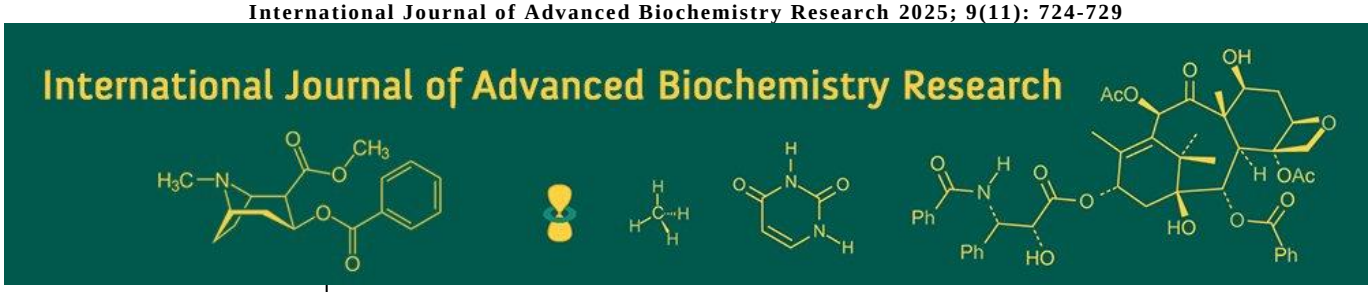

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Acaricide resistance: Status, mechanism of development, methods of detection and its management: A Review

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Abstract
The livestock sector is vital to India's economy, supporting millions of livelihoods. However, vector-borne diseases significantly hinder this growth, causing major health, social, and economic impacts. Ticks and the acaricides used to control them contribute heavily to livestock losses through blood feeding, toxin injection, and transmission of pathogens. Although synthetic acaricides are the main tool for tick control, their indiscriminate and frequent use has led to widespread resistance in field tick populations. This paper outlines the current status of acaricide resistance in ticks, the mode of action of commonly used acaricides, mechanisms and contributing factors behind resistance development, and strategies to manage resistant strains while prolonging the effectiveness of available acaricides.

Keywords: Acaricide resistance, ticks, livestock health, vector-borne diseases, management strategies

Introduction
Ticks and tick-borne diseases pose a major threat to livestock health and productivity, especially in tropical and subtropical regions. They are among the most important blood-sucking ectoparasites, causing heavy economic losses through blood loss, stress, reduced production, immune suppression, and hide damage. Ticks transmit several significant pathogens, including *Theileria*, *Babesia*, *Hepatozoon*, *Ehrlichia*, and *Anaplasma* species. Globally, they are second only to mosquitoes as vectors of human diseases.
Acaricides remain the primary method of tick control, but ticks rapidly develop resistance due to their short life cycle and high reproductive rate. Many chemical classes—organophosphates, carbamates, pyrethroids, amidines, and macrocyclic lactones—have lost effectiveness. Of the 904 tick species known worldwide, 106 occur in India. Crossbreeding in India has altered natural immunity, increasing susceptibility to tick infestation. Hot and humid seasons further favor tick survival and amplify the burden of tick-borne diseases in livestock.

What is resistance?
Drug resistance becomes evident when a treatment no longer controls a parasitic infection, but scientifically it refers to a measurable decrease in the parasite's sensitivity to a drug. As defined by the WHO (1965), resistance is the ability of a parasite strain to survive or multiply even when the host receives recommended or higher doses of a drug. In ticks, acaricide resistance is an inherited trait and is usually recognized when acaricides fail to effectively reduce tick populations.

Different Kind of Acaricide and it's Modes of Action

Primary Site of Action	Class	Examples
ACEh Inhibitors (Nerve action)	Ops Cabametes	Coumaphos, Malathione Carbaryl, Propoxur
Sodium channel modulators (Nerve action)	SPs CED	Delta. Cyper. Flu. Per. DDT, DDE
GABA gated chloride channel antagonists (nerve action)	Cyclodienes Phenylpyrazoles	Chlordane, Aldrin Fipronil, Ethiprole.
Chloride channel activators (nerve & muscle action)	Avermectins Milbemycins	Abamectin, Emamectin Milbectin
Octopamine receptor agonists	Amitraz	Amitraz
nAchR agonist (nerve action)	Neonicotinoids	Imidacloprid Spinosad

Status of acaricides in India and Abroad

Ticks are distributed globally across all continents, including Asia, Australia, the Americas, and Europe. The first report of acaricide resistance in cattle infested with ixodid ticks was recorded in Australia in 1918. Studies show that resistance develops rapidly after the introduction of new acaricides—typically within 4 to 7 years (Sutherst *et al.*, 1979) [22]. For example, coumaphos resistance appeared after 7 years (Aguirre *et al.*, 1986), while amitraz resistance evolved in about 12 years (Foil *et al.*, 2004) [5]. Multiclass resistance is now widespread, especially in countries like Brazil and Mexico, where ticks often show resistance to multiple acaricide groups.

India: Widespread use of chemical acaricides such as organophosphates, pyrethroids, amitraz, and ivermectin. Reports of acaricide resistance in cattle ticks (e. g., *Rhipicephalus microplus*) across different climatic zones. Indiscriminate and frequent application has contributed to resistance buildup, reducing efficacy. Regulatory oversight exists, but awareness and proper usage among farmers remain inconsistent.

Abroad: Countries like Australia, Brazil, and Mexico face similar issues with multi-acaricide resistant ticks. Integrated approaches, including vaccination (e. g., TickGARD), resistant cattle breeds, pasture management, and rotational acaricide use, are more widely implemented. Advanced diagnostic tools (bioassays, molecular testing) are used for early detection of resistance. Research is ongoing into botanical acaricides, biological control, and novel vaccines to reduce chemical dependency.

Reports of acaricide resistance in India.

The first report of Benzene Hexachloride (BHC) resistance in the *R.* (*B.*) *microplus* population infesting cattle was made by Chaudhari & Naithani in 1964. Pesticide resistance to carbaryl and broad-spectrum carbamates was described by Basu and Haldar in 1997 [4]. The most recent reports on acaricide resistance are included below.

The most recent reports on acaricide resistance are included below.

Drug	Reference	State
Diazinone	Kumar *et al.*, 2011 [13]	Uttar Pradesh
Deltamethrin, cypermethrin and Diazinone	Shyma *et al.*, 2012 [18]	Uttar Pradesh
Amitraz	Kumar *et al.*, 2014 [14]	Uttar Pradesh
Malathion, Coumaphos, Fenvalerate and Fipronil	Kumar *et al.*, 2014[14]	Uttar Pradesh
Deltamethrin, cypermethrin and Diazinone	Ghosh *et al.*, 2015 [10]	Uttar Pradesh
Cypermethrin	Ghosh *et al.*, 2017 [9]	Uttar Pradesh
Ivermectin	Nandi *et al.*, 2018 [17]	Uttar Pradesh
Deltamethrin and Cypermethrin	Upadhyay *et al.*, 2020	Uttar Pradesh
Deltamethrin	Jyothimol *et al.*, 2014 [12]	Kerala
Deltamethrin	Ahanger *et al.*, 2015 [1]	Jammu & Kashmir
Ivermectine	Singh *et al.*, 2015 [19, 20]	Punjab
Deltamethrin and Diazinone	Gaur *et al.*, 2016 [7]	Haryana and Rajsthan
Deltamethrin, Cypermethrin and Amitraz	Kumar *et al.*, 2017 [15]	Andhra Pradesh
Deltamethrin and Chlorpyriphos	Kutari *et al.*, 2017	Madhya Pradesh
Deltamethrin and Cypermethrin	Godara *et al.*, 2019 [1]	Jammu
Amitraz	Singh *et al.*, 2015 [19, 20]	SKnagar, Gujarat
Deltamethrin, Flumethrin and Fipronil	Shyma *et al.*, 2016 [18]	SKnagar, Gujarat
Deltamethrin, cypermethrin and Ivermectin	Kumar *et al.*, 2017 [15]	Junagadh, Gujarat
Deltamethrin and Cypermethrin	Sharma *et al.*, 2018	S Knagar, Gujarat

Resistance Mechanism for Various Acaricides

Organophosphates (OPs): OP resistance in ticks develops through three main mechanisms: Metabolic detoxification: Increased esterase activity and elevated production of acetylcholinesterase (AChE) help detoxify OP compounds, reducing their effectiveness. Carboxylesterase gene mutation: A mutation in the carboxylesterase (CaE) gene has been associated with OP resistance, playing a key role in reducing OP susceptibility (Baffi *et al.*, 2007) [2]. Acetylcholinesterase gene mutation: Changes in the AChE gene alter the target site of OPs, preventing the drug from inhibiting the enzyme effectively, thereby conferring resistance (Fournier & Mutero, 1994) [6].

Organochlorines act by binding to the picrotoxinin site of the GABA-gated chloride channel, blocking Cl^- entry and causing nerve hyperexcitation leading to death. Widespread resistance has developed against lindane, dieldrin, and DDT due to both target-site and metabolic mechanisms. In Rhipicephalus microplus, resistance to dieldrin and DDT is linked to mutations in the GABA-gated chloride channel and voltage-gated sodium channel genes, causing key amino acid changes (Bandara & Karunaratne, 2017) [3].

Synthetic pyrethroids (SPs), widely used since the 1970s, have led to resistance in *R. microplus* through target-site mutations and enhanced metabolic detoxification. A key mechanism is a mutation in the voltage-gated sodium channel gene (T2134A), causing the F712I substitution linked to high SP resistance. Additionally, overexpression of esterases—especially carboxylesterase (CaEs) such as CzEst9—contributes to metabolic detoxification of pyrethroids. Together, these mechanisms make SP resistance a major challenge in tick control.

Amitraz, a formamidine used widely against livestock ticks, has developed resistance in R. microplus across many regions, likely through recessive and multigenic mechanisms. Resistance is associated with enhanced metabolic detoxification involving P450 monooxygenases, monoamine oxidase, and increased ABC transporter activity.

Macrocyclic lactones (MLs), including ivermectin and milbemycins, act by activating glutamate-gated and GABA-gated chloride channels, causing paralysis and death in ticks. Ivermectin is widely used as an alternative acaricide, but R. microplus resistance has emerged in countries such as

Brazil, Mexico, Uruguay, and India. The molecular mechanisms of ML resistance remain poorly understood.
Phenylpyrazoles such as fipronil act by blocking GABA-gated chloride channels in the insect nervous system. Introduced in the mid-1990s for livestock, fipronil is used to control cattle ticks and horn flies. Resistance in R. microplus has been reported in Uruguay, Brazil, and Mexico, though the exact molecular mechanism of fipronil resistance in this tick species is still unknown.
Carbamates are carbamic acid esters that act similarly to organophosphates but are generally less harmful to mammals. Their primary mode of action is the inhibition of acetylcholinesterase, leading to disrupted nerve function (Spickett, 1998) [21].

Mechanism of resistance: Reduced cuticular penetration occurs when changes in the tick's outer cuticle limit the entry of acaricides, reducing their effectiveness. Metabolic detoxification involves three phases: Phase I enzymes (like P450 monooxygenases and carboxylesterases) modify and activate toxic compounds; Phase II enzymes (such as GSTs and UDP-glycosyltransferases) conjugate them; and Phase III transports these conjugates out of the cell. Increased activity or overproduction of detoxifying enzymes—esterases, GSTs, and P450s—leads to metabolic resistance. Synergists like piperonyl butoxide (PBO), triphenyl phosphate (TPP), and diethyl maleate (DEM) help identify which enzyme groups are involved in resistance.

Target Site Insesitivity: Target-site resistance occurs when a single nucleotide mutation alters an amino acid in the target gene, reducing the tick's susceptibility to an acaricide. Resistant genes often exist at low levels before a new acaricide is introduced, and their spread depends on factors like mutation frequency, inheritance pattern, frequency of treatment, acaricide concentration, and the proportion of ticks not exposed (refugia). Reduced target-site sensitivity can make the acaricide dose ineffective, while other mechanisms—reduced penetration, sequestration, or detoxification—further lower drug efficacy. Understanding resistance mechanisms is essential to: (1) develop rapid diagnostic tools for early detection; (2) select alternative chemicals and avoid cross-resistance; (3) combine acaricides with suitable synergists to extend their useful life; and (4) identify new molecular targets and support the development of novel acaricides.

Behavioural/Physiological Change: Arthropods may simply stop feeding if they come across certain insecticides, or leave the area where spraying occurred.

Evolution of Resistance to Acaricide?
a) Genetic: Parasite genetic factors influencing acaricide resistance include dominance of resistance alleles, number of genes involved, initial gene frequency, population genetic diversity, fitness of resistant organisms, and potential for recombination. Resistance develops in three stages: establishment, development, and emergence. Resistant genes often exist at very low levels before acaricide use and increase under continuous selection pressure. Factors affecting the speed of resistance spread include inheritance pattern, treatment frequency, acaricide concentration, and the proportion of ticks in refugia. Initially, heterozygous resistant ticks are few, but with ongoing exposure, homozygous resistant ticks eventually dominate. Genetic factors are difficult to control as they are intrinsic to the parasite.

b) Biological: Biological factors include biotic traits (breeding patterns, offspring per generation, generation time) and behavioral traits affecting gene flow and selection, such as refugia, survival, mobility, migration, and host range. Host-parasite interactions influence selection for resistance, as parasites triggering strong host immunity face weaker drug selection. Pathogenicity affects treatment frequency, and larger refugia populations slow the development of resistance.

(3) Operational Chemical: Operational factors influencing acaricide resistance include the chemical properties, formulation, application method, life stage targeted, treatment frequency, and persistence of the drug. Frequent or improper use, underdosing, and reliance on a single chemical group increase resistance risk. Sub-therapeutic exposure, long drug half-life, and ineffective regimens select for resistant ticks. Proper management and farmer education can mitigate these operational risks.

Types of resistance: Acquired resistance occurs when heritable decreases in drug sensitivity develop over time due to selection of resistant ticks. Cross-resistance happens when resistance to one acaricide confers resistance to others with a similar mode of action, such as organophosphates and carbamates targeting acetylcholinesterase. Multiple resistance is resistance to several acaricides with different modes of action, seen in *R. microplus* in Mexico, often due to target site mutations and sometimes metabolic mechanisms.

Phase of Resistance
Development of resistance occurs in three phases: Establishment: Resistance alleles exist naturally in the population, independent of chemical use.

Development: Resistant ticks survive chemical treatment, increasing their numbers. Selection is rapid if the allele is dominant and slow if recessive.

Emergence: Resistance alleles become widespread, reducing acaricide efficacy. Diagnosis: Resistance is suspected when treatments fail repeatedly. Detection uses bioassays, molecular tests, and biochemical methods, and judicious acaricide use can slow resistance development.

(a) Bioassay Tools: Bioassays are key tools for detecting resistant tick populations due to their simplicity and low cost, providing phenotypic data on resistance levels. Four main bioassays exist: Adult Immersion Test (AIT), Larval Packet Test (LPT), Larval Immersion Microassay (LIM), and Larval Tarsal Test (LTT). AIT gives results in 7 days but requires many engorged females, while LPT and LIM take 5-6 weeks using larvae. Bioassays detect resistance even when mechanisms are unknown but do not clarify how resistance occurs. Advantages: Simple, inexpensive, and provides phenotypic resistance data. Disadvantages: Requires sufficient engorged females, time-consuming, and offers limited insight into resistance mechanisms.

Biochemical Tool

Metabolic Resistance: The most common mechanism of acaricide resistance is metabolic, where detoxifying enzymes like esterases, monooxygenases, and GST break down or sequester multiple acaricide classes. Biochemical tests using enzyme inhibitors (e. g., chlorfenethol, EPE-oxon, DEF, piperonyl butoxide) help detect resistance patterns. Methods include filter paper assays and microtitre plate tests, providing phenotypic evidence of enzyme-mediated resistance.

Advantages: Broad detoxification ability, shows metabolic resistance patterns, effective against multiple acaricide classes.

Disadvantages: Requires many samples, early resistance may go undetected, no genetic information revealed.

Molecular Tools

Molecular Tests for Acaricide Resistance: Molecular assays are faster than bioassays, providing results in a day, requiring fewer ticks, and detecting resistance mechanisms early. Techniques include allele-specific PCR, gene amplification, DNA/RNA sequencing, transcriptomics, and qPCR to identify resistance genes and SNP markers. They help monitor low-frequency or recessive resistance alleles and guide effective acaricide use. Advantages: Rapid results, early detection, highly sensitive, enables informed control strategies. Disadvantages: Requires precise molecular markers, skilled personnel, and costly equipment.

Management of Acaricide Resistance

Integrated Tick Management Strategies

Rotational Use of Acaricides

Regular Monitoring: Limit treatments to ≤5 per season; monitor tick species and resistance levels.

Acaricide Rotation: Alternating chemicals with different modes of action reduces resistance development; recommended every ≥2 years.

Combination Use: Using two compatible acaricides with different mechanisms can delay resistance and improve efficacy.

Vaccination: Anti-tick vaccines (e. g., TickGARD, BM86-based) induce host antibodies to reject ticks. Advantages: Long-term protection, independent of environment. Disadvantages: Variable efficacy by region, short-lived immunity (every 3 months), often used with acaricides, cost issues.

Nutritional Management: High-protein diets improve T-cell function and resistance to ticks. Benefits include reduced tick load and better host health. Limitations: Cost, labor, and storage requirements.

Botanicals: Plants with acaricidal properties (e. g., Ocimum, Eucalyptus, Citrus oils) can kill or repel ticks. Limitations: Often act as deterrents, low systemic effect, availability, and potential toxicity to non-target species.

Genetic Resistance in Cattle: Zebu cattle (*B. indicus*) and hybrids are naturally tick-resistant, reducing acaricide use. Limitations: Expensive, breeding challenges, potential ecological impact, and lower survival in some cases.

Environmental Management: Pasture Burning: Reduces tick populations but requires monitoring and may be resource-intensive. Pasture Rotation/Alternation: Grazing management can reduce larvae survival. House Management: Maintain low animal density, cleanliness, and separate species to reduce infestation.

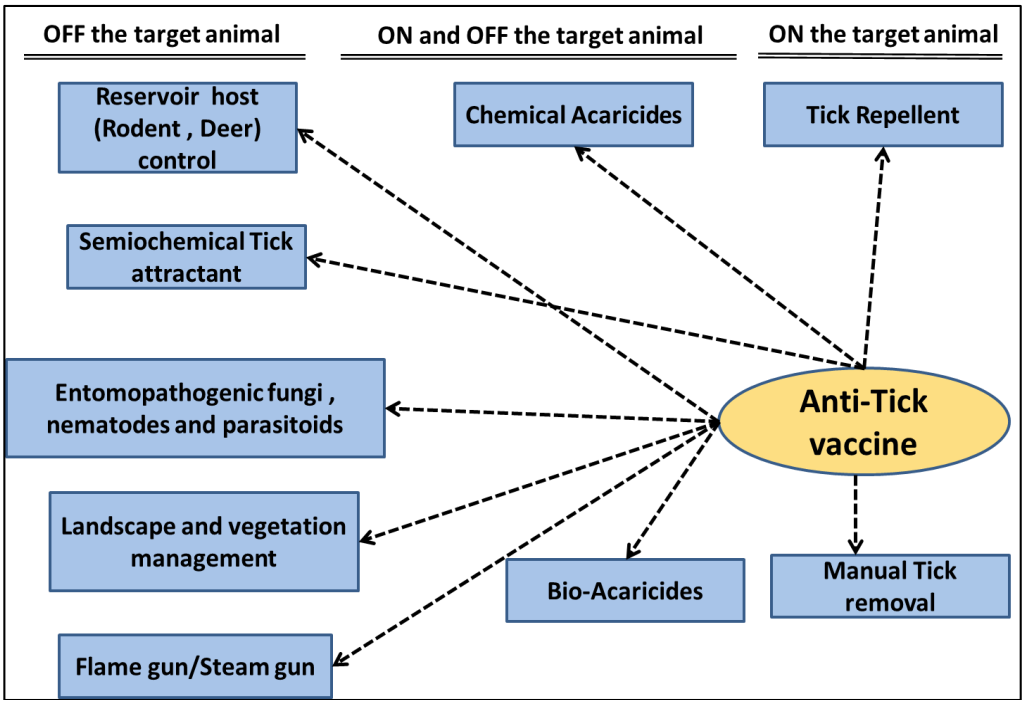
Improving Resistance Diagnostics: Bioassays: LPT, LIT, AIT-inexpensive but slow and less sensitive to early resistance. Molecular Tests: PCR-based assays detect resistance genes quickly and guide effective acaricide use.

Biological Control: Parasitoids: Some Hymenoptera species (e. g., I. hookeri) can target ticks. Fungi: Entomopathogenic fungi attack ticks but require high humidity and careful application. Nematodes: Steinernematid and Heterorhabditid nematodes infect and kill engorged females. Sterile Male Technique: Irradiated males reduce reproduction; further study needed. Birds: Certain bird species consume ticks, e. g., chickens can reduce tick numbers in close quarters.

Integrated Tick Management

There are numerous prevention and control techniques, including those that are taken both "on the target animal" and "off the target animal," that can be used to lessen the tick infestation on animals. The on-host precautions include manual tick removal, anti-tick vaccination, and the application of repellents. Rodent and deer control, the use of semiochemicals and kairomones as attractants, the use of entomopathogenic fungi, nematodes, and parasitoids, management of the landscape and vegetation, and the use of flame guns and steam guns are some examples of off-host measures. Some tick-control strategies, like chemical and biological acaricides, can be applied on and off the host. A smart and long-term solution to control cow ticks is to raise genetically chosen tick resistant cattle breeds. Chemical acaricides have long been the primary means of managing ticks on cattle or in the environment, but their indiscriminate use has resulted in the selection of acaricide-resistant ticks, making the majority of chemical acaricide classes useless at doing so.

Additionally, the risks to the environment and human health make widespread application of chemical acaricides problematic. The management of tick infection in cattle using all available on and off host interventions failed. The complexity of the tick life cycle, genetic variety of ticks, reservoir hosts, host genetics and immunity, and their intricate interactions cannot be adequately addressed by single intervention measures due to their short duration or efficacy. Though the main factors influencing farmers and veterinarians to choose the intervention measures are market accessibility and ease of use, product shelf life, cost, and the length of time to see the visible effect on host, there is an increased interest among cattle farmers in implementing integrated tick control measures.



Conclusions

Tick Resistance and Control: Acaricide resistance is inevitable with long-term use. In India, resistance to deltamethrin, cypermethrin, amitraz, and ivermectin has been reported across different climates. Irrational, continuous use can lead to permanent resistance. Diagnostic tests like ALT, AIT, and LPT help identify and manage resistance. Due to the limitations of chemical acaricides, alternative methods are recommended: breeding tick-resistant cattle, pasture spelling and burning, planting special grasses, using entomopathogenic fungi, and plant-based acaricides for eggs, larvae, and adults. Integrated Tick Management (ITM) combines multiple strategies considering ecological, economic, and social factors to reduce tick infestations below economic injury thresholds.

Future Prospect: Tick Control Strategies: Effective tick vaccines can reduce chemical use and resistance. Breeding resistant cattle, regular monitoring, acaricide rotation, and combination treatments help manage resistance. Herbal acaricides and vaccines are promising future strategies, with botanical acaricides offering potential for exploration.

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