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RVIEW ARTICLE

An Overview of the Insecticide Resistance Status of Vectors Responsible for Transmitting Human Diseases

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ABSTRACT: Insect vector-borne diseases contribute to 17% of the global burden of parasitic and infectious diseases. Currently, more than one billion individuals, primarily in developing nations, are at risk of contracting illnesses such as malaria, filariasis, leishmaniasis, dengue, yellow fever, Japanese encephalitis, Plague, relapsing fevers, and various rickettsial diseases. Insecticides play a critical role in managing the primary vectors of these diseases, including mosquitoes, sandflies, fleas, lice, and triatomine insects. However, the escalating issue of insecticide resistance presents a significant obstacle in the management and control of insect vector-borne diseases. Due to the improper and excessive use of insecticides over time, insect vectors have developed resistance, complicating efforts to manipulate and control them. Consequently, key insect vectors have become resistant to major classes of insecticides. Effective vector control is crucial in reducing vector-borne diseases by targeting vectorial capacity and transmission. The primary method for preventing vectors remains the use of chemical substances in bed nets and indoor residual spraying. Unfortunately, the widespread use of insecticides since the 1950s has led to the global emergence of strong resistance, creating a significant public health challenge in implementing insecticidal vector control. Therefore, we propose to investigate the current level of insecticide resistance in vectors of human diseases and assess its impact on the efficacy of vector control measures.

Keywords: Insecticide Resistance, Vectors, Human Diseases.

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INTRODUCTION

Vector-borne diseases are a significant cause of illness and death, posing major public health challenges in the South Asian region.[1] Bangladesh and other countries in the Indian subcontinent are known to be endemic in malaria, kala-azar (KA), and Dengue. These diseases, transmitted by mosquito and sandfly vectors, have a high incidence in border regions and contribute to cross-border transmission among these countries.[2, 3] In the twenty-first century, the emergence and re-emergence of vector-borne diseases continue to pose a significant threat to human health, resulting in over one million deaths and substantial mortality and morbidity worldwide. Global warming plays a crucial role in creating environmental conditions that directly and indirectly contribute to entomological and epidemiological challenges for disease transmission.[4] It also facilitates the maintenance of the transmission cycle between vectors and human hosts through the resurgence of vectors, increased contact between humans and vectors, and parasite exposure to various strains. Consequently, controlling vector-borne diseases has become an immense challenge, necessitating the development of novel and innovative approaches.[5, 6]

An insect is considered a vector when it transmits pathogens or parasites from one animal, including humans, to another. For example, Anopheles mosquitoes act as vectors for malaria. Some insects, however, are accidental vectors, and their methods of disease transmission are simple and relatively inefficient, such as houseflies.[7] Insecticides are substances or combinations of substances, including microorganisms and viruses, designed to repel, destroy, or control insects, including vectors of human or animal diseases, unwanted plant species, or animals causing harm.[8]

The use of insecticides for vector control in disease control programs continues to play a significant role.[9] These insecticides include ovicides and larvicides, which target insect eggs and larvae respectively. However, it is important to note that the use of insecticides can have a profound impact on ecosystems, as many of them are toxic to humans and animals. Furthermore, some insecticides can accumulate in the food chain.[10] Insecticide campaigns for vector control have primarily focused on mosquitoes, indirectly impacting other insect vectors as well.[11] Unfortunately, the extensive and indiscriminate use of insecticides in the past has led to the development of resistance in malaria, KA, and JE vectors. Consequently, these diseases remain a significant public health concern in South Asian countries.[2]

Insecticide classes

There are various classes of insecticides available for controlling agricultural pests and disease vectors that are medically important to humans and animals.[12] These include organochlorines such as DDT, which was widely used in eradication campaigns during the 1950s.[13] Organophosphates, such as fenitrothion, malathion, and pirimiphos-methyl, are recommended for vector control in indoor residual spraying (IRS) [11]. Carbamates, specifically bendiocarb, are used for IRS vector control as well.[14] Pyrethroids, such as α-cypermethrin, bifenthrin, cyfluthrin, deltamethrin, permethrin, λ-cyhalothrin, and etofenprox, are utilized for both IRS and long-lasting insecticidal nets (LLINs).[15]

The artificial insecticides compound available may be categorized as organochlorine (DDT), organophosphates, carbamates, pyrethroids and miscellaneous insecticides, etc, have been significantly used for the control of both agricultural pests and medically important human/animal disease vectors.[12] Organochlorines are utilized in IRS in the variety of DDT, which was the insecticide used predominantly within the eradication campaigns of the 1950s [13]. Organophosphates comprise a massive range of chemicals; however, those recommended to be used for IRS vector control are fenitrothion, malathion and pirimiphos-methyl.[11] Carbamates are used for IRS vector control within the form of bendiocarb [14]. Pyrethroids are used for both IRS and LLINs within the variety of α-cypermethrin, bifenthrin, cyfluthrin, deltamethrin, permethrin, λ-cyhalothrin and etofenprox.[15] Synthetic pyrethroids have become extremely popular and are extensively utilized in public health because of their comparatively low toxicity to mammals. Nevertheless, their ability to effectively immobilize ('knockdown') and exterminate invertebrates at minimal concentrations has established them as the preferred option.[16] These active ingredients have been extensively employed to combat insect pests such as cockroaches, ants, bedbugs, and mosquitoes.[17] In fact, pyrethroids account for over 80% of the active ingredients currently utilized in households, often in the form of aerosols.

While the widespread use of pyrethroids for vector control has proven effective, it has also raised significant concerns regarding the development of resistance mechanisms induced by these insecticides.[18, 19] The indiscriminate and careless application of pesticides has led to the emergence of resistance. Repeated exposure to excessive and improper use of insecticides has favored the selection of individuals possessing biochemical machinery capable of detoxifying the insecticides more efficiently or exhibiting reduced sensitivity to them.[20] This recurrent and inappropriate use of insecticides is a major contributor to the development of resistance, posing a potential threat to global public health. Therefore, it is crucial to address this issue promptly in order to sustain the current success of vector control, lest it become uncertain.[21]

Insecticide resistance in insect vectors

Insecticide resistance refers to the heritable change in a population's sensitivity to insecticides. This change is evident when the product fails to provide the expected level of control, despite following the guidelines for the specific pest species. Factors such as storage issues, application problems, and unusual environmental conditions can be ruled out.[22] This resistance has led to the evolution of various insect species, including disease vectors, becoming resistant to commonly used insecticides.[23] It is crucial to note that not only mosquitoes, but also other insects of public health importance, such as fleas, ticks, cockroaches, bedbugs, sand flies, and houseflies, have developed resistance due to continuous exposure.[24]

The development of resistance is a complex and dynamic process that depends on multiple factors. Frequent use of the same insecticide selects for individuals in a population that possess genetic advantages, allowing them to survive the recommended dose. Over time, this selective pressure leads to the establishment of a resistant population. In such cases, other compounds within the same chemical class are often affected as well. For example, resistance to one type of pyrethroid typically confers resistance to the entire group, known as cross-resistance. Sometimes, depending on the nature of the resistance mechanism, cross-resistance can occur between different chemical classes. For instance, organophosphates and carbamates may exhibit cross-resistance, as well as DDT and pyrethroids, resulting in multi-resistance.[25, 26, 27] Resistance can also emerge from the excessive or improper use of pesticides against the pest species. This leads to the selection of resistant forms of the pest and the subsequent evolution of populations that are resistant to the pesticide and its mode of action.[28] Over the past four decades, the extensive use of synthetic insecticides to control arthropod pests and disease vectors has resulted in pesticide resistance among more than 450 species.[29] Insecticides have been widely employed to regulate both urban and peri-urban insect vectors and pests.[30] The rapid development of resistance to dichlorodiphenyltrichloroethane (DDT) and organophosphates (OPs) has been well-documented.[31]

Although DDT has been banned in many countries, some African nations have recently allowed its use in malaria control programs due to its superior attributes compared to alternative active ingredients.[32] In 1984, the Insecticide Resistance Action Committee (IRAC) was established to coordinate a private-sector response aimed at preventing or at least delaying the development of resistance. IRAC facilitates communication, raises awareness about insecticide resistance, and promotes the development of resistance management guidelines for sustainable agriculture and improved public health.[24]

The Mechanisms of Insecticide Resistance

A part of insect populations has developed the ability to withstand lethal doses of insecticides that would typically kill most individuals in a normal population of the same species. This resistance is achieved through various mechanisms, including: (i) the ability of resistant mosquitoes to break down or detoxify insecticides at a faster rate than susceptible mosquitoes, resulting in their quick elimination from the body (known as metabolic resistance); the modified activities of three major enzyme groups, such as esterases, oxidases, or glutathione S-transferases (GST), which inhibit the insecticides from reaching their intended target sites; (ii) genetic alterations in the insecticide's target, preventing it from binding and reducing its effectiveness (known as target-site resistance); (iii) the outer layer of resistant insects, known as the cuticle, absorbs the toxin at a slower rate than susceptible insects (known as penetration resistance). Cuticular resistance enhances the effectiveness of other resistance mechanisms; (iv) resistant insects alter their behavior to avoid contact with insecticide-treated surfaces (known as behavioral resistance). For example, the An. arabiensis mosquito has changed its resting behavior from indoors to outdoors in order to avoid exposure to indoor residual spray in Africa.[33, 34]

Tracking the mechanisms of insecticide resistance is crucial for control programs that utilize indoor residual spraying with insecticides.[35] In a review by Hemingway and Ranson, insecticide resistance in major mosquito vectors was reported for DDT, benzene hexachloride/dieldrin, organophosphates, carbamates, and pyrethroid insecticides.[26] Mosquitoes develop resistance to insecticides through enzymatic metabolism or changes in the target sites and proteins that insecticides bind to.[36] Metabolism-based resistance is a mechanism that can lead to resistance against various insecticides, such as organochlorines, organophosphates, carbamates, and pyrethroids.[37] This resistance occurs due to the action of three major enzyme groups. These enzymes can either be overexpressed, allowing them to detoxify the insecticides, or undergo amino acid substitutions that alter their affinity for the insecticide. The enzyme groups responsible for metabolism-based resistance are esterases, glutathione S-transferases, and cytochrome P450 monooxygenases.[38] The increased expression of insecticide resistance genes can be attributed to cis- or trans-acting elements within the promoter or gene amplification. This overexpression leads to a higher production of enzymes in resistant mosquitoes, enabling them to break down the insecticide at a faster rate before it reaches its target site.[39]

Target site resistance, on the other hand, is primarily caused by point mutations in structural genes. These mutations result in amino acid changes that reduce the binding of the insecticide to the target site without affecting its primary function. The target sites for insecticides include acetylcholinesterase, the gamma-aminobutyric acid receptor, and sodium channels in mosquitoes. Modifying these target sites can also confer resistance to organophosphorus, carbamate, organochlorine, and pyrethroid insecticides that act on the nervous system.[40] Mutations in these target sites, particularly in receptors, lead to decreased sensitivity. For instance, PYs and DDT act on voltage-gated sodium channels (VGSCs), and mutations in the amino acid sequence of this gene result in reduced sensitivity of the channels, preventing PYs and DDT from binding. Insects with this mutation can withstand prolonged exposure to an insecticide without being knocked down, hence the term "knockdown resistance" (kdr).[39] An illustration of these mechanisms is represented in Figure 1.

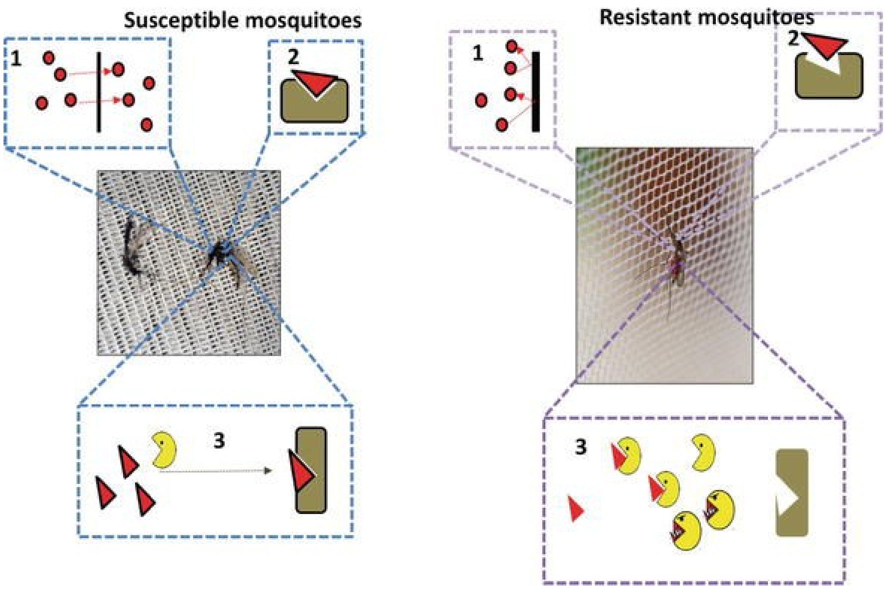

Figure 1: Different Physiological Mechanisms of Insecticide Resistance in Mosquitoes

This illustration shows the different physiological mechanisms of insecticide resistance in mosquitoes. The first mechanism is reduced penetration, which refers to the physiological changes in the mosquito's cuticle that hinder the absorption or penetration of insecticides. The second mechanism is target-site resistance, where the insecticides' effectiveness in binding to a specific site within the mosquito is compromised due to alterations. Lastly, metabolic resistance involves the amplification of enzyme systems that break down insecticides before they can harm the mosquito.

Methods used to examine resistance mechanisms

The study of insecticide resistance is crucial for understanding the true risk and the spread of resistance among disease-carrying insects.[41] Once resistance reaches high levels within a population, traditional strategies to restore susceptibility may no longer be effective. Therefore, regular monitoring is essential. There are three detection techniques that can be used to monitor insecticide resistance, each providing different information.[42]

Susceptibility bioassays

One popular method is susceptibility bioassays, where mosquitoes are exposed to fixed doses of insecticides for a set period, and the percentage of mortality is recorded 24 hours later.[43] This method involves exposing vectors to specific insecticide concentrations and then recording the level of vector mortality. The results are expressed as the percentage of vectors knocked down, alive, or dead. To conduct susceptibility testing, a minimum of one hundred live mosquitoes per testing site is required.[44] These tests are commonly used for routine monitoring and can be performed in the field. They provide standardized data that is easily interpreted. Two types of bioassays, WHO paper bioassays and CDC bottle bioassays, can be used, but the results obtained from these methods are not comparable.[45] To observe longitudinal or temporal patterns in resistance, it is important for countries and academic institutions to consistently use the same method over time.

Biochemical Assays

Another technique is biochemical assays, which detect the presence of specific resistance mechanisms or an increase in enzyme activity. These assays require fresh mosquitoes, but fewer samples are needed compared to bioassays. Unlike bioassays, biochemical assays can identify specific resistance mechanisms and indicate an increase in metabolic enzyme activity. They are often used in conjunction with synergist and molecular assays.[44] By improving our understanding of insecticide resistance through these monitoring techniques, we can develop more effective strategies to combat resistance and protect against disease-carrying insects.

Molecular Assays

Molecular assays are utilized to examine genes at the molecular level, enabling a thorough and direct evaluation of resistance genes. These tests can be conducted using reliable polymerase chain reaction techniques with DNA or more sophisticated microarray tests with RNA. Advanced molecular techniques can provide intricate genetic information, such as determining the specificity or spread of a mutation. These tests are considered the most accurate in measuring the frequency of resistance in vector populations. However, it is crucial to correlate molecular tests with susceptibility testing to ensure accurate results.[46]

Insecticide Resistance in Some Disease Vectors

Current strategies for controlling malaria vectors consist of indoor residual spraying (IRS) with synthetic DDT/pyrethroids and the use of long-lasting insecticidal nets (LLINs).[47] The World Health Organization (WHO) recommends monitoring the susceptibility status of these insecticides on an annual basis.[48] However, over the past two decades, there has been widespread usage of insecticides, particularly pyrethroids, leading to the emergence of resistance and compromising the effectiveness of vector control. It is crucial to gather accurate information about the underlying resistance mechanisms and the intensity or frequency of resistance in malaria vectors in order to update the vector control program and ensure timely management of insecticide resistance.[49, 50] So, effective control of malaria vectors is crucial in the fight against this deadly disease. Resistance to pyrethroids and, to a lesser extent, organophosphates in the primary dengue vector, Ae. aegypti, is widespread. Molecular analysis has identified the primary mechanism of resistance in Aedes spp. Resistance to temephos is associated with increased esterase activity, while resistance to pyrethroid-based insecticides is linked to target-site mutations and elevated expression of cytochrome P450s, specifically the CYP9 family.[19] In regions where the main vector, P. argentipes, is regularly exposed to DDT, resistance to this insecticide has developed. In the main kala-azar-endemic region of India, where P. argentipes resistance is prevalent, alternative pyrethroids such as deltamethrin, alphacypermethrin, lambda-cyhalothrin, and permethrin, which are used for malaria control, may be introduced, at least in highly endemic districts. P. papatasi has also developed resistance, but it does not pose a significant public health problem due to the low prevalence of cutaneous leishmaniasis in the region.[51]

Resistance And Disease Control

The form of insecticide and the application approach vary from country to country and vector to vector, depending on the susceptibility/resistance and behavior of the vector populations. Most insect vectors of human disease develop resistance to specific insecticides. Aedes aegypti has shown strong resistance against Deltamethrin and Fenitrothion [(2)]. For insecticide vector control to be compromised, the level of resistance must be high enough to negatively impact disease transmission. However, in many cases, vector control is not affected by the level of resistance. For example, control measures may be effective against 75% of the vector population. If the level of resistance decreases by 10%, it will not impact disease control efforts. In this situation, increasing surveillance and monitoring levels and frequency of resistance may be sufficient, without the need for changes in control methods.[25] The worldwide increase in insecticide resistance has necessitated the adaptation of vector control measures to a changing environment. As a result, conventional vector control programs that relied on the repeated use of the same class of insecticide for decades have had to shift towards using combinations of insecticides.[52] Several studies have highlighted insecticide resistance in sand fly populations. For example, research in India has revealed resistance of sand fly vectors to dichlorodiphenyltrichloroethane (DDT).[53-55] Countries need to carefully consider the judicious and cautious use of insecticides, considering the potential epidemiological benefits.[56] It is important for vector control programs to diversify from relying solely on pyrethroids to maintain their effectiveness. While pyrethroids may continue to be used for insecticide-treated bed nets in the near term, they should not always be deployed for indoor residual spraying in areas where bed nets are already in use. Integrated resistance management principles and strategies should be implemented to ensure the long-term effectiveness of vector control efforts.[57]

CONCLUSION

Insecticide resistance is a rapidly growing problem that poses a significant challenge to vector control. It is a widespread and evolving phenomenon that demands urgent action to preserve the effectiveness of existing insecticides. Failure to address this issue could have serious consequences. Therefore, it is essential to implement stringent measures to combat resistance and promote the development of effective resistance management strategies. By addressing insecticide resistance, we can ensure the continued efficacy of vector control programs and safeguard public health on a global scale. It is imperative that we take proactive steps to mitigate this problem and prevent its further escalation.

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