Chronic exposure reduces agricultural labor capacity and household income.

vi. Endocrine and reproductive health effects

Pesticides function as endocrine disruptors, impairing growth, metabolism, reproduction, and immunity. Prenatal exposure affects neurodevelopment and behavior in children, threatening future agricultural productivity.

vii. Socioeconomic implications

Pesticide resistance imposes financial burdens, increases farmer vulnerability, and exacerbates poverty-driven food insecurity. Repeated pesticide applications due to resistance degrade ecosystems and reduce sustainable farming options. Export rejections and domestic contamination incidents further depress incomes (IPEN, 2021).

viii. Food security implications

Health burdens from pesticide-induced cancers and chronic illness reduce labor productivity, increase medical costs, and constrain investment in agriculture. Residue contamination in staple crops compromises availability, access, utilization, and stability of food systems. Nigeria's 2015 EU bean ban exemplifies the interconnected risks of pesticide misuse, public health, and food security (FAO, 2016; Oguntade *et al*., 2020).

Addressing these challenges requires integrated approaches: stricter regulation, biomonitoring, farmer education, and adoption of safer, eco-friendly pest control methods aligned with One Health and Planetary Health frameworks.

3.5. Malaria Vectors (Anopheles spp.) and Pesticide Resistance

Mosquito-borne diseases, particularly malaria, remain a major public health challenge in sub-Saharan Africa, with Nigeria bearing the highest global burden. Malaria alone causes over 100 million cases and 200,000 deaths annually in Nigeria, contributing significantly to outpatient visits, hospitalizations, childhood mortality, and maternal morbidity (Wariso & Oboro, 2015; Yusuf *et al*., 2021; Babalola *et al*., 2021). Other mosquito-borne diseases, including lymphatic filariasis, dengue, yellow fever, and chikungunya, further strain Nigeria's health system (Babalola *et al*., 2025).

Human malaria is caused primarily by *Plasmodium falciparum*, the most virulent species, and is transmitted by the *Anopheles gambiae* complex—comprising seven morphologically similar species, including *A. gambiae* s.s., *A. coluzzii*, and *A. arabiensis*. These vectors are highly anthropophilic and ecologically adaptable, contributing to widespread malaria transmission and associated socioeconomic losses, such as reduced agricultural productivity and school absenteeism (Geleta & Ketema, 2016; Adewale *et al*., 2016). In Nigeria, *An. gambiae* s.l. accounts for 65.2% of vectors, followed by *An. funestus* at 17.3% (Ibrahim *et al*., 2014).

Vector control relies heavily on insecticide-based strategies, including long-lasting insecticide-treated nets (LLINs) and indoor residual spraying (IRS) (Adeogun *et al*., 2023). However, the emergence of insecticide resistance threatens their effectiveness (Thiaw *et al*., 2018; Yusuf *et al*., 2021) (Table 2). Insecticides in use include organophosphates (chlorpyrifos, malathion), carbamates (bendiocarb, propoxur), pyrethroids (permethrin, deltamethrin, cypermethrin), and neonicotinoids, while restricted organochlorines such as DDT and endosulfan remain in limited use (Ibrahim *et al*., 2014; Rejmánková *et al*., 2005).

Resistance to pyrethroids—the only class approved for LLINs—is now widespread across Nigeria. Mechanisms include metabolic detoxification via cytochrome P450s, esterases, and glutathione S-transferases, as well as target-site knockdown resistance (kdr) mutations L1014F and L1014S in voltage-gated sodium channels, conferring resistance to DDT and pyrethroids (Oyewole *et al*., 2009; Fagbohun *et al*., 2020; Awolola *et al*., 2003).

State-specific studies highlight the severity of resistance: in Kano State, *A. coluzzii* exhibited high resistance to DDT and pyrethroids, with emerging bendiocarb resistance; pre-exposure to piperonyl butoxide partially restored susceptibility, implicating metabolic mechanisms (Ononamadu *et al*., 2020; Liu, 2015). Bichi (Northern Nigeria)

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reported elevated detoxification enzymes and very high resistance to permethrin and DDT, with lower bendiocarb resistance (Safiyanu *et al*., 2016; Habibu *et al*., 2017). Resistance levels are highest in forest savannah, mosaic, and Guinea savanna zones (Mohammed *et al*., 2015). Auyo and Bunkure populations also showed kdr-mediated resistance (Ibrahim *et al*., 2014). Yusuf *et al*. (2021) reported widespread *An. gambiae* resistance to deltamethrin, DDT, malathion, and bendiocarb across three northern states (Table 2).

The indiscriminate use of agricultural pesticides—including organophosphates, pyrethroids, carbamates, and organochlorines—exacerbates resistance selection pressure. Resistance compromises malaria control programs, as most LLINs rely on pyrethroids, and the coexistence of multiple resistance mechanisms limits rotation or combination strategies (Fagbohun *et al*., 2020). Resistance is also reported in *Aedes* and *Culex* species, affecting all major insecticide classes (Babalola *et al*., 2025; Olayinka *et al*., 2024).

Recent studies confirm escalating pyrethroid resistance across sub-Saharan Africa, including high levels in Okitipupa, Southwest Nigeria (Owolabi & Ayankoya, 2023) (Table 2). Increased use of LLINs, IRS, aerosols, and agricultural pesticides drives this trend. Overall, target-site mutations and enhanced metabolic detoxification are major drivers of resistance, posing significant challenges to malaria elimination in Nigeria and across Africa (Ranson *et al*., 2011).

3.6. Environmental Impacts of Pesticide Resistance

Pesticides are widely used to protect crops, stored produce, gardens, and to control disease vectors, with global spending estimated at nearly **$38 billion** annually (Pan-Germany, 2012). Ideally, pesticides should target only harmful organisms and be biodegradable and environmentally safe (Rosell *et al*., 2008). In practice, most are non-selective; only **0.1%** reach target pests, while the remainder contaminates soil, water, and air (Carriger *et al*., 2006). Persistent pesticides bioaccumulate in food chains, posing acute and chronic health risks to humans and wildlife (Mostafalou & Abdollahi, 2012). Integrated Pest Management (IPM) offers an eco-friendly alternative that reduces reliance on chemical pesticides.

3.6.1 Implications for ecosystem services

Pesticide exposure significantly affects ecosystem services. Declines in insect populations—particularly pollinators and decomposers—reduce food production, soil health, and nutrient cycling. Sensitive species suffer direct toxicity or habitat disruption. For example, neonicotinoids affect bee foraging behavior, immunity, and reproduction, with cascading effects on plant pollination and predator–prey relationships. Reduced insect diversity can result in overpopulation of pest species, impaired pollination, and diminished soil fertility. Insect-mediated ecosystem services, including pollination, organic matter decomposition, and biological pest control, are essential for natural ecosystems and agriculture. Loss of these services has direct economic and food security implications.

3.6.2 Pesticides, biodiversity loss, and ecosystem resilience

Biodiversity underpins ecosystem resilience, enabling recovery from disturbances. Pesticide-induced declines in insect populations weaken resilience, making ecosystems more vulnerable to environmental changes. Global concern has grown over the loss of biodiversity and ecosystem services caused by pesticide misuse. Neonicotinoids, in particular, have sublethal effects on bees, butterflies, aquatic organisms, and soil biota, affecting foraging, reproduction, and population dynamics (Goulson & Nicholls, 2020). Pesticides also affect birds and mammals, with bioaccumulation causing reproductive failures. Granivorous birds are particularly vulnerable to seed-treatment exposure, while top predators experience persistent toxic effects due to lipid accumulation (Gilden *et al*., 2021; Nehul, 2025). Maintaining ecosystem resilience requires sustainable pest management and protection of biodiversity.

3.6.3 Pesticides, soil biota, and soil health

Soil acts as the primary sink for pesticides, where repeated applications alter microbial diversity, biomass, enzymatic activity, and key biochemical processes such as nitrogen fixation and organic matter mineralization (Hussain *et al*., 2009; Muñoz-Leoz *et al*., 2011). Soil microorganisms, crucial for nutrient cycling and soil structure, are particularly vulnerable. Fungicides like **carbendazim** reduce soil respiration and enzyme activity, while herbicides such as **atrazine** persist in soil and disrupt nitrogen-fixing bacteria and mycorrhizal fungi (Nehul, 2025). Repeated pesticide applications reduce organic carbon content and populations of beneficial microbes responsible for phosphorus solubilization and nitrogen fixation, affecting long-term agricultural sustainability (Singh *et al*., 2022).

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Table 2: Mechanisms of Malaria Vector Resistance to Different Pesticides in Nigeria

State / Location	Dominant Vector Species	Insecticide Resistance Profile	Mechanism / Notes	Reference
Kano (Northern Nigeria)	*A. coluzzii*	High resistance to DDT and pyrethroids; emerging resistance to bendiocarb	Resistance partially restored by PBO, indicating metabolic detoxification	Ononamadu *et al*., 2020; Liu, 2015
Bichi (Northern Nigeria)	*A. gambiae s.s.*	Very high resistance to permethrin and DDT; lower resistance to bendiocarb	Elevated detoxification enzyme levels in resistant strains	Safiyanu *et al*., 2016; Habibu *et al*., 2017
Auyo (Northern Nigeria)	*A. gambiae s.l.*	Resistance to commonly used insecticides (DDT, pyrethroids, carbamates)	Presence of kdr mutations	Ibrahim *et al*., 2014
Bunkure (Northern Nigeria)	*A. gambiae s.l.*	Resistance to DDT, pyrethroids, carbamates	kdr mutations confirmed	Ibrahim *et al*., 2014
Forest Savanna Zone	*A. gambiae s.s.*	High resistance to permethrin and DDT	Multiple resistance mechanisms	Mohammed *et al*., 2015
Mosaic / Guinea Savanna Zones	*A. gambiae s.s.*	High resistance to permethrin and DDT	Resistance associated with kdr and metabolic detoxification	Mohammed *et al*., 2015
Okitipupa (Southwest Nigeria)	*An. gambiae s.l.*	High pyrethroid resistance	Resistance exacerbated by LLINs, IRS, aerosols, and agricultural pesticide exposure	Owolabi & Ayankoya, 2023

Certain pesticides, including carbamates and organophosphates, chelate metal ions, reducing plant nutrient uptake and affecting photosynthesis, reproduction, and seed production (Ayilara *et al*., 2023). Soil contamination also impacts earthworms and other soil-dwelling organisms critical for nutrient cycling. Some microbes metabolize pesticides as carbon sources, while others are suppressed, altering ecosystem functions (Bhagobaty & Malik, 2010; Lupwayi *et al*., 2009). Factors such as soil texture, pH, organic matter, moisture, and microbial composition influence pesticide degradation and persistence (Pal & Tah, 2012; Gianelli *et al*., 2013). Advanced techniques like metagenomics are increasingly used to assess pesticide effects on soil microbial diversity and function (Ono *et al*., 2007; Souza *et al*., 2013).

3.6.4 Pesticides in water

Pesticides enter aquatic systems via runoff, effluents, or spray drift, threatening fish, amphibians, and aquatic communities (Larson *et al*., 2010). Volatilization also contributes to atmospheric contamination (Trajkovska *et al*., 2009). Water contamination affects ecosystem health and drinking water quality. Pesticide mobility and persistence depend on chemical properties, soil characteristics, climate, and application methods.

Aquatic ecosystems are particularly vulnerable. In Nigeria, **aldrin** was detected in the Ikpoba River, while **diazinon, endrin, glyphosate, endosulfan I, heptachlor**, and **carbofuran** have been reported in surface waters, with associated **Fe, Mn, Cd,** and **Pb** exceeding WHO limits (Tongo *et al*., 2022; Oyetunji *et al*., 2024). Organophosphate insecticides can cause acute toxicity in fish, amphibians, and invertebrates. **Atrazine** disrupts amphibian endocrine systems, causing hermaphroditism and reproductive abnormalities (Nehul, 2025). **Glyphosate** residues were found in all 75 fish tissue samples from Ibadan markets, exceeding the maximum residue limit of **0.01 mg/L** (Alarape *et al*., 2023). Other detected herbicides include **butachlor, metolachlor, paraquat, propachlor, propanil**, and **alachlor** (Akan *et al*., 2019; Olatoye *et al*., 2021).

Pesticide exposure can cause deformities in reptiles and amphibians, including malformed jaws, limbs, and cranial structures, while fish show gill, liver, brain, and reproductive damage (Garces *et al*., 2020; Konar, 2011; Kumari, 2012). Herbicides like glyphosate increase tadpole mortality, and insecticides like **malathion** disrupt aquatic food chains (Brühl *et al*., 2013). Even legally permissible concentrations can pose ecological risks, highlighting the importance of monitoring.

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3.6.5 Pesticides, bioaccumulation, and biomagnification

Persistent pesticides accumulate in organisms' tissues, leading to bioaccumulation and biomagnification, particularly for lipophilic compounds like organochlorines. Biomagnification occurs when pesticide concentration increases at successive trophic levels, disproportionately affecting top predators such as birds of prey, fish-eating species, and mammals (Chandler *et al*., 2011). Classic examples include DDT-induced eggshell thinning in raptors, leading to population declines in the 1960s–1970s (Nehul, 2025).

In Louisiana lakes, tertiary consumers such as green-backed herons and snakes contained higher organochlorine residues than secondary consumers like bluegill and shiners (Niethammer *et al*., 1984). In Lake Ziway, Ethiopia, top predator catfish (*Clarias gariepinus*) had higher 4,4′-DDE concentrations than tilapia (*Oreochromis niloticus*), *Tilapia zillii*, and goldfish (*Carassius auratus*) (Deribe *et al*., 2013). In Nigeria, similar patterns are documented, with DDT and endosulfan residues in aquatic systems and crops posing chronic toxicity risks (Adegun *et al*., 2020; Omeje *et al*., 2021; UNEP, 2023).

Bioaccumulation and biomagnification also impact terrestrial wildlife. Pesticides can cause reproductive failures, deformities, and chronic toxicity in birds, mammals, and pollinators (Ayilara *et al*., 2023). For instance, crossed-bill deformities in wild bald eagles have been linked to high DDT exposure. Persistent, lipophilic pesticides show higher accumulation and environmental persistence than organophosphates, emphasizing the need for pre-application risk assessments (Favari *et al*., 2002). Biomagnification is quantified using the biomagnification factor **(**BMF**)**, representing the predator-to-prey tissue concentration ratio; values above 1 indicate increasing concentrations along trophic levels (Nehul, 2025). Trophic magnification factors (TMFs) provide ecosystem-wide measures, highlighting the highest pesticide burdens in apex predators.

3.6.6 Pesticides, heavy metals, and food safety

Inappropriate pesticide and herbicide use can result in heavy metal accumulation in soils and crops, posing risks to human health. In Nigeria, cassava and bean–yam cultivation have shown excessive Cd, Fe, Mg, and Cl due to herbicide application, contributing to soil and water contamination (Onasanya *et al*., 2021; Oyetunji *et al*., 2024). Chronic exposure to heavy metals through contaminated crops can cause food poisoning and long-term health risks. These findings underscore the need for careful pesticide management and monitoring to protect human health and the environment.

3.7.1 Integrated Pest Management

Integrated Pest Management (IPM) is a holistic approach that combines multiple strategies to minimize reliance on chemical pesticides. It emphasizes understanding pest ecology and employing biological control, habitat manipulation, cultural practices, and resistant crop varieties. Chemical pesticides are used only as a last resort and applied in a targeted manner to minimize harm to non-target species (Gill *et al*., 2013; Sharma & Ortiz, 2020).

IPM promotes the development and use of eco-friendly pesticides that are specific to target pests and degrade rapidly in the environment. Biopesticides derived from plants, bacteria, or minerals are less toxic, more sustainable, and safer for ecosystems. Diversifying agricultural landscapes through hedgerows, flower strips, and cover crops provides habitats for beneficial insects and enhances natural pest control.

Education and training for farmers are essential, increasing awareness of pesticide risks, biodiversity conservation, and IPM principles. Supportive policies and regulations also promote sustainable pest management through incentives, research investment, and biodiversity conservation initiatives. Continuous monitoring and research help track pest populations, assess mitigation strategies, and develop safer alternatives.

Core IPM tenets include:

- **Accurate Identification & Monitoring:** Using field scouting, traps, and modern tools, including satellite-based systems, to assess pest populations.
- **Action Thresholds:** Implementing control measures only when pest levels exceed predetermined thresholds.
- **Prevention:** Employing cultural practices such as crop rotation, sanitation, and the use of resistant varieties.
- **Control:** Prioritizing biological and mechanical methods before chemical approaches.

Key IPM measures also include pesticide rotation to reduce selection pressure, cultural and mechanical control (crop rotation, tillage, weeding), and host plant resistance through breeding and biotechnology (Gill *et al*., 2013; Sharma &

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Ortiz, 2020).

3.7.2 The use of biopesticides

Biopesticides are pest-control agents derived from natural materials such as plants, animals, bacteria, fungi, and minerals (Daraban *et al*., 2023). According to the European Environment Agency (EEA, 2023), biopesticides destroy pests through specific biological mechanisms rather than chemical poisoning, resulting in minimal impact on humans and the environment. Unlike synthetic pesticides, they are highly specific, environmentally friendly, and less harmful to non-target organisms (Nehul, 2025). Biopesticides are cost-effective, sustainable, non-persistent, and help prevent resistance development (Borges *et al*., 2021; Ayilara *et al*., 2023).

Biopesticides function by denaturing proteins, causing metabolic disorders, paralyzing pests, activating target-specific toxic mechanisms, and releasing bioactive compounds. These diverse modes of action help counter resistance (Ayilara *et al*., 2023).

The U.S. Environmental Protection Agency classifies biopesticides into three categories:

1. **Microbial Pesticides:** Contain active microorganisms such as bacteria, fungi, viruses, or protozoa. *Bacillus thuringiensis* (Bt) is widely used against insect larvae, producing crystal proteins toxic to pests but safe for mammals, birds, and beneficial insects (Mostafalou *et al*., 2013). Fungal biopesticides such as *Beauveria bassiana* and *Metarhizium anisopliae* infect insects through spore germination and mycelial growth.
2. **Plant-Incorporated Protectants (PIPs):** Genetically modified crops engineered to express pesticidal proteins (e.g., Bt toxins) for season-long protection.
3. **Biochemical Pesticides:** Naturally occurring compounds that work through non-toxic mechanisms, such as pheromones, plant growth regulators, and essential oils (Šunjka & Mechora, 2022; Daraban *et al*., 2023). Common examples include nicotine, rotenone, neem, pyrethrins, Bt, and spinosad.

Biopesticides degrade rapidly—within days or weeks—significantly reducing soil and water contamination and limiting bioaccumulation (Daraban *et al*., 2023). Their specificity enables effective control of resistant pest strains, making them essential components of Integrated Resistance Management (IRM). They also protect beneficial organisms such as pollinators and natural enemies, thus supporting ecosystem stability (Flexner *et al*., 1986).

Plant Extracts and Essential Oils:

Extracts from *Artemisia annua*, *A. absinthium*, *A. camphorata*, *A. dracunculus*, and *A. vulgaris* have shown 80–96% efficacy against *Diaphania hyalinata* larvae, while remaining selective toward beneficial species (Seixas *et al*., 2018; Chaieb *et al*., 2018; Daraban *et al*., 2023). Other potent extracts—such as those from *Achillea millefolium* and *Origanum vulgare*—have shown activity against *Plutella xylostella*, *Acrobasis advenella*, and *Glyphodes pyloalis* (Yazdani *et al*., 2014; Nas *et al*., 2017; Ahmadi *et al*., 2018).

Microbial Biopesticides:

Key genera include *Chromobacterium*, *Pseudomonas*, *Yersinia* (bacteria); *Beauveria*, *Paecilomyces*, *Verticillium*, *Hirsutella*, *Metarhizium*, *Lecanicillium* (fungi); and nematodes such as *Steinernema* and *Heterorhabditis* (Kumar *et al*., 2021). Bt remains the most widely used microbial pesticide. After ingestion, its toxins dissolve in the insect gut, releasing delta-endotoxins that cause death. Bt is particularly effective in controlling lepidopteran pests in cabbage and potatoes (Xiao & Wu, 2019; Ayilara *et al*., 2023).

Fungal Biopesticides (Mycoinsecticides): Mycoinsecticides use living fungi to invade and kill insects through stages including attachment, germination, penetration, invasion, replication, and host death (Zaki *et al*., 2020). *Trichoderma spp.* controls soil-borne fungi causing root rot, whereas *Beauveria bassiana* and *Metarhizium brunneum* target thrips, beetles, aphids, whiteflies, and mites.

Biopesticides therefore provide sustainable, safe, and effective pest management, offering low environmental persistence, reduced resistance risk, and protection of beneficial species. Their integration into pest-management systems is essential for maintaining crop productivity and safeguarding ecosystems.

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3.7.3 policy and regulatory measures

Government and regulatory agencies play a critical role in managing pesticide resistance. Effective policies promote responsible pesticide use, ensure product quality, and encourage sustainable alternatives. Key strategies include:

- Strengthening regulatory oversight and product-quality control
- Establishing national resistance-monitoring systems and data-sharing platforms
- Promoting safer vector-control tools
- Enhancing extension services and farmer training
- Providing subsidies and incentives for adopting biopesticides and hermetic storage technologies
- Supporting research and innovation in sustainable agriculture
- Conducting public-awareness campaigns and enforcing food-safety standards
- Implementing and enforcing bans on highly hazardous pesticides

When combined, IPM practices, biopesticides, and strong regulatory frameworks can significantly reduce pesticide resistance, protect biodiversity, and maintain ecosystem integrity while supporting agricultural productivity.

4.0. Conclusion

Comparative Pesticide resistance in Nigeria is a rapidly increasing threat to agriculture, public health, and environmental sustainability. Evidence indicates widespread pesticide misuse, rising residue levels in food and water, and growing resistance among agricultural pests, stored-product beetles, and disease vectors. These trends threaten food safety, reduce productivity, undermine biodiversity, and compromise disease-control efforts. Addressing these challenges requires collaborative action involving regulators, researchers, farmers, and public-health stakeholders. Strengthening surveillance systems, promoting IPM and safer alternatives, enforcing pesticide regulations, and enhancing farmer education are essential strategies for safeguarding human health, environmental quality, and national food security.

5.0. Recommendation

The following recommendations were made:

- The Federal Government should establish a national pesticide-residue and resistance-monitoring program.
- Extension services for farmer training and safe pesticide use should be strengthened.
- IPM and biopesticide adoption should be expanded through subsidies and demonstration farms.
- Regulatory enforcement against illegal imports and counterfeit products should be improved.
- Highly hazardous pesticides (HHPs) should be phased out and replaced with safer ecological alternatives.
- Laboratory capacity for residue detection and toxicological analysis should be enhanced.
- Public awareness on pesticide risks and safe food-handling practices should be increased.
- Cross-sectoral collaboration among agriculture, health, and environmental ministries should be encouraged.
- Government and NGOs should invest more in research on resistance mechanisms, safer alternatives, and socio-economic impacts.

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