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Suspected bromadiolone resistance in *Bandicota bengalensis* (Gray & Hardwicke) from Andhra Pradesh, India: VKORC1 analysis

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The lesser bandicoot rat, *Bandicota bengalensis* (Gray & Hardwicke) is a major rodent pest affecting agriculture and public health in India. Bromadiolone, a second-generation anticoagulant rodenticide, is extensively used for community rodent control in rice-based ecosystems of the Godavari Delta, raising concerns over resistance development. This study assessed bromadiolone resistance in *B. bengalensis* populations from six villages in the Godavari districts of Andhra Pradesh using feeding mortality tests, blood clotting response assays, and VKORC1 gene sequencing. Of 152 individuals, 23.7% showed delayed mortality and prolonged clotting times, indicating emerging tolerance. Sequencing of suspected resistant rats did not reveal known VKORC1 resistance mutations, although *B. bengalensis* specific variants with high homology to *Rattus norvegicus* sequences were detected. The absence of classical VKORC1 polymorphisms suggests alternative biochemical mechanisms, such as cytochrome P450 mediated detoxification, may reduce susceptibility. This study provides the first experimental evidence of bromadiolone resistance in *B. bengalensis* in India, underscoring the need for regular resistance monitoring and integrated rodent management to prevent control failures in agricultural landscapes.

Keywords: *Lesser bandicoot rat*, Blood clotting response, Rice, Integrated rodent management

Rodent pests are a persistent constraint to rice production and food security across Asia[1]. In India, the lesser bandicoot rat, *Bandicota bengalensis* (Gray & Hardwicke), is recognized as the most destructive field rodent in rice ecosystems[2]. The species causes substantial pre- and post-harvest yield losses, commonly ranging from 5-30% and exceeding 60% during severe outbreaks, along with significant contamination of stored grain and increased risks of zoonotic pathogen transmission, including leptospirosis[3] and hantavirus[4]. The Godavari Delta of Andhra Pradesh represents an intensively cultivated rice landscape where infestations of *B. bengalensis* are endemic. Multiple cropping cycles, continuous food supply, and highly suitable burrowing habitats enable year-round population growth and reinfestation, making rodent management extremely challenging[5].

Anticoagulant rodenticides remain the primary tool for large-scale community rodent control in such rice ecosystems. Bromadiolone, a second-generation anticoagulant used widely in India since 1988, is applied extensively through coordinated campaigns that deploy large quantities (1500-2000 kg annually) across approximately 3.5 lakh hectares of paddy fields in the Godavari Delta to suppress *B. bengalensis* populations[6]. However, recent field observations in these regions indicate delayed mortality, rapid population rebound, and frequent control failure following bromadiolone treatments. These outcomes raise concerns about emerging tolerance or resistance under prolonged and repeated selection pressure.

International studies have established that resistance to anticoagulant rodenticides is often driven by mutations in the Vitamin K epoxide reductase complex subunit 1 (VKORC1) gene, which impair rodenticide binding affinity and thereby reduce toxic efficacy[7-9]. Additionally, enhanced metabolic detoxification, particularly via cytochrome P450–mediated hepatic biotransformation, has been implicated in non-VKORC1 mechanisms of resistance[10]. Resistance associated with VKORC1 exon mutations has already been documented in *Rattus norvegicus* and

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Mus musculus, conferring reduced susceptibility to second-generation anticoagulant rodenticides including bromadiolone[11-14]. Despite these global reports, the molecular determinants of anticoagulant susceptibility in *B. bengalensis* populations in India remain poorly characterized, highlighting the urgent need for comprehensive genomic and toxicological investigations to inform resistance-management strategies.

In this context, the present study investigates bromadiolone resistance in *B. bengalensis* populations from the Godavari Delta by employing standardized feeding trials, blood-clotting response assays, and VKORC1 gene sequencing. The outcomes provide essential baseline information for detecting early resistance development in field populations and contribute to the refinement of integrated rodent management strategies in rice ecosystems. Furthermore, the findings offer critical guidance for policy interventions aimed at rational rodenticide use and the establishment of robust resistance surveillance frameworks to ensure long-term protection of India's rice-based food security.

Material and Methods

The study was conducted at the Vertebrate Pest Management Division and Plant Biotechnology Laboratory, Regional Agricultural Research Station (RARS), Maruteru, West Godavari, Andhra Pradesh, India (latitude: 16.34°N; longitude: 81.23°E).

Study area and Animal Collection

Field locations were selected through farmer consultation, focusing on areas with prolonged bromadiolone use and reported difficulties in rodent control (indicated by rapid post-treatment resurgence). A total of 152 lesser bandicoot rats (92 males, 60 females; mean weight 195.8 ± 18.6 g), were captured from six villages in East and West Godavari districts using multi-catch traps baited with coconut pieces between January and March 2023 (Table 1). No rodenticides had been applied in the rodent collection fields during the 30 days prior to trapping. Immediately after capture, rats were transported to the laboratory, sexed, weighed, and housed individually in PVC cages (60×30×30 cm) under controlled ambient conditions (28 ± 2°C; RH 65 ± 5%) with a 12-hour light-dark cycle. All animal care and handling procedures were approved by the Institutional Animal Ethics Committee (IAEC), RARS Maruteru, ANGRAU (Approval No: IAEC/VPM/2023-02, dated March 15, 2023).

Chemicals and Bait Preparation

Bromadiolone (0.25% w/w concentrate) was procured from M/s Ultima Search Pvt. Ltd., Gujarat, India. A standard 0.005% bromadiolone bait was prepared by thoroughly mixing 20 g of bromadiolone concentrate with 940 g of broken rice, 20 g of powdered sugar, and 20 ml of groundnut oil. The ingredients were mechanically homogenized to ensure uniform distribution of the active ingredient throughout the bait matrix.

Feeding trial protocol

To establish baseline feeding behaviour, rats were provided *ad libitum* RSO bait for five days prior to treatment. On Day 6, each rat received a precisely calculated LD_{50} dose of bromadiolone (1.2 mg/kg body weight, determined from preliminary toxicity assays). The bait was consumed completely within 2–4 h, confirmed by direct visual monitoring, after which plain RSO bait was reinstated. Mortality and time-to-death were recorded for 30 days post-exposure. Separate control groups (n=20 per location; not included among the 152 test rats) remained on plain RSO bait throughout the observation period to assess natural mortality.

Blood clotting response (BCR) test

Blood samples (1 ml) were collected from each rat at two time points: pre-treatment baseline (0 h) and post-treatment (48 h after LD_{50} exposure). Blood was drawn from the retro-orbital plexus using heparinized microcapillary tubes and transferred into tubes containing 0.1 ml of 3.2% trisodium citrate as an anticoagulant. The tubes were gently inverted to ensure proper mixing and subsequently centrifuged at

Table 1 — Rats collected from different farmer fields of districts East and West Godavari, Andhra Pradesh, India

S. No	Location	Total No. of rats trapped	Male	Female
1	Maruteru, Penumantra (M), West Godavari, A.P	42	25	17
2	Bhimavaram (V&M), West Godavari, A.P	18	11	7
3	Tadepalligudem (V&M), West Godavari, A.P	22	14	8
4	Jonnada (V), Ravulapalem (M), East Godavari, A.P	26	17	9
5	Ramachandrapuram, East Godavari, A.P	16	10	6
6	Ambajipeta (V), Amalapuram (M), East Godavari, A.P	28	15	13

3000 × g for 15 min. Separated plasma samples were analyzed within 2 h of collection. Coagulation parameters, namely Prothrombin Time (PT), International Normalized Ratio (INR), Prothrombin Ratio (PR), and Activated Partial Thromboplastin Time (APTT), were measured using a semi-automated coagulation analyzer (Robonik Smart III), with rabbit brain thromboplastin reagents (WAKO Diagnostics)[15]. Percentage of coagulation Activity (PCA) was calculated based on PT values generated in the laboratory using the following formula:PCA (%) =100 × (Control PT/ Sample PT). Where, control PT represents the mean PT of untreated control rats maintained on plain RSO bait, and sample PT represents the PT measured from each experimental rat 48 h after LD_{50} bromadiolone feeding. This parameter quantified the residual functional activity of vitamin K-dependent clotting factors. Numerical PCA values obtained in this study are presented in Table 2, derived directly from experimentally measured PT values[16].

Resistance interpretation was based on anticoagulant susceptibility thresholds established in rodent toxicology literature. Individuals exhibiting PT < 70 s and INR < 4.0were classified as resistant, while those with PT ≥ 70 s and/or INR ≥ 4.0 were considered susceptible to bromadiolone. Both extrinsic (PT, PR, INR) and intrinsic (APTT) coagulation pathways were assessed to evaluate the extent of impairment in vitamin K–dependent hemostasis following anticoagulant exposure, ensuring a robust classification of resistance phenotypes.

Molecular analysis of VKORC1 gene

DNA isolation

Tail tip tissue (~0.5 cm) was collected from both resistant and susceptible rats immediately after death by treatment and preserved in 70% ethanol at -20 °C until analysis. Genomic DNA was extracted using the Proteinase K method[17].

VKORC1gene amplification

The VKORC1 gene was amplified from genomic DNA of both resistant and susceptible individuals. Three sets of forward and reverse primers were designed from the *R. norvegicus* VKORC1 sequence (GenBank Accession ID: LC218154.1) using NCBI Primer-BLAST (Table 3). Each primer set targeted

Table. 2 — Mean days to death and blood coagulation parameters of suspected to be resistant and susceptible rats collected from different farmer fields and fed on LD50 dose of 0.005% bromadiolone

Location	Treated rats suspected to be resistant/susceptible	Changes in blood clotting parameters after 48 h of treatment						Mean days to death (range)
		Mean body weight (g)	PT (s)	INR	R	APTT (s)	PCA (%)	
Maruteru, Penumantra (M), West Godavari, A.P	Resistant (n = 8)	191.81± 3.41	42.86±2.71	2.42±0.15	2.36±0.15	42.59±3.52[a*]	105.0	11.13±7.90 (5-30)
	Susceptible (n = 34)	210.89±21.34	162.97±20.84[a]	9.49±3.51[a]	7.98±1.10[a]	38.27±6.56	27.6	5.18±0.58 (4-6)
Bhimavaram, (V&M), West Godavari, A.P	Resistant (n = 6)	183.07±13.38	40.98±4.41	3.48±0.18	2.65±0.40	44.35±7.17[a*]	109.8	7.50±1.87[a*] (5-10)
	Susceptible (n = 12)	193.98±16.83	195.98±12.58[a]	11.66±2.64[a]	6.57±0.51[a]	34.29±6.44	23.0	4.67±0.89 (4-6)
Thadepalligudem, (V&M), West Godavari, A.P	Resistant (n = 6)	196.40±18.58	42.65±10.71	3.52±0.32	2.06±0.54	43.68±7.07[a*]	105.5	7.67±1.86[a*] (5-10)
	Susceptible (n = 10)	194.95±13.04	177.11±14.78[a]	12.12±2.67[a]	6.32±2.16[a]	33.95±4.14	25.4	4.40±0.52 (4-5)
Jonnada (V), Ravulapalem (M), East Godavari, A.P	Resistant (n = 7)	192.93±13.80	46.34±5.66	3.48±0.29	2.21±0.44	40.34±3.52	97.1	8.00±1.53[a*](6-10)
	Susceptible (n = 15)	202.12±10.29	151.29±16.77[a]	9.88±2.01[a]	6.44±1.41[a]	37.81±4.83	29.7	5.07±1.03 (4-7)
Ramachandhrapuram, East Godavari, A.P	Resistant (n = 5)	179.97±13.88	38.88±7.39	2.56±0.21	2.05±0.54	43.32±2.51[a*]	115.7	9.60±4.39 (6-17)
	Susceptible (n = 21)	182.65±11.24	159.99±15.76[a]	8.76±2.57[a]	6.22±2.20[a]	36.33±4.07	28.1	4.52±0.60 (4-6)
Ambajipeta (V), Amalapuram (M), East Godavari, A.P	Resistant (n = 4)	190.25±12.95	46.50±7.47	3.59±0.27	2.42±0.56	39.35±3.37	96.8	7.25±1.71 (5-9)
	Susceptible (n = 24)	201.81±8.59	156.03±14.33[a]	9.15±2.69[a]	5.76±1.18[a]	35.35±5.41	28.8	5.17±0.82(4-7)

Values are mean ± SD, n = No. of rats. PT= = Prothrombin Time, INR = International Normalized Ratio, R = Prothrombin Ratio, APTT = Activated Partial Thromboplastin Time and PCA= Percentage Coagulation Activity.

a-Significantly high values of PT, INR and R in susceptible rats after 48 h of treatment with LD_{50} dose of bromadiolone bait

a*- Significantly high values of APTT and Mean days to death in resistant rats after 48 h of treatment with LD50 dose of bromadiolone bait

Table 3 — Three sets of primer used to amplify VKORC1 gene with estimated amplicon sizes

S. No	Primer Name	Primer Sequence	Length (bp)	Product size (bp)
1	Rn vkorc1Ex1F	CCGTCAGGTACACTGGTTTTC	21 bp	324
	Rn vkorc1Ex1R	GGCTTGTCAACCTTCCGGT	19 bp	
2	Rn vkorc1Ex2F	GGTGGGGTTGGTCTGGATTT	20 bp	207
	Rn vkorc1Ex2R	ACCAGGCTGACTGCACTTAC	20 bp	
3	Rn vkorc1Ex3F	AGGCTCATGTGCTAAGGCAA	20 bp	312
	Rn vkorc1Ex3R	GGGAGGTGTTACAGAGTTCCA	21 bp	

one of the three exons in *B. bengalensis*, allowing amplification of the full coding sequence (CDS).

PCR reaction and programme

PCR was carried out in 50 µl reaction volumes containing: 20 ng template DNA, 5 µl 10X Taq buffer (Taq DNA polymerase buffer, Sigma Aldrich), 2.5 µl dNTPs, 5 µl forward primer, 5 µl reverse primer, 1 µl Taq polymerase, and 14 µl sterile dH_2O. All PCR amplifications were accomplished in a programmable DNA thermocycler (Mastercycler Gradient-eppendorf™) using a PCR amplification programme of 30 cycles consisting of temperature 95 °C for 5 min (Initial denaturation), 95 °C for 1 min (denaturation), 56 °C for 1 min (annealing), 72 °C for 2 min (extension), 72 °C for 10 min (final extension) and stored at 4 °C until use[17].PCR products were verified by running 10 µl on 0.75% agarose gel stained with ethidium bromide. The remaining 40 µl was purified and sent for Sanger sequencing (Yodha Diagnostics, Hyderabad).

VKORC1 gene analysis

Following Sanger sequencing, the resultant sequences were aligned using BioEdit software, and single nucleotide polymorphisms (SNPs) were identified by comparing sequences from resistant and susceptible individuals.Sequences were also compared against GenBank entries using BLAST (www.ncbi.nlm.nih.gov/Blast).Taxonomy reports confirmed identity as *B. bengalensis*. A phylogenetic tree was constructed with obtained sequences and published VKORC1 sequences of other rat species using MEGA 11 software tool.

Statistical analysis

Data were expressed as mean ± standard deviation (SD). Statistical comparisons between resistant and susceptible groups were performed using a two-sample Student's *t*-test. All analyses were conducted using SPSS software (version 22.0), and differences were considered statistically significant at $P \leq 0.05$.

Results

Survey findings in farmers' fields

Field surveys conducted across rice-growing areas of the Godavari Delta revealed major concerns among farmers regarding frequent rodent infestations and substantial economic losses. Zinc phosphide and bromadiolone were the primary chemical control agents in use for containing the bandicoots in rice fields. However, farmers in high-intensity cultivation areas such as Maruteru, Ramachandrapuram, and Bhimavaram reported more frequent bromadiolone use (2–4 times annually) compared to moderately affected areas (Jonnada, Thadepalligudem, and Ambajipeta), where usage was typically 1-2 cycles per year. Despite intensive baiting campaigns, rapid rodent population recovery within 2-4 weeks was a universal complaint, suggesting declining field efficacy of anticoagulants.

Feeding trials and mortality patterns

All rats consumed the LD_{50} dose (1.2 mg/kg body weight) of 0.005% bromadiolone bait within 24 hours. Feeding was generally uniform, indicating good bait palatability. Mortality commenced on Day 4 and extended until Day 30 of observation. Overall, 116 rats (76.3%) died within the expected window of 4–12 days, while 36 rats (23.68%) showed extended survival or delayed mortality, indicative of reduced susceptibility. Significant differences in mean days-to-death were observed between suspected resistant and susceptible groups in Bhimavaram, Thadepalligudem, Jonnada, and Ramachandrapuram populations ($P \leq 0.05$), whereas differences in Maruteru and Ambajipeta were not statistically significant. Males showed slightly prolonged survival compared to females, consistent with sex-related differences in anticoagulant metabolism reported earlier.

Blood coagulation response (BCR) assay

Coagulation parameters at baseline were within normal physiological limits and showed no inter-location variation. At 48 hours post-exposure, resistant rats exhibited markedly lower PT, INR, and Prothrombin Ratio values compared to susceptible rats ($P \leq 0.05$), confirming functional impairment of vitamin K-dependent clotting pathways but with lesser magnitude of toxicity expression. APTT values also differed significantly in five study locations ($P \leq 0.05$), demonstrating intrinsic pathway involvement. Based on the BCR classification criteria ($PT < 70$ s and INR < 4.0), 36 rats (23.68%) were categorized as resistant phenotypes (Table 2). Across all six locations, resistant rats consistently exhibited higher PCA values (96.8–115.7%)**,** indicating comparatively better preservation of clotting efficiency following bromadiolone exposure. In contrast, susceptible rats showed markedly reduced PCA values (23.0–29.7%)**,** confirming severe

anticoagulant-induced hypocoagulability. These distinct PCA profiles strongly support metabolic resistance in field populations of *B. bengalensis*.

VKORC1 gene sequencing and molecular evidence

PCR amplification using exon-specific primers successfully generated the complete 486 bp VKORC1 coding region from both resistant and susceptible *B. bengalensis* individuals. Sequence analysis confirmed the absence of SNPs associated with anticoagulant resistance in resistant rats. Alignment with publicly available *Rattus* VKORC1 sequences showed 94–98% identity, while revealing species-specific nucleotide variations not previously reported in global databases (Fig.1). Three representative sequences were deposited in GenBank in November 2024: OQ632531 (843 bp, encoding 281 amino acids), OQ632532 (839 bp, encoding 279 amino acids; single silent SNP), and OQ632533 (841 bp, encoding 280 amino acids; species-specific indel). These sequences exhibited ~98% homology to *R. norvegicus* VKORC1 (LC218154.1). Phylogenetic clustering positioned *B. bengalensis* VKORC1 as a distinct clade within Muridae, indicating species-level genetic divergence and corroborating the absence of target-site mediated resistance.

Phylogenetic analysis was performed using the VKORC1 sequences generated in this study along with

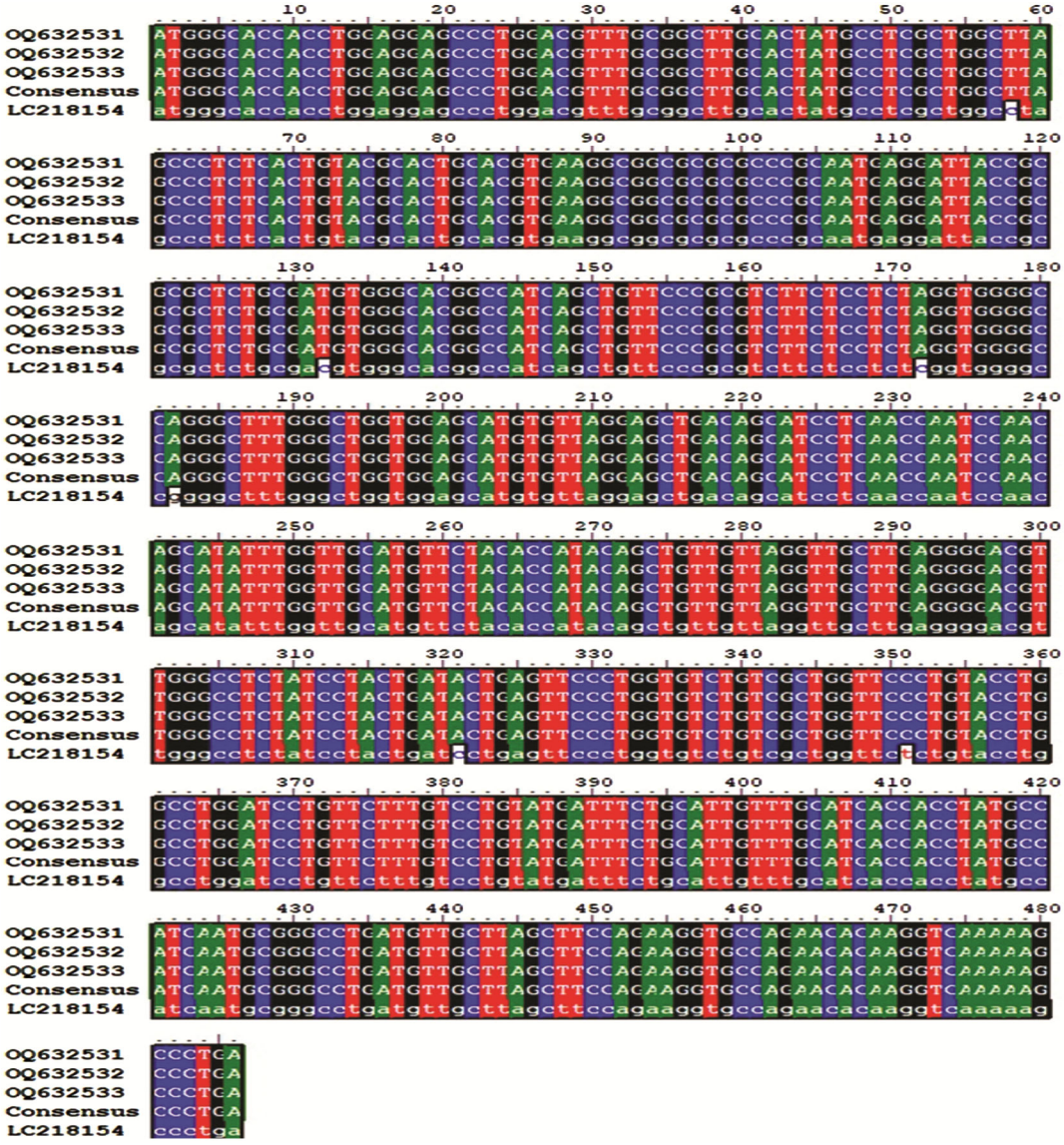

Fig. 1— Genetic sequence comparison of VKORC1 with the published sequence of *Rattus norvegicus*

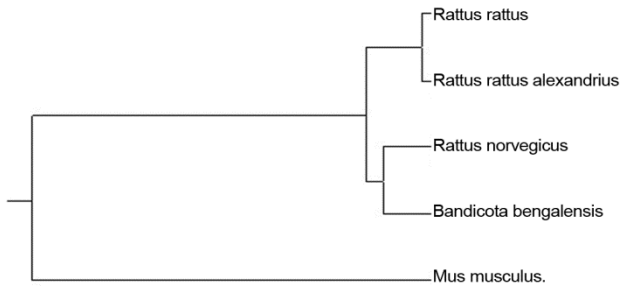

Fig. 2 — Phylogenetic tree of *B. bengalensis* with other rat species based on VKORC1 gene sequence

published reference sequences of related rodent species (Fig. 2). The study samples exhibited approximately 98% sequence homology with *R. norvegicus*, followed by *R. rattus* and *R. rattus alexandrinus*. However, none of the sequences demonstrated complete nucleotide identity or full query coverage with any deposited reference sequence, suggesting that the VKORC1 alleles identified here represent novel genetic variants. The resulting phylogenetic tree showed a distinct clustering of *B. bengalensis* sequences within the *Rattus* genus clade, supporting accurate species identification. Notably, the observed branch-point separations indicate unique nucleotide differences characteristic of these newly detected VKORC1 variants in *B. bengalensis*.

Discussion

Bromadiolone has been extensively used in India since 1988 and is currently the most widely adopted second-generation anticoagulant rodenticide for both agricultural and commensal rodent control. In Andhra Pradesh, its application is frequent in rice-based ecosystems, and increasing tolerance in field rodent populations has been observed. Over-reliance on a single anticoagulant exerts strong selection pressure and facilitates the spread of resistance alleles in wild populations[19]. In the present study, 48 h after exposure to 0.005% bromadiolone, resistant and susceptible *B. bengalensis* exhibited significant differences in PT, INR and R values across locations, whereas APTT did not differ significantly in some populations. These findings align with earlier studies in *B. bengalensis*[15] and *R. rattus*[20], where susceptible rodents showed markedly prolonged coagulation times compared to resistant individuals. The delayed coagulation response in susceptible animals likely reflects bromadiolone-induced disruption of the extrinsic pathway[21]. Similar impairments in clotting function have been observed with other anticoagulants such as chlorophacinone[22]. Although sex-associated differences have been noted previously in house mice, where males were more susceptible to bromadiolone[18]. The present study revealed the opposite trend, with male *B. bengalensis* surviving longer than females following exposure.

Globally, diverse VKORC1 mutations in brown and black rats have been linked with resistance to second-generation anticoagulants[21-23]. In contrast, the present study detected no VKORC1 resistance-associated SNPs, including the widely reported Tyr139 variants, even among rats classified as resistant by feeding and BCR bioassays. Similar cases have been documented in suspected resistant *R. rattus* populations that lacked VKORC1 mutations[16], indicating that resistance can arise through other mechanisms[24-26]. Enhanced cytochrome P450-mediated hepatic metabolism has been recognized as an alternative pathway for bromadiolone detoxification[27]. Warfarin-resistant *R. rattus* from Tokyo exhibited elevated CYP2B, CYP2C and CYP3A enzyme activities and increased microsomal metabolism of warfarin; co-administration of a P450 inhibitor (SKF-525A) restored susceptibility[28]. The moderate coagulation impairment observed in resistant *B. bengalensis* despite lethal exposure in this study strongly supports metabolic detoxification as the predominant resistance mechanism.

Approximately 24% of rats exhibited reduced susceptibility in BCR tests and prolonged survival following bromadiolone exposure, clearly demonstrating a functional resistance phenotype in *B. bengalensis* populations of the Godavari Delta. Farmer surveys further validated these findings by reporting repeated field control failures and rapid rodent resurgence, indicating that resistance has already reached operational significance. These observations, together with the mechanistic insights on enhanced hepatic metabolism and preserved prothrombin activity, confirm the emergence and progressive spread of bromadiolone resistance in a major rice-growing region. Therefore, routine resistance surveillance incorporating both genetic (VKOR-related) and metabolic (cytochrome P450-mediated detoxification) markers is essential. A strategic shift towards integrated rodent management combining chemical rotation, habitat manipulation and ecologically based interventions is urgently required to prevent further resistance escalation and safeguard long-term crop protection outcomes.

Conclusion

This study confirms early-stage physiological resistance to bromadiolone in *B. bengalensis* from the Godavari Delta, with 23.68% of rats exhibiting reduced susceptibility based on feeding and coagulation responses. The absence of VKORC1 mutations indicates that resistance may be driven by alternative mechanisms such as cytochrome P450-mediated enhanced metabolic detoxification. These findings highlight the need for routine resistance surveillance, rational and rotational use of rodenticides, and integration of non-chemical strategies to prevent further resistance escalation. Strengthened policy support is critical to regulate rodenticide use and promote coordinated community rodent management, thereby protecting rice productivity, food security, and farmer livelihoods in India's rice-growing regions.

Ethical statement

Ethical approval was obtained from Institutional Animal Ethics Committee (Approval No: IAEC/VPM/2023-02, dated March 15, 2023).

Conflict of interest

The authors declare that they have no conflict interests.

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