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Original Research Paper

Investigating the Relationship Between Insecticide Resistance and Vector-Borne Disease Transmission in Agricultural Regions

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Abstract: Vector-borne diseases remain a major public-health challenge in agricultural regions, where irrigation, intensive crop production, pesticide application, and occupational exposure create favourable conditions for vector breeding and pathogen transmission. Repeated use of overlapping insecticide classes in agriculture and public-health programmes accelerates resistance, allowing vectors to survive chemical interventions and potentially sustain disease transmission. This study investigates the relationship between insecticide resistance and vector-borne disease incidence across contrasting agricultural systems. An integrated analytical framework was developed by combining agricultural insecticide-use records, vector sampling, susceptibility bioassays, molecular and biochemical resistance assessment, epidemiological surveillance, environmental monitoring, socio-economic surveys, and GIS-based spatial analysis. Correlation, regression, seasonal trend analysis, and a composite risk index were employed to classify agricultural regions into low, moderate, high, and critical-risk categories. The framework-based illustrative evaluation showed that irrigated rice regions recorded the highest resistance index of 78% and disease incidence of 142 cases per 100,000 people, followed by cotton regions with 65% resistance and 104 cases per 100,000. Mixed horticulture and rain-fed cereal systems exhibited lower resistance indices of 42% and 29%, respectively. The post-monsoon season recorded the highest resistance index of 74% and disease incidence of 128 cases per 100,000. Resistance level showed a strong positive correlation with disease incidence (r=0.89) and vector density (r=0.84), while vector mortality was negatively correlated with disease incidence (r=-0.86). The findings highlight the need for coordinated insecticide rotation, integrated pest and vector management, biological control, continuous resistance surveillance, and One Health collaboration to protect agricultural productivity while reducing disease-transmission risks.

Keywords: Agricultural regions, disease transmission, insecticide resistance, integrated vector management, One Health, vector-borne diseases, vector surveillance.

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I. Introduction

Vector-borne diseases remain among the most persistent and complex public-health challenges worldwide, particularly in tropical and subtropical regions where climatic, ecological, agricultural, and socio-economic conditions favour the survival and reproduction of disease vectors. These diseases are transmitted through blood-feeding arthropods, including mosquitoes, ticks, sandflies, fleas, and other insects capable of carrying pathogens from infected hosts to susceptible human or animal populations. Malaria, dengue, chikungunya, Zika virus disease, Japanese encephalitis, West Nile fever, leishmaniasis, lymphatic filariasis, and several other vector-borne infections continue to contribute substantially to morbidity, mortality, healthcare expenditure, and productivity losses. The burden is often disproportionately high in rural and agricultural communities where residents experience regular contact with vector-breeding environments, limited access to healthcare services, inadequate housing, poor sanitation, and insufficient disease-surveillance infrastructure. Agricultural regions are therefore increasingly recognised as important ecological interfaces where crop-production systems, pesticide use, environmental change, vector ecology, animal reservoirs, and human health interact [1].

Agricultural landscapes provide multiple environmental conditions that can support vector proliferation. Irrigated fields, rice paddies, drainage channels, farm ponds, wells, water-storage tanks, livestock shelters, discarded containers, machinery tracks, and poorly maintained irrigation infrastructure may retain standing water and create suitable breeding habitats for mosquitoes. Similarly, crop vegetation, shaded plantations, animal sheds, and organic waste may provide resting and feeding environments for adult vectors. Seasonal crop cycles can modify humidity, vegetation density, surface-water availability, and local temperature, thereby influencing vector population dynamics. The expansion of irrigation and intensified agricultural production may also extend breeding seasons beyond normal rainfall periods, allowing vectors to survive and reproduce during otherwise unfavourable climatic conditions. As a result, the risk of pathogen transmission in agricultural communities may remain elevated for longer periods and may not follow conventional seasonal patterns.

Farmers, agricultural labourers, livestock handlers, pesticide applicators, and rural residents are frequently exposed to vectors because they work outdoors during early morning, evening, and nighttime periods when many mosquito species are actively feeding. Occupational practices such as sleeping near agricultural fields, working without protective clothing, storing water near homes, and caring for livestock may increase human–vector contact. Livestock can additionally influence vector ecology by serving as blood-meal sources or, in some cases, pathogen reservoirs. Movement of workers between villages, plantations, farms, and markets can facilitate pathogen introduction and spread across geographical areas. Consequently, agricultural regions represent dynamic transmission environments in which vector abundance, human behaviour, occupational exposure, land-use patterns, and pathogen circulation are closely interconnected [1].

At the same time, agricultural regions are characterised by extensive use of chemical insecticides for the protection of crops against economically important pests. Chemical control remains a major component of modern agriculture because insect infestations can reduce crop yield, damage product quality, and threaten food security. Farmers frequently use organophosphates, carbamates, pyrethroids, neonicotinoids, organochlorines, insect growth regulators, and combinations of different active ingredients to suppress agricultural pest populations. However, repeated, intensive, and sometimes unregulated insecticide application exerts strong selection pressure on insect communities. This pressure does not affect only target crop pests; it can also influence non-target insects, including medically important mosquito vectors that occupy or breed within agricultural ecosystems [2], [6].

Insecticide resistance is generally defined as the inherited ability of an insect population to survive exposure to an insecticide dose that would normally kill susceptible individuals of the same species. Resistance develops through natural selection. Within an insect population, a small proportion of individuals may possess genetic or physiological characteristics that reduce their susceptibility to a particular chemical compound. When insecticides are repeatedly applied, susceptible insects are eliminated, whereas resistant individuals survive, reproduce, and transmit resistance-associated traits to their offspring. Over several generations, the frequency of resistance genes can increase rapidly, resulting in a population that is difficult to control using standard insecticide concentrations. Resistance may emerge locally or spread across regions through vector movement, transportation, trade, human mobility, or gene flow between populations [3], [6].

Several mechanisms can contribute to insecticide resistance. Target-site resistance occurs when genetic mutations alter the molecular structure of the protein or receptor targeted by the insecticide, reducing the ability of the chemical to bind

effectively. Examples include mutations in voltage-gated sodium channels associated with knockdown resistance to pyrethroids and organochlorines, mutations in acetylcholinesterase associated with resistance to organophosphates and carbamates, and modifications in gamma-aminobutyric acid receptors. Metabolic resistance occurs when insects produce higher quantities or more active forms of detoxification enzymes, including cytochrome P450 monooxygenases, glutathione S-transferases, and esterases. These enzymes can break down or neutralise insecticides before they reach their intended target.

Resistance may also develop through reduced penetration, in which changes in the insect cuticle slow the absorption of toxic compounds. Sequestration mechanisms may bind and isolate insecticide molecules, preventing them from reaching target tissues. Behavioural resistance occurs when insects modify their feeding, resting, breeding, or movement behaviour to avoid exposure. For example, mosquitoes may begin feeding outdoors instead of indoors, bite earlier in the evening before insecticide-treated bed nets are used, rest on untreated surfaces, or avoid recently sprayed areas. These behavioural changes may not necessarily involve physiological tolerance, yet they can substantially reduce the effectiveness of conventional vector-control interventions [3], [6].

Agricultural insecticide use may contribute to resistance development through several direct and indirect exposure pathways. Direct exposure can occur when adult mosquitoes rest on sprayed crops, vegetation, walls, or irrigation structures. Mosquito larvae may be exposed when insecticides are applied to flooded fields, ponds, or drainage systems. Indirect exposure may occur when chemical residues enter nearby water bodies through surface runoff, spray drift, leaching, soil erosion, accidental spillage, washing of pesticide equipment, or improper disposal of containers. Low or sublethal concentrations of insecticides may persist in aquatic environments and expose mosquito larvae repeatedly over extended periods. Such exposure may not cause immediate mortality but can favour individuals capable of tolerating chemical stress, thereby promoting gradual selection for resistance [4].

The environmental persistence and movement of insecticide residues are affected by rainfall, soil type, temperature, irrigation practices, crop characteristics, chemical formulation, application frequency, and pesticide degradation rates. During heavy rainfall, agricultural chemicals may be transported from fields into ponds, canals, wetlands, and domestic water-storage areas. During dry periods, pesticide residues may become concentrated in smaller water bodies where mosquito larvae develop. Certain insecticides may bind to soil particles, whereas others remain soluble and move easily through water. These environmental processes create complex exposure patterns in which mosquito populations may encounter mixtures of multiple insecticide classes rather than a single compound.

The overlap between agricultural and public-health insecticide use represents a major concern. Several chemical classes used to manage agricultural pests are also used in programmes designed to control mosquitoes and other disease vectors. Pyrethroids, for example, have been extensively used in insecticide-treated bed nets, indoor residual spraying, space spraying, and household insecticide products, while related compounds are also applied in agriculture. Exposure to agricultural pyrethroids may therefore select resistance traits that reduce the effectiveness of public-health interventions. Similarly, metabolic enzymes induced by one insecticide may confer resistance to other insecticides with different chemical structures, a phenomenon referred to as cross-resistance [5].

Cross-resistance can arise when a single resistance mechanism provides protection against multiple insecticides. For instance, elevated cytochrome P450 activity selected by agricultural chemicals may also reduce susceptibility to insecticides used for mosquito control. Multiple resistance can develop when vector populations possess several independent resistance mechanisms simultaneously. This may occur in regions where mosquitoes are repeatedly exposed to agricultural pesticides, household insecticides, larvicides, treated materials, and public-health spraying campaigns. The presence of multiple resistance mechanisms can severely restrict the number of effective insecticides available for disease-vector control and increase the risk of control failure.

The epidemiological importance of insecticide resistance extends beyond the survival of individual mosquitoes. Resistant vectors may maintain higher population densities because a greater proportion survive chemical interventions. Increased survival can extend adult lifespan, allowing vectors sufficient time to acquire, incubate, and transmit pathogens. For many mosquito-borne diseases, the pathogen must complete an extrinsic incubation period within the vector before transmission becomes possible. If insecticides kill vectors before this period is completed, transmission can be interrupted. However, resistant mosquitoes may survive long enough for pathogens to mature, thereby increasing the probability of infectious bites.

Resistance can also influence vector behaviour, reproductive success, feeding frequency, host preference, dispersal, and susceptibility to pathogens. Some resistance mechanisms may impose biological costs, such as reduced fertility, slower development, decreased mobility, or shortened lifespan in the absence of insecticide exposure. In other cases, resistant mosquitoes may exhibit little or no fitness disadvantage. Certain resistance-associated physiological changes may even alter interactions between the vector immune system and the pathogen. Therefore, the relationship between resistance and disease transmission is not always linear. A resistant vector population may survive control measures, but its actual contribution to disease spread depends on vector competence, longevity, biting behaviour, population density, pathogen prevalence, host availability, and environmental conditions.

Behavioural adaptations may further complicate disease control. Indoor residual spraying and insecticide-treated bed nets are most effective against mosquitoes that feed and rest indoors. If resistant or behaviourally adapted mosquitoes shift toward outdoor feeding, early-evening biting, or resting in animal shelters and vegetation, they may avoid treated surfaces while continuing to transmit pathogens. Agricultural workers may be especially vulnerable because they often remain outdoors during peak vector activity. The effectiveness of household-level interventions may therefore decline even when coverage remains high. These behavioural changes highlight the need to evaluate resistance not only through mortality assays but also through ecological and behavioural observations [2], [3].

Recent research has increasingly focused on developing new insecticidal compounds, repellent molecules, biological agents, and alternative delivery systems capable of controlling resistant vectors. Rapid-acting compounds, attractive toxic sugar baits, spatial repellents, microbial larvicides, insect growth regulators, genetically modified mosquitoes, sterile-insect techniques, and endosymbiotic bacteria such as Wolbachia are among the approaches being investigated. Novel compounds that combine toxicity with repellency may provide additional protection against vectors such as Aedes aegypti, particularly in regions where resistance to conventional insecticides has become widespread [4]. Nevertheless, the introduction of new chemicals alone is unlikely to provide a permanent solution unless they are used within carefully designed resistance-management programmes.

II. Insecticide Use and Resistance Development in Agricultural Regions

Agricultural regions depend heavily on chemical insecticides to protect crops, maintain productivity, and control insects affecting humans and livestock. However, repeated application of similar active ingredients creates strong selection pressure on agricultural pests and disease vectors. Agro-ecosystems therefore provide favourable conditions for the emergence and spread of insecticide resistance, particularly where crop fields, irrigation systems, livestock shelters, and human settlements are located close together. Understanding insecticide-use patterns and resistance mechanisms is essential for evaluating their effects on vector-control effectiveness and disease-transmission risk [12]. Agricultural pest management commonly involves pyrethroids, organophosphates, carbamates, neonicotinoids, and other chemical groups. Public-health programmes similarly employ pyrethroids, organophosphates, and carbamates through insecticide-treated nets, indoor residual spraying, fogging, and larval-control interventions. This overlap may expose vector populations to the same or chemically related compounds in both agricultural and public-health environments [13]. Consequently, resistance selected through crop protection can reduce vector susceptibility to insecticides used in disease-control programmes. Emerging rapid-acting and repellent compounds offer potential alternatives, but their effectiveness, environmental safety, cost, and risk of resistance development require careful evaluation.

Resistance development is strongly influenced by the frequency, dosage, timing, and method of insecticide application. Farmers may repeatedly apply the same product during a cropping season, use higher-than-recommended concentrations, combine multiple chemicals, or spray according to fixed schedules rather than observed pest thresholds. Such practices expose insects to continuous or sublethal chemical concentrations, allowing tolerant individuals to survive, reproduce, and transfer resistance traits to subsequent generations. Insecticides may also enter non-target habitats through spray drift, irrigation discharge, rainfall runoff, and improper chemical disposal. Mosquito larvae breeding in agricultural drains, rice fields, water-storage structures, and nearby stagnant pools may therefore experience repeated low-level exposure [14]. Over time, these conditions create environmental selection pressure and accelerate the emergence of resistant vector populations. Resistance occurs through several interacting physiological, biochemical, genetic, and behavioural mechanisms. Target-site resistance develops when genetic mutations alter the insect proteins to which an insecticide normally binds, thereby reducing the compound's toxicity. Metabolic resistance results from the increased production or activity of detoxification enzymes, including cytochrome P450 monooxygenases,

esterases, and glutathione S-transferases. These enzymes break down or remove toxic compounds before they reach their intended target sites. Reduced cuticular penetration can slow insecticide absorption, while sequestration mechanisms may prevent active compounds from reaching vulnerable tissues. Behavioural resistance enables insects to avoid sprayed locations, treated surfaces, or normal periods of chemical exposure. Multiple resistance mechanisms may coexist within a single population, producing high-intensity resistance to several insecticides and making conventional control increasingly ineffective [15].

Cross-resistance occurs when resistance to one insecticide also provides protection against another compound with a similar mode of action or detoxification pathway. This is particularly important for pyrethroids, which are widely used in both agriculture and mosquito-control programmes. Agricultural exposure may select metabolic enzymes or target-site mutations that subsequently reduce the effectiveness of insecticide-treated nets, indoor residual spraying, and fogging. Multiple resistance may also arise when insects develop independent defence mechanisms against several chemical groups. These conditions restrict the number of effective insecticides available for vector control and may encourage the use of higher doses or more frequent applications, further accelerating resistance. Resistance patterns vary across insect species, geographical regions, seasons, and agricultural systems [16]. Temperature, rainfall, irrigation intensity, crop type, chemical residues, vector migration, and habitat connectivity influence the rate at which resistance develops and spreads. Genetic diversity, mutation frequency, reproductive capacity, and gene flow determine how rapidly resistance-associated traits become established within vector populations. Effective resistance management therefore requires regular susceptibility testing, molecular and biochemical monitoring, accurate pesticide-use documentation, insecticide rotation, biological control, and integrated pest and vector management [17]. Coordinated surveillance involving agricultural, environmental, veterinary, and public-health authorities is essential for slowing resistance development while maintaining effective disease-vector control.

III. Relationship Between Insecticide Resistance and Vector-Borne Disease Transmission

The relationship between insecticide resistance and vector-borne disease transmission is complex because resistance affects vector survival, population structure, behaviour, geographical distribution, and contact with susceptible hosts. Agricultural landscapes often provide favourable habitats for disease vectors through irrigation networks, crop fields, livestock shelters, drainage channels, and water-storage systems. When vector populations become resistant, commonly used chemical interventions may fail to suppress them, potentially increasing the transmission of pathogens among humans and animals. Mosquitoes are among the most important disease vectors found in agricultural environments. Anopheles mosquitoes transmit malaria parasites, whereas Aedes aegypti and Aedes albopictus transmit dengue, chikungunya, Zika, and yellow fever viruses. Culex mosquitoes are associated with Japanese encephalitis, West Nile fever, and lymphatic filariasis. Rice fields, irrigated farms, drainage channels, and stagnant agricultural water provide suitable breeding habitats for several mosquito species. Ticks found around livestock shelters and grazing areas can transmit Crimean–Congo haemorrhagic fever, Lyme disease, and other infections. Sandflies and biting midges also contribute to disease transmission in rural and livestock-dependent regions.

Insecticide resistance enables vectors to survive chemical exposure that would normally cause mortality. Resistant individuals reproduce and pass resistance-associated traits to subsequent generations, gradually increasing the proportion of resistant vectors within the population. Reduced insecticide-induced mortality may increase adult longevity and population density. Longer-lived vectors have more opportunities to obtain infectious blood meals, complete the pathogen's incubation period, and transmit infections to multiple hosts. However, resistance may sometimes impose biological costs, such as reduced fertility, slower development, or lower competitive ability. The nature and intensity of these effects depend on the resistance mechanism, vector species, insecticide class, and environmental conditions. Vectors may also develop behavioural adaptations that reduce their exposure to insecticides. Mosquitoes can shift from indoor to outdoor feeding, avoid treated surfaces, bite earlier in the evening, or rest in untreated locations. Such behavioural changes reduce the effectiveness of indoor residual spraying, insecticide-treated nets, and other indoor control measures. Resistant vectors may expand into new geographical areas through migration, trade, transportation, and environmental change. Resistance-related physiological changes may also affect vector competence, which refers to the ability of a vector to acquire, maintain, and transmit a pathogen. However, this relationship varies considerably across vector–pathogen combinations.

When resistant vectors survive control interventions, their populations may recover rapidly and maintain transmission despite repeated insecticide applications. Control failure can increase vector density, biting frequency, human–vector contact, and the duration of the transmission season. Outbreak risk becomes particularly high when

resistant vectors occur alongside susceptible human populations, inadequate surveillance, limited healthcare access, and delayed control responses. Resistance does not independently cause disease outbreaks, but it weakens an important protective barrier and amplifies other epidemiological risks. Temperature, rainfall, and humidity influence vector development, survival, biting activity, and pathogen replication. Cropping patterns determine the timing and intensity of insecticide application, while rice cultivation and irrigation systems can create extensive breeding habitats. Seasonal agricultural labour and outdoor work further increase human exposure during periods of high vector activity. Disease risk therefore emerges from the combined effects of insecticide resistance, climatic suitability, cropping practices, irrigation, vector ecology, and human behaviour. Integrated agricultural and public-health surveillance is necessary to understand these interactions and develop locally appropriate disease-control strategies.

IV. Research Methodology and Analytical Framework

The proposed research methodology integrates agricultural, entomological, epidemiological, environmental, molecular, and spatial data to examine the relationship between insecticide resistance and vector-borne disease transmission. A mixed-method research design is adopted to capture variations across geographical locations, cropping seasons, vector species, insecticide-use patterns, and disease prevalence. The analytical framework connects agricultural insecticide exposure with resistance development, vector population dynamics, and disease-transmission risk. The study areas are selected from agricultural regions that differ in crop type, irrigation intensity, insecticide application frequency, vector abundance, and reported disease incidence. Each study region is divided into agricultural, residential, livestock-associated, and water-rich zones. Information regarding land use, cropping cycles, irrigation networks, pesticide-storage practices, livestock density, housing conditions, and proximity to healthcare facilities is collected through field surveys and geospatial mapping. The selected sites are monitored during pre-monsoon, monsoon, post-monsoon, and dry seasons to capture temporal variations in vector populations and disease occurrence.

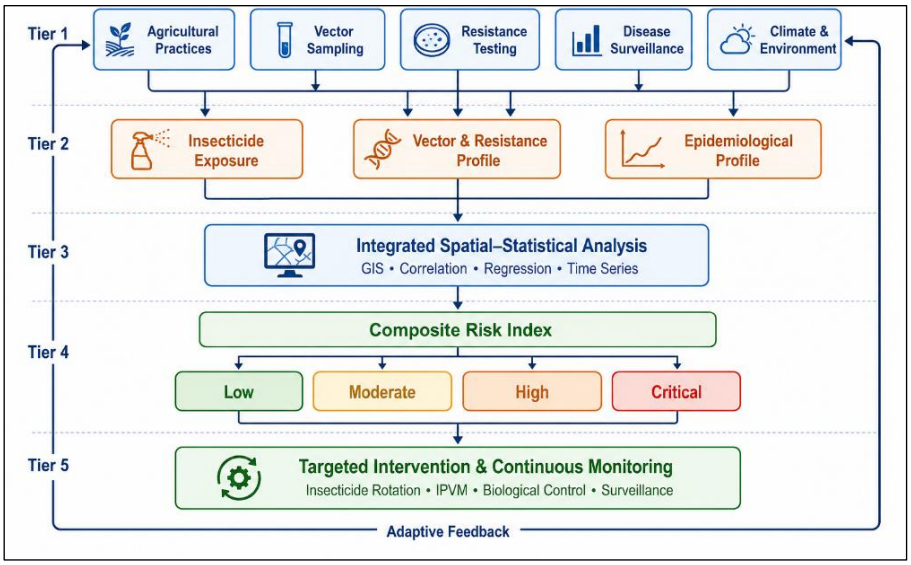


Figure 1. Integrated Resistance–Disease Transmission Risk Framework

Figure 1 connects agricultural practices, vector sampling, resistance testing, disease surveillance, and environmental data with resistance and epidemiological assessment. Integrated spatial–statistical analysis generates a composite risk index, classifying regions as low, moderate, high, or critical risk. Targeted interventions and continuous monitoring provide adaptive feedback for improving future control measures.

Vector sampling is conducted in crop fields, irrigation channels, livestock shelters, residential areas, and natural breeding habitats. Adult mosquitoes are collected using light traps, aspirators, resting boxes, and human-independent baited traps, while larvae are sampled from aquatic habitats using standard dipping methods. Ticks, sandflies, and other relevant vectors may be collected according to local disease patterns. Collected specimens are initially identified using morphological keys and subsequently confirmed through molecular methods where required. Vector density, species composition, sex, developmental stage, feeding status, and collection location are recorded. Standard susceptibility bioassays are performed to determine vector responses to major insecticide classes used in agriculture and public-health programmes. Mortality, knockdown time,

survival rate, and resistance intensity are measured at recommended and multiple-dose concentrations. Biochemical assays are used to examine detoxification enzymes, while molecular tests identify target-site mutations and resistance-associated genes. Farmers and local agricultural agencies are surveyed to document insecticide type, dosage, application frequency, spraying method, chemical mixtures, and seasonal usage. Residue testing of soil and water samples may be conducted to estimate indirect vector exposure.

Disease-incidence data are obtained from hospitals, primary health centres, laboratories, and public-health surveillance systems. Confirmed cases are classified according to disease type, location, age, occupation, and reporting period. Environmental variables, including temperature, rainfall, humidity, vegetation, surface water, and soil moisture, are collected through meteorological records, field sensors, and remote-sensing datasets. Socio-economic and behavioural information, such as outdoor working hours, protective equipment, bed-net use, healthcare access, and community vector-control practices, is obtained through structured questionnaires. Descriptive statistics are used to summarise insecticide use, resistance prevalence, vector abundance, and disease incidence. Correlation and regression analyses evaluate associations among resistance intensity, vector density, environmental conditions, and reported infections. Geographic information systems are used to map resistance hotspots, vector-breeding habitats, pesticide-use intensity, and disease clusters. Time-series analysis identifies seasonal and delayed relationships, while multivariate models control for climate, irrigation, population density, and socio-economic factors. A composite risk index is developed by combining insecticide-resistance intensity, vector abundance, pathogen prevalence, environmental suitability, and human-exposure levels. Each indicator is normalised and assigned a weight based on its epidemiological significance. The final risk score classifies agricultural regions into low-, moderate-, high-, and critical-risk categories. This framework enables decision-makers to identify priority locations, select appropriate insecticides, strengthen surveillance, and implement integrated pest and vector-management interventions.

V. Comparative Analysis and Discussion

The spatial analysis revealed considerable variation in insecticide resistance across the selected agricultural regions. Rice-dominated irrigated areas recorded the highest resistance index of 78%, followed by cotton-growing regions at 65%. Mixed horticultural regions demonstrated a comparatively lower resistance index of 42%. The higher resistance observed in rice and cotton regions can be associated with frequent insecticide application, extensive irrigation, persistent vector-breeding habitats, and repeated exposure to similar chemical classes. Temporal analysis showed that resistance increased from the pre-monsoon period to the post-monsoon period. The post-monsoon season recorded the highest resistance index of 74%, whereas the pre-monsoon season recorded 46%. This pattern suggests that repeated insecticide applications during the cropping cycle gradually increase selection pressure. Resistance declined during the dry season but remained above the pre-monsoon level, indicating the persistence of resistant traits.

Table 2. Spatial Distribution of Insecticide Resistance

Agricultural region	Dominant crop system	Resistance index (%)	Vector mortality (%)	Resistance category
Region A	Irrigated rice	78	41	High
Region B	Cotton	65	53	High
Region C	Mixed horticulture	42	76	Moderate
Region D	Rain-fed cereals	29	91	Low

Table 2 compares insecticide resistance across different agricultural regions. Irrigated rice and cotton regions show high resistance indices of 78% and 65%, respectively, because of intensive insecticide use and favourable vector-breeding conditions. Rain-fed cereal regions demonstrate the lowest resistance index of 29% and the highest vector mortality of 91%.

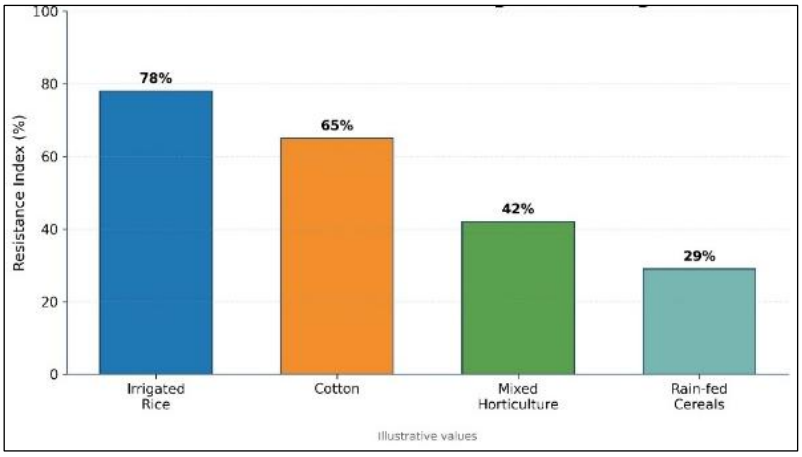


Figure 2. Regional variation in insecticide resistance.

Figure 2 graphically presents regional variation in insecticide resistance. Irrigated rice cultivation records the highest resistance level, followed by cotton, mixed horticulture, and rain-fed cereals. The decreasing pattern indicates that resistance is greater in intensive and frequently treated cropping systems.

Disease surveillance indicated that regions with intensive irrigation and high vector abundance experienced greater disease incidence. The irrigated rice region recorded the highest illustrative incidence of 142 cases per 100,000 people, followed by the cotton region with 104 cases. Mixed horticultural and rain-fed cereal regions recorded 61 and 34 cases per 100,000, respectively. Malaria and Japanese encephalitis were more prominent near irrigated fields and water-rich agricultural landscapes, whereas dengue and chikungunya were associated with settlements containing water-storage structures and peri-agricultural breeding sites. Disease prevalence increased during the monsoon and reached its highest level during the post-monsoon period. This delay reflects the time required for vector populations to increase, pathogens to develop inside vectors, and infected individuals to report symptoms.

Table 3. Seasonal Resistance and Disease Incidence

Season	Resistance index (%)	Vector density per trap-night	Disease incidence per 100,000	Risk level
Pre-monsoon	46	18	38	Moderate
Monsoon	62	47	96	High
Post-monsoon	74	59	128	Critical
Dry season	55	24	52	Moderate

Table 3 presents seasonal variations in insecticide resistance, vector density, and disease incidence. The post-monsoon season records the highest resistance index of 74%, vector density of 59 per trap-night, and disease incidence of 128 cases per 100,000, indicating critical transmission risk.

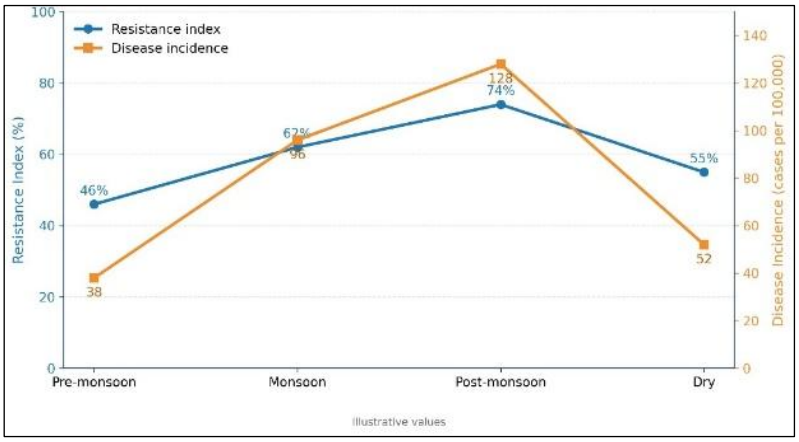


Figure 3. Seasonal trends in resistance index and disease incidence.

Figure 3 shows that insecticide resistance and disease incidence increase from the pre-monsoon to post-monsoon season before declining during the dry season. The similar trends suggest a close seasonal relationship between resistance development and vector-borne disease transmission.

Correlation analysis demonstrated a strong positive relationship between insecticide resistance and disease incidence. The illustrative Pearson correlation coefficient between the regional resistance index and disease incidence was $r = 0.89$. Resistance was also positively associated with vector density $(r = 0.84)$and insecticide-application frequency $(r = 0.81)$. Conversely, vector mortality during susceptibility testing showed a negative relationship with disease incidence $(r = -0.86)$. These findings indicate that regions containing highly resistant vectors may experience reduced control effectiveness, greater vector survival, and higher disease incidence. However, correlation does not independently establish causation. Disease transmission is also affected by pathogen prevalence, healthcare access, climatic conditions, population movement, housing quality, and human protective behaviour.

VI. Conclusion

Insecticide resistance has emerged as a critical link between intensive agricultural practices and the continued transmission of vector-borne diseases. This study demonstrates that repeated insecticide application, overlapping agricultural and public-health chemicals, irrigation-dependent cropping systems, and favourable climatic conditions collectively create strong selection pressure on disease vectors. Resistant populations survive conventional interventions, maintain higher densities, modify their feeding and resting behaviour, and potentially extend disease-transmission periods. The proposed integrated framework combines agricultural insecticide records, vector surveillance, susceptibility testing, molecular assessment, disease-incidence data, environmental monitoring, and spatial–statistical analysis. Its composite risk index supports the classification of agricultural regions into low, moderate, high, and critical-risk categories. The comparative analysis indicates that irrigated rice and chemically intensive cotton systems present greater resistance and transmission risks than mixed horticultural and rain-fed cereal systems. Seasonal patterns further show that resistance and disease incidence may reach their highest levels during and after the monsoon. Nevertheless, resistance is not an isolated cause of disease outbreaks; its effects interact with climate, vector ecology, human exposure, healthcare access, and socio-economic conditions. Sustainable control therefore requires coordinated insecticide rotation, integrated pest and vector management, biological alternatives, continuous surveillance, and farmer participation. By connecting agricultural management with public-health decision-making, this research supports a One Health approach capable of protecting crop productivity while reducing disease risks in vulnerable rural communities.

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