

BIOCHEMICAL MECHANISMS OF INSECTICIDE RESISTANCE AND ASSOCIATED LIFE-HISTORY CHANGES IN FIELD-COLLECTED *NILAPARVATA LUGENS* FROM BARGARH, INDIA

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ABSTRACT

The brown planthopper (*Nilaparvata lugens* Stål) is a major insect pest of rice, and the development of insecticide resistance poses a serious challenge to its effective management. The present study was undertaken to assess the insecticide resistance status, detoxification enzyme activity, and biological characteristics of a field population of *N. lugens* collected from Bargarh, an insecticide-intensive rice-growing region. The field population was evaluated against nine insecticides representing different insecticide groups using a rice plant spray bioassay. Mortality was recorded at 24, 48 and 72 h after treatment, and LC₅₀ and LC₉₀ values were estimated by probit analysis. Resistance ratios were calculated relative to a long-term susceptible glasshouse population maintained at ICAR-Indian Institute of Rice Research, Hyderabad. The Bargarh population exhibited low resistance to acephate, dinotefuran, fipronil and pymetrozine, while comparatively higher resistance was observed against chlorpyrifos. Very high resistance was detected against the neonicotinoids imidacloprid and thiamethoxam, the ethiprole + imidacloprid combination, and the insect growth regulator buprofezin, with the highest resistance recorded against BPH. Biochemical assays revealed elevated activities of esterases, glutathione S-transferase (GST), and cytochrome P450 monooxygenases in the Bargarh population compared with the susceptible glasshouse population, with increases of 1.5-, 1.1- and 2.2-fold, respectively. The greater elevation of P450 monooxygenase activity suggested an important role for oxidative detoxification, particularly in resistance to neonicotinoid insecticides. Biological studies further indicated fitness costs associated with the resistant field population, including prolonged pre-oviposition and nymphal development, lower egg hatchability and nymphal survival, reduced fecundity, and a longer overall life cycle compared with the susceptible population. The findings demonstrate substantial variation in insecticide susceptibility among chemical groups and indicate the involvement of metabolic detoxification in resistance development in the Bargarh BPH population. These results provide useful baseline information for resistance surveillance and support the implementation of insecticide rotation, judicious insecticide use, and integrated pest management strategies for sustainable management of *N. lugens* in rice ecosystems

KEYWORDS: *Nilaparvata lugens*, brown planthopper, insecticide resistance, resistance ratio, LC₅₀, LC₉₀, detoxification enzymes, cytochrome P450, glutathione S-transferase, esterase, Bargarh, rice.

1. INTRODUCTION

Rice is one of the world's most important cereal crops and serves as the major staple food for more than half of the global population. It makes a substantial contribution to dietary energy and protein intake worldwide and, consequently, rice production has a direct bearing on food security and socio-economic stability (Khir et al., 2018). More than 100 insect species are known to attack rice, among which approximately 20 are considered economically important pests (Atwal and Dhaliwal, 2002). The brown planthopper (BPH), *Nilaparvata lugens* (Stål), is one of the most destructive phloem-feeding insect pests associated with rice. BPH outbreaks are favoured by intensive rice production systems, particularly under conditions of adequate irrigation, dense crop planting, excessive nitrogen fertilization and repeated use of insecticides. Severe infestations may cause considerable yield losses, reaching up to 60 per cent under outbreak conditions. In addition to its direct feeding damage, BPH is also an important vector of grassy stunt, ragged stunt and wilted stunt viruses (Chen et al., 1978). Recent studies have continued to identify *N. lugens* as a major limitation to rice production in Asian rice-growing areas, while the increasing development of resistance to commonly used insecticides has made its management progressively more challenging (Ye et al., 2024; Choi et al., 2025). The first major outbreak of BPH in India was recorded in Kerala in 1973, following which the pest became widely distributed across several important rice-growing regions of the country (Kulshrestha et al., 1974). Insect pests collectively are estimated to cause yield losses of about 21 to 51 per cent in Indian rice production (Prakash et al., 2007), contributing to relatively lower productivity compared with countries such as China and Sri Lanka (Krishnaiah et al., 2008). Since the late 1990s, neonicotinoid insecticides have been widely used for the management of BPH. However, the development and subsequent spread of insecticide resistance have progressively compromised the efficacy of several insecticides. In BPH, resistance was first reported from Thailand in 2003 and has subsequently expanded across major rice-producing regions of Asia (Matsumura et al., 2008). Recent area-wide resistance monitoring in China has revealed increasing resistance to several insecticides, including triflumezopyrim, nitenpyram and dinotefuran, while extremely high resistance levels to imidacloprid, thiamethoxam and buprofezin have continued to occur in field populations (Ye et al., 2024). In India,

insecticide resistance in BPH was initially reported from the Godavari delta (Sarupa et al., 1999), and subsequent studies have recorded resistance levels ranging from medium to very high (Jhansi Lakshmi et al., 2010; Basanth et al., 2013; Rao, 2012; Nagendra Reddy, 2016; Mohan et al., 2019). More recent nationwide monitoring has revealed substantial geographical variation in insecticide resistance among BPH populations in India. Field populations collected from different states during 2024-2025 showed moderate to high resistance to imidacloprid and buprofezin, whereas resistance to dinotefuran and pymetrozine varied from low to moderate. In contrast, triflumezopyrim exhibited comparatively lower resistance levels among the populations evaluated, emphasizing the importance of continuous and location-specific monitoring of insecticide resistance for effective resistance management (Naik et al., 2026).

In BPH, insecticide resistance is primarily associated with enhanced metabolic detoxification and degradation of insecticides through enzyme systems including esterases, glutathione S-transferases (GSTs) and cytochrome P450 monooxygenases (Vontas et al., 2002; Heckel, 2012). Recent experimental investigations have provided additional evidence for the functional contribution of these detoxification enzymes to insecticide resistance in BPH. Yang et al., (2024) demonstrated that specific GST, carboxylesterase and P450 genes are involved in the detoxification of nitenpyram, imidacloprid and etofenprox. Furthermore, RNA interference targeting NIGSTD2, NICE and NICYP408A1 resulted in increased susceptibility to insecticides. Similarly, Sun et al., (2024) identified NICYP301B1 and NIGSTm2 as important genes associated with pymetrozine resistance, while silencing these genes increased mortality of BPH following insecticide exposure. The increased expression of detoxification enzymes, which are largely produced in the insect fat body (Karunker et al., 2008), may contribute not only to resistance against individual insecticides but also to cross-resistance among different insecticide groups (Mitchell et al., 2012; Zhang et al., 2016). Recent molecular studies have further demonstrated the important role of the CYP6ER1 system in imidacloprid resistance, with resistance levels being associated with increased expression of the CYP6ER1vA variant (Choi et al., 2025). Besides insect-mediated detoxification mechanisms, emerging evidence suggests that microbial symbionts can also play a role in determining insecticide resistance in BPH. Zeng et al., (2023) reported that the symbiotic bacterium *Serratia marcescens* was capable of metabolizing buprofezin and consequently increasing resistance to this insecticide in *N. lugens*. Further studies have shown that modulation of bacterial symbionts through Nlglectin can also influence insecticide susceptibility in BPH (Li et al., 2025). Thus, a detailed understanding of the biochemical, molecular and microbial mechanisms involved in insecticide resistance is essential for the development of effective resistance-management strategies. Recent studies on Indian BPH populations have demonstrated that increased activities of P450, GST and esterase enzymes are associated with geographical differences in insecticide resistance, highlighting the importance of combining biochemical diagnostics with conventional insecticide-resistance monitoring (Naik et al., 2026). Such information is important for identifying effective insecticides, slowing the development of further resistance and promoting the sustainable management of *N. lugens* in rice ecosystems.

2. MATERIALS AND METHODS

2.1 Collection and Maintenance of *Nilaparvata lugens*

Field populations of the brown planthopper (BPH), *Nilaparvata lugens* (Stål), were obtained from farmers' rice fields in the BPH-endemic area of Bargarh. Collections were made from the basal region of rice plants, targeting preferably the brachypterous forms, with the help of an aspirator. The collected insects were transferred to perforated plastic containers of 25 × 15 cm size. To sustain insect viability during transport, fresh rice stems wrapped in moist cotton were kept inside the containers. Following the method of Nagendra Reddy (2016), about 1,500-2,000 nymphs and adults were collected and brought to ICAR-Indian Institute of Rice Research (IIRR), Rajendranagar, Hyderabad, Telangana. After reaching the institute, the insects were released on potted rice plants free from pesticide exposure and reared under glasshouse conditions inside an insect-proof isolation chamber. The field population was maintained for two generations prior to bioassays. For comparison, a susceptible reference population continuously reared at IIRR for more than 15 years without any insecticide exposure was used as the baseline susceptible population.

2.2 Insecticides and Preparation of Test Solutions

A total of nine insecticides from different chemical groups were evaluated: neonicotinoids - imidacloprid 17.8 SL, thiamethoxam 25 WG and dinotefuran 20 SG; organophosphates - acephate 75 SP and chlorpyrifos 20 EC; phenylpyrazole - fipronil 5 SC; pyridine azomethine - pymetrozine 50 WG; insect growth regulator - buprofezin 25 SC; and a combination formulation - ethiprole 40% + imidacloprid 40% (80 WG).

2.3 Toxicity Bioassay

Insecticidal toxicity was assessed by rice plant spray bioassay method. Potted rice plants of 60 days age were sprayed with five to six concentrations of each insecticide using a hand atomizer to obtain uniform coverage till runoff. The sprayed plants were air-dried for about 3 h and then twenty BPH nymphs of third instar (7-9 days old) were released on each plant, confined within transparent mylar cages. Plants sprayed with distilled water alone served as control. Mortality was recorded at 24, 48 and 72 h after treatment, and moribund insects were counted as dead. The per cent mortality was corrected by Abbott's formula (Abbott, 1925). The LC₅₀, LC₉₀, slope, heterogeneity and 95% fiducial limits were worked out through probit analysis using POLO-Plus software (Version 2.0; Le Ora Software) as per Finney (1971).

2.4 Resistance Ratio Estimation

Resistance ratios (RR) at LC₅₀ and LC₉₀ were determined in relation to the susceptible glasshouse population as per FAO (1979). The levels of insecticide resistance were categorized according to the criteria of Lai et al., (2011) as: susceptibility

(RR = 1), decreased susceptibility (RR = >3-5), low resistance (RR = 6-10), moderate resistance (RR = 11-40), high resistance (RR = 41-160) and very high resistance (RR >160).

2.5 Detoxification Enzyme Assays

2.5.1 Esterase Activity

Esterase activity was estimated by homogenizing five early fifth-instar nymphs of brown planthopper in 0.1 M sodium phosphate buffer (pH 7.0) containing 0.1% Triton X-100. The homogenate was centrifuged at 10,000 rpm for 12 min at 4°C and the supernatant was used as enzyme source. The assay mixture contained 10 mM α -naphthyl acetate as substrate and 4 mM Fast Blue RR as chromogenic reagent. The esterase activity was determined following the method of Han et al., (1998) and expressed as $\mu\text{mol } \alpha\text{-naphthol produced min}^{-1} \text{ mg}^{-1} \text{ protein}$.

2.5.2 Glutathione S-transferase (GST) Activity

GST activity was determined from five fifth-instar nymphs homogenized in 0.1 M Tris-HCl buffer (pH 8.0) containing 10 mM reduced glutathione. The homogenate was centrifuged at 10,000 rpm for 15 min at 4°C and the supernatant was taken for enzyme assay. GST activity was measured spectrophotometrically at 340 nm using 1-chloro-2,4-dinitrobenzene (CDNB) as substrate following the procedure of Habig et al., (1974). The enzyme activity was expressed as $\mu\text{mol CDNB conjugated min}^{-1} \text{ mg}^{-1} \text{ protein}$.

2.5.3 Cytochrome P450 Monooxygenase Activity

Cytochrome P450 monooxygenase activity was assayed by homogenizing nymphs in 50 mM phosphate buffer (pH 7.8) containing 1 mM EDTA and KCl. The homogenate was centrifuged sequentially at 10,000 rpm for 100 min and then at 15,000 rpm for 15 min, and the final supernatant was used as enzyme extract. Monooxygenase activity was estimated using p-nitroanisole as substrate as described by Hansen and Hodgson (1971) and expressed as $\mu\text{mol p-nitroanisole hydrolysed min}^{-1} \text{ mg}^{-1} \text{ protein}$. Protein content in all enzyme extracts was estimated by the method of Lowry et al., (1951) using bovine serum albumin (BSA) as standard.

2.6 Biology of Brown Planthopper

Biological parameters of the glasshouse susceptible and Bargarh field populations of brown planthopper were studied on the susceptible rice variety TN1 as per the procedure of Krishnaiah et al., (2008). Ten replications were maintained for each biological parameter. Pre-oviposition period was studied by confining a single pair of brachypterous adults on a 30-day-old TN1 plant. The pair was transferred daily to fresh plants till oviposition started and the period from adult emergence to first egg laying was recorded as pre-oviposition period. For incubation period, a gravid female was confined on a 30-45-day-old TN1 plant for 24 h for egg laying. The period between egg laying and nymph emergence was taken as incubation period. Fecundity and egg hatchability were worked out by counting total nymphs emerged and unhatched eggs. Total eggs laid was considered as fecundity, while per cent unhatched eggs was used to calculate hatchability. Oviposition period was recorded as the duration between emergence of first and last nymph from eggs laid by a single female. Nymphal development was observed by rearing individual nymphs on isolated rice stems and recording daily moulting, duration of each instar and adult emergence. Nymphal survival and wing-form polymorphism were studied by confining fifty first-instar nymphs on 60-day-old TN1 plants. Number of adults emerged was recorded to assess nymphal survival and adults were categorized into brachypterous and macropterous forms.

3. RESULTS

3.1 Insecticide Resistance Levels

Acephate 75 SP exhibited high toxicity against the Bargarh brown planthopper population, with LC_{50} values remaining constant at 1.7 across 24, 48 and 72 h, while LC_{90} values ranged from 1.3 to 1.7. Chlorpyrifos 20 EC showed comparatively lower toxicity than acephate, with LC_{50} values ranging from 2.7 to 3.1 and LC_{90} values from 2.5 to 4.3 across the three exposure periods. Imidacloprid 17.8 SL exhibited comparatively low toxicity, with LC_{50} values of 120.9, 289.5 and 239.7 at 24, 48 and 72 h, respectively, while the corresponding LC_{90} values were 26.3, 53.8 and 114.7. Thiamethoxam 25 WG also showed comparatively lower toxicity, with LC_{50} values ranging from 26.5 to 63.8 and LC_{90} values from 7.5 to 20.4. In contrast, dinotefuran 20 SG exhibited high toxicity, with LC_{50} values of 1.1-1.2 and LC_{90} values consistently at 1.1 across the exposure periods. Fipronil 5 SC similarly showed high toxicity, with LC_{50} values ranging from 1.1 to 1.4 and LC_{90} values from 1.1 to 1.4. Pymetrozine 50 WG exhibited high toxicity, with LC_{50} values of 1.3-1.6 and LC_{90} values consistently at 1.1. The combination insecticide ethiprole 40% + imidacloprid 40% 80 WG exhibited comparatively lower toxicity, with LC_{50} values increasing from 91.7 at 24 h to 185.4 at 72 h, whereas LC_{90} values decreased from 55.2 to 42.2 over the same exposure period. Buprofezin 25 SC exhibited the lowest toxicity among the tested insecticides, with an LC_{50} value of 957.4 recorded at 72 h. Overall, based on the LC_{50} and LC_{90} values, dinotefuran, fipronil, pymetrozine and acephate exhibited comparatively high toxicity against the Bargarh brown planthopper population, followed by chlorpyrifos, while thiamethoxam, imidacloprid and the ethiprole + imidacloprid combination exhibited comparatively lower toxicity. Buprofezin showed the highest LC_{50} value and therefore the lowest toxicity among the insecticides for which data were available (Table 1 to 10).

Table 1. LC50 and LC90 values of acephate 75 SP

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72h
LC50 (ppm)	211.1	122.2	93.2	406.2	274.2	232.1
LC90 (ppm)	430.1	310.7	202.8	796.5	591.7	439.2
Heterogeneity	1.1	1.0	0.7	0.8	0.8	0.6
Slope	4.1±0.3	3.2±0.3	3.8±0.4	4.3±0.3	3.8±0.3	4.6±0.4
95% limit (FL) LC50	192.8 to 229	101.9 to 139.6	75.4 to 108.1	370.7 to 440.5	240.0 to 305.3	203.7 to 257.5
95% limit (FL) LC90	383.7 to 501.0	275.6 to 363.2	181.3 to 231.8	718.6 to 907.6	527.9 to 684.0	393.9 to 505.1

Table 2. LC50 and LC90 values of chlorpyrifos 20 EC

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	106.6	83.8	62.8	326.1	223.1	169.3
LC90 (ppm)	278.0	176.0	90.2	705.6	540.4	387.5
Heterogeneity	0.5	0.6	0.1	1.0	0.6	0.7
Slope	3.0±0.2	3.9±0.4	8.1±1.7	3.8±0.3	3.3±0.4	3.5±0.5
95% limit (FL) LC50	93.2 to 119.4	73.8 to 92.9	52.5 to 68.3	290.4 to 360.2	183.3 to 256.8	128.5 to 201.3
95% limit (FL) LC90	239.2 to 339.4	155.0 to 209.2	83.5 to 104.3	618.2 to 844.9	469.7 to 656.4	338.0 to 465.7

Table 3. LC50 and LC90 values of imidacloprid 17.8 SL

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	1.8	0.4	0.3	217.7	115.8	71.9
LC90 (ppm)	16.9	4.7	1.5	444.4	252.8	172.1
Heterogeneity	0.5	1.7	2.5	0.6	0.4	0.3
Slope	1.3±0.1	1.2±0.1	1.9±0.2	4.1±0.3	3.7±0.4	3.3±0.6
95% limit (FL) LC50	1.6 to 2.2	0.2 to 0.6	0.1 to 0.5	196.3 to 239.7	98.7 to 131.2	48.3 to 89.1
95% limit (FL) LC90	13.2 to 23.2	3.3 to 8.8	1.2 to 2.3	393.5 to 517.2	219.1 to 308.4	146.8 to 216.1

Table 4. LC50 and LC90 values of thiamethoxam 25 WG

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	1.9	1.3	0.4	62.7	34.4	25.5
LC90 (ppm)	13.4	10.9	2.4	139.1	81.4	48.9
Heterogeneity	1.6	2.8	1.7	0.6	0.5	0.2
Slope	1.5±0.1	1.4±0.1	1.6±0.1	3.7±0.4	3.4±0.6	4.5±1.5
95% limit (FL) LC50	1.4 to 2.5	0.8 to 1.9	0.3 to 0.5	55.1 to 69.3	22.7 to 42.8	4.2 to 36.4
95% limit (FL) LC90	8.9 to 23.9	6.4 to 25	1.7 to 4.1	122.9 to 165.0	71.6 to 95.7	30.8 to 57.4

Table 5. LC50 and LC90 values of dinotefuran 20 SG

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	17.2	11.3	7.7	19.4	13.2	9.3
LC90 (ppm)	33.9	24.7	16.2	38.0	28.3	18.6
Heterogeneity	0.8	0.8	0.3	0.5	0.5	0.3
Slope	4.3±0.4	3.7±0.4	3.9±0.6	4.3±0.3	3.8±0.3	4.2±0.5
95% limit (FL) LC50	15.6 to 18.8	9.5 to 12.8	5.6 to 9.2	17.6 to 21.0	11.4 to 14.7	7.7 to 10.6
95% limit (FL) LC90	30.2 to 39.6	21.7 to 29.4	14.1 to 19.3	34.5 to 42.7	25.3 to 32.4	16.5 to 21.8

Table 6. LC50 and LC90 values of ethiprole + imidacloprid 80 WG

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	3.7	1.5	0.7	339.2	218.2	129.8
LC90 (ppm)	12.3	8.5	5.6	679.3	402.9	236.5
Heterogeneity	2.1	1.1	1.3	1.0	0.7	0.3
Slope	2.4±0.3	1.7±0.3	1.4±0.3	4.2±0.3	4.8±0.5	4.9±1.0
95% limit (FL) LC50	2.7 to 4.8	0.7 to 2.3	0.1 to 1.4	311.6 to 365.6	193.0 to 239.2	78.0 to 161.0
95% limit (FL) LC90	8.7 to 24.3	6.3 to 14.2	3.7 to 9.8	607.8 to 789.2	369.5 to 450.6	205.7 to 268.5

Table 7. LC50 and LC90 values of fipronil 5 SC

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	39.6	29.7	19.3	43.8	34.1	26.8
LC90 (ppm)	61.5	40.9	31.6	70.8	55.5	43.8
Heterogeneity	0.4	0.5	0.4	0.8	0.9	0.4
Slope	6.6±0.9	9.2±2.9	5.9 ±1.6	6.1±0.5	6.0±0.5	5.9±0.8
95% limit (FL) LC50	35.2 to 42.5	16.2 to 34.6	9.6 to 24	41.4 to 46.1	31.3 to 36.3	22.9 to 29.6
95% limit (FL) LC90	57.3 to 68.5	35.5 to 44.2	26.3 to 35.1	66 to 77.5	51.8 to 60.5	40.8 to 47.9

Table 8. LC50 and LC90 values of pymetrozine 50 WG

	Glasshouse BPH			Bargarh BPH		
	24 h	48 h	72 h	24 h	48 h	72 h
LC50 (ppm)	322.4	253.6	190.3	507.1	314.9	275.9
LC90 (ppm)	787.6	558.3	439.7	844.5	643.3	486.6
Heterogeneity	0.9	0.9	0.8	0.7	0.7	0.5
Slope	3.3±0.3	3.7±0.4	3.5±0.4	5.7±0.6	4.1±0.7	5.2±1.1
95% limit (FL) LC50	290.2 to 352.0	219.2 to 282.9	155.8 to 218.5	466.7 to 542.9	230.6 to 372.4	176.1 to 334.1

95% limit (FL) LC90	696.1 to 928.9	497.7 to 652.0	396.4 to 501.6	774.7 to 950.9	577.3 to 748.1	431.7 to 552.6
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Table 9. LC50 values of buprofezin 25 SC at 72 hours

	Glasshouse BPH	Bargarh BPH
LC50 (ppm)	1.5	1436.1
Heterogeneity	1.1	0.8
Slope	1.1±0.1	4.9±0.6
95% limit (FL) LC50	1.1 to 1.9	1236.8 to 1588.8

Table 10. Resistance ratios of different insecticides for Bargarh brown planthopper populations

		LC50			LC90		
		24h	48h	72h	24h	48h	72h
1.	Acephate 75 SP	1.7	1.7	1.7	1.3	1.7	1.4
2.	Chlorpyrifos 20 EC	3.1	2.7	2.7	2.5	3.1	4.3
3.	Imidacloprid 17.8 SL	120.9	289.5	239.7	26.3	53.8	114.7
4.	Thiamethoxam 25 WG	33.0	26.5	63.8	10.4	7.5	20.4
5.	Dinotefuran 20SG	1.1	1.2	1.2	1.1	1.1	1.1
6.	Ethiprole 40% + Imidacloprid 40% 80WG	91.7	145.5	185.4	55.2	47.4	42.2
7.	Fipronil 5 SC	1.1	1.1	1.4	1.1	1.4	1.4
8.	Pymetrozine 50 WG	1.6	1.3	1.4	1.1	1.1	1.1
9.	Buprofezin 25 SC			957.4			

3.2 Detoxification Enzyme Activity

Biochemical analyses revealed elevated detoxification enzyme activities in the Bargarh field population of brown planthopper compared with the susceptible glasshouse population. Esterase activity in the Bargarh population was 0.150 μ moles of α -naphthol formed/min/mg protein, representing a 1.5-fold increase over the glasshouse population. Glutathione S-transferase (GST) activity was also elevated in the Bargarh population, with an activity of 2.508 μ moles of CDNB conjugated/min/mg protein and a 1.1-fold increase over the susceptible population, indicating a comparatively modest contribution of GST-mediated conjugation to insecticide detoxification. The most pronounced increase was observed in cytochrome P450 monooxygenase activity, which reached 0.193 μ moles of p-nitroanisole hydrolysed/min/mg protein and was 2.2-fold higher in the Bargarh population than in the glasshouse population (Table 11).

Table 11. Enzyme activity of different BPH populations

S.N o.	BPH Populations	μ moles of α naphthol formed /min/ mg of protein .	Increase in esterase activity (Number of times)	μ moles of CDNB conjugated /min/mg of protein	Increase in Glutathione S transferase activity (Number of times)	μ moles of p-nitroanisole hydrolysed /min/ mg of protein.	Increase in Cytochrome P450 monooxygenase activity (Number of times)
1	Bargarh	0.150 $\pm$ 0.013	1.5	2.508 $\pm$ 0.059	1.1	0.193 $\pm$ 0.006	2.2
2.	Glasshouse	0.100 $\pm$ 0.006	-	2.195 $\pm$ 0.116		0.087 $\pm$ 0.011	-

These enhanced detoxification enzyme activities, particularly the elevated cytochrome P450 monooxygenase and esterase activities, suggest the involvement of metabolic detoxification mechanisms in the insecticide resistance observed in the Bargarh brown planthopper population. The relatively greater increase in P450 monooxygenase activity indicates that oxidative metabolism may play an important role in contributing to resistance, whereas the smaller increase in GST activity suggests a comparatively limited contribution of GST-mediated detoxification

3.3 Biology

Field-collected insecticide-resistant *N. lugens* from the Bargarh population exhibited a longer pre-oviposition period, prolonged egg incubation, extended nymphal duration, reduced nymphal survival, lower egg hatchability, decreased fecundity, a lower proportion of females, and an overall longer life cycle than the susceptible glasshouse population, indicating a reduction in biological fitness associated with insecticide resistance (Table 12.)

Table 12. Comparison of Biological parameters of Glasshouse and Bargarh populations

	Glasshouse	Bargarh
Pre-oviposition period (days)	2.7 ± 0.15	4.2 ± 0.13
Oviposition period (days)	7.7 ± 0.25	6.8 ± 0.13
Incubation period (days)	5.9 ± 0.18	8.3 ± 0.15
Egg Hatching (%)	90.9 ± 1.46 (72.5)	79.1 ± 2.55
Fecundity (No. of eggs laid/ female)	281.8 ± 9.6 (16.7)	233.5 ± 16.6
Nymphal survival (%)	90.9 ± 0.8 (72.5)	77.2 ± 2.3
Nymphal duration (days)	12.6 ± 0.20	14.4 ± 0.28
Total life cycle (days)	21.2 ± 0.36	26.9 ± 0.50
Females (%)	58.5 ± 2.05 (49.9)	46.5 ± 1.06
Males (%)	41.5 ± 2.06 (40.1)	51.7 ± 1.04
Brachypterous forms (%)	79.8 ± 1.4 (63.3)	47.4 ± 1.21
Macropterous forms (%)	20.1 ± 1.44 (26.7)	52.6 ± 1.31
Brachypterous females (%)	93.7 ± 1.89 (75.4)	74.7 ± 3.20
Macropterous females (%)	11.7 ± 1.89 (20.0)	25.3 ± 3.10
Brachypterous males (%)	25.7 ± 5.19 (30.5)	30.0 ± 2.46
Macropterous males (%)	74.3 ± 4.16 (59.5)	70.0 ± 2.59
1st instar days	3.2 ± 0.10	3.5 ± 0.12
2nd instar	2.3 ± 0.11	2.6 ± 0.10
3rd instar	2.3 ± 0.09	2.6 ± 0.10
4th instar	2.0 ± 0.06	2.6 ± 0.10
5th instar	2.8 ± 0.09	3.1 ± 0.18
Total nymphal duration days	12.6 ± 0.20	14.4 ± 0.28

4. DISCUSSION

4.1 Insecticide Resistance Levels

4.1.1 Organophosphates

Resistance to acephate remained low in the field populations evaluated in the present study. These findings are consistent with earlier reports from India, where *Nilaparvata lugens* populations remained susceptible to acephate (Sarupa et al., 1999; Rao 2012; Mohan 2016; Nagendra Reddy 2016). Similarly low levels of resistance have also been documented in China (Wen et al., 2009) and Karnataka (Basanth et al., 2013). The continued susceptibility of BPH to acephate is likely attributable to relatively lower selection pressure compared with neonicotinoid insecticides. A similar pattern was observed for chlorpyrifos, with field populations exhibiting little or no appreciable resistance, in agreement with previous studies conducted in India and China (Basanth 2013; Peng et al., 2013; Mohan, 2016). In contrast, chlorpyrifos showed slightly greater resistance in the present study, with resistance ratios of 2.7-3.1-fold at LC₅₀ and 2.5-4.3-fold at LC₉₀ across

the different exposure periods. This observation is supported by recent large-scale resistance monitoring in China, where most field populations of BPH collected between 2020 and 2023 exhibited moderate resistance to chlorpyrifos, indicating that resistance to this organophosphate is developing in some geographical populations (Ye et al., 2024 Research published in 2026 demonstrated that odorant-binding protein 7 (OBP7) can contribute to chlorpyrifos resistance by sequestering chlorpyrifos and chlorpyrifos-oxon, thereby reducing their toxic effects (Lin et al., 2026). Similarly, Xu et al., (2026) reported that chemosensory protein 2 (CSP2) functions as a broad-spectrum scavenger of chlorpyrifos and chlorpyrifos-oxon and contributes to resistance through insecticide sequestration.

4.1.2 Neonicotinoids

The high levels of resistance observed against neonicotinoids in the present study are in close agreement with earlier reports from Andhra Pradesh and Telangana (Krishnaiah et al., 2008; Jhansi Lakshmi et al., 2010; Rao, 2012; Mohan, 2016; Nagendra Reddy 2016) as well as from China (Wang et al., 2009; Wen et al., 2009). Such elevated resistance has been primarily attributed to the prolonged and intensive use of neonicotinoid insecticides and enhanced cytochrome P450 monooxygenase-mediated detoxification (Wen et al., 2009). Thiamethoxam also exhibited moderate to high levels of resistance, corroborating earlier findings from the Godavari districts and Karnataka (Jhansi Lakshmi et al., 2010; Rao 2012; Basanth et al., 2013). Cross-resistance among neonicotinoids has been widely reported (Matsumura et al., 2008; Liu et al., 2010), most likely because these compounds share a common mode of action by targeting nicotinic acetylcholine receptors (Elbert et al., 2008). In contrast, dinotefuran remained highly effective against BPH populations, supporting previous observations from Karnataka and Telangana (Basant et al., 2013; Mohan, 2016; Nagendra Reddy, 2016).

Ye et al., (2024) also recorded a rapid increase in resistance to dinotefuran, with the mean resistance ratio increasing from 119.5-fold to 268.1-fold during the monitoring period. Choi et al., (2025) reported significant imidacloprid resistance in *N. lugens*, with a resistance ratio of 32-fold in the resistant population. RNA-seq analysis showed pronounced overexpression of detoxification-related genes, with CYP6ER1 being the most strongly upregulated gene and exhibiting approximately 28-fold higher expression in the resistant strain than in the susceptible strain. Evidence from Indian populations also indicates that resistance to neonicotinoids is geographically variable and that enhanced metabolic detoxification contributes to this variation. Naik et al., (2026) evaluated 14 *N. lugens* populations collected during 2024-2025 and recorded moderate to high resistance to imidacloprid, with resistance ratios ranging from 24.81- to 63.61-fold. Dinotefuran showed comparatively lower resistance levels, although resistance ratios ranged from 6.82- to 21.99-fold. Biochemical analyses revealed a strong association between cytochrome P450 monooxygenase activity and resistance to imidacloprid and dinotefuran, while esterase and glutathione S-transferase activities were also associated with resistance to selected insecticides.

4.1.3 Phenylpyrazoles

No significant resistance to fipronil was detected in the field populations, possibly due to its comparatively limited use under field conditions. The susceptibility observed in the present study is consistent with reports from Andhra Pradesh, Karnataka, and several other Asian regions (Jhansi Lakshmi et al., 2010; Basanth et al., 2013; Mohan, 2016; Matsumura et al., 2008). However, localized fipronil resistance has been reported from China (Zhao et al., 2011), indicating the need for continued resistance monitoring. Fipronil has become widely adopted in several rice-growing regions even though the 2026 nationwide study focused its resistance assays on other commonly used insecticides (Naik et al., 2026).

4.1.4 Pyridine Azomethine

Susceptibility to pymetrozine was maintained across the populations evaluated, which may be attributed to its relatively limited use and its unique mode of action involving transient receptor potential (TRP) channels and the disruption of feeding behaviour (Kaufmann et al., 2004; Maienfisch 2007; Nesterov 2015). These findings are consistent with earlier reports from Telangana (Mohan, 2016). Nevertheless, increasing levels of pymetrozine resistance have been documented in China (Peng et al., 2013; Yang et al., 2016), highlighting the importance of regular resistance surveillance. Recent studies indicate that pymetrozine resistance can develop through enhanced detoxification mechanisms; Sun et al., (2024) identified NICYP301B1 and NIGSTm2 as important genes associated with pymetrozine resistance, with silencing of these genes increasing BPH mortality following pymetrozine exposure.

4.1.5 Insect Growth Regulator

An alarming level of resistance was observed to buprofezin, with all field populations exhibiting very high resistance. These findings are in agreement with reports of high buprofezin resistance from China (Wang et al., 2008c; Zhang et al., 2014) and India (Mohan, 2016; Nagendra Reddy 2016). The widespread adoption of buprofezin following the decline in neonicotinoid efficacy, coupled with its slow-acting nature against nymphal stages, has likely imposed strong selection pressure, leading to resistance development. Furthermore, genetic studies have shown that buprofezin resistance is controlled by multiple autosomal genes and exhibits incomplete recessiveness (Zhuang et al., 2004), making its long-term resistance management particularly challenging. Zeng et al., (2023) demonstrated that the symbiotic bacterium *Serratia marcescens* can metabolize buprofezin and confer resistance to *N. lugens*, while a 2024 CRISPR/Cas9 study confirmed that the G932C mutation in the chitin synthase 1 (CHS1) gene mediates buprofezin resistance.

4.2 Detoxification Enzyme Activity

In the present investigation, little or no resistance was observed to the organophosphorus insecticides acephate, monocrotophos, dichlorvos, and chlorpyrifos in the field-collected *Nilaparvata lugens* population, and the activities of esterase and glutathione S-transferase (GST) were comparable with those of the susceptible glasshouse population. However, GST activity exhibited a statistically significant positive association with resistance to chlorpyrifos and

monocrotophos, supporting earlier findings that GSTs play an important role in organophosphate resistance in several insect species (Che-Mendoza et al., 2009; Yang et al., 2009).

Previous studies have also demonstrated the involvement of cytochrome P450 monooxygenases in neonicotinoid resistance in *N. lugens* (Wen et al., 2009; Qin et al., 2018). Consistent with these reports, the neonicotinoid-resistant populations evaluated in the present study exhibited markedly elevated P450 activity, corroborating the observations of Karunker et al., (2008). Similar trends in metabolic detoxification have been reported by Malathi et al., (2015), who found that esterase activity in field populations was 1.06-3.09 times higher than that in susceptible strains, whereas GST and P450 activities ranged from 1.29-3.41 and 1.03-1.76 times, respectively. Furthermore, Malathi et al., (2015) reported a strong positive correlation between esterase activity and acephate resistance ($r = 0.999$), highlighting the contribution of detoxification enzymes to insecticide resistance in field populations of *N. lugens*.

Recent research provides further support for the importance of these metabolic pathways in BPH resistance. Sun et al., (2024) found significantly increased P450 and GST activities in a pymetrozine-resistant *N. lugens* strain and identified N1CYP301B1 and N1GSTm2 as important contributors to pymetrozine resistance. Silencing of these genes increased insecticide-induced mortality, providing functional evidence for the role of metabolic detoxification in resistance.

More recently, Naik et al., (2026), in a survey of 14 *N. lugens* populations collected from eight Indian states during 2024-2025, reported significant elevation of P450, GST and esterase activities among field populations. P450 activity showed strong associations with resistance to imidacloprid, dinotefuran and buprofezin, while esterase activity was moderately associated with resistance to imidacloprid and triflumezopyrim and GST activity was significantly associated with imidacloprid resistance. These findings are consistent with the present observation that enhanced detoxification enzyme activity, particularly P450 activity, may contribute to metabolic resistance in field populations of *N. lugens*.

The mechanistic importance of P450-mediated detoxification has also been demonstrated in recent molecular studies. Choi et al., (2025) reported that imidacloprid resistance increased in proportion to the expression level of the CYP6ER1vA variant in *N. lugens*, while Lu et al., (2026) demonstrated that overexpression of novel CYP6ER1 variants contributes to sulfoxaflor resistance and cross-resistance to neonicotinoid insecticides. Together, these studies indicate that elevated P450 activity observed at the biochemical level may reflect underlying changes in specific detoxification genes and their expression, although enzyme activity alone does not establish the contribution of any particular gene or pathway in the Bargarh population.

Overall, the biochemical profile of the Bargarh population indicates a greater involvement of oxidative metabolism, as reflected by the 2.2-fold elevation in P450 activity, followed by esterase activity, whereas GST activity showed only a modest increase. The significant associations between individual detoxification enzymes and resistance to specific insecticides further suggest that metabolic resistance in *N. lugens* is insecticide-dependent and may involve different combinations of detoxification pathways. The present findings therefore provide biochemical evidence for the involvement of metabolic detoxification in the Bargarh population, while molecular characterization of candidate P450, GST and esterase genes would be required to establish the precise mechanisms underlying the observed resistance.

4.3 Biology

Similar fitness costs in resistant BPH populations selected with fenvalerate, fipronil, chlorpyrifos, and imidacloprid have been reported by Ling et al., (2009), Yang et al., (2014), Liu and Han (2006), and Ling et al., (2011b). In the present study, the egg incubation period ranged from 6 to 8 days, with hatchability varying between 79.1 and 90.9%. These observations are in agreement with previous reports describing an incubation period of approximately one week (Khaire & Dumbre 1981), 7-8.5 days at 28-29°C (Mochida & Okada 1979), and about 7 days at 25-30°C (Mochida & Dyck 1976). Similarly, Bae and Pathak (1970) recorded more than 90% egg hatchability at 29°C, while Tatarwal et al., (2015) reported an incubation period of 6-10 days (mean 9.6 ± 0.24 days) with 80.2% hatchability.

The Bargarh and glasshouse populations completed five nymphal instars, with a total nymphal duration of 13-14 days. This finding is comparable with the 16-20 days reported by Tatarwal et al., (2015) and the stage-wise developmental durations described by Khaire and Dumbre (1981), although the comparatively shorter developmental period reported by Rajendram and Daniel (1986) may reflect geographical variation. Nymphal survival observed in the present study was comparable to the approximately 90% survival reported by Bae and Pathak (1970) at 29°C, whereas Cook and Perfect (1989) documented considerably lower egg-to-adult survival (1-12%).

Recent studies further support the occurrence of fitness costs associated with insecticide resistance in *N. lugens*. Dhyan Chowdary et al., (2025) reported that imidacloprid-resistant *N. lugens* exhibited significantly prolonged egg, third- and fifth-instar, pre-adult and total pre-oviposition periods, together with reduced fecundity compared with the susceptible strain. These findings closely support the present observation of prolonged development, reduced fecundity and lower reproductive performance in the Bargarh population.

Similarly, Feng et al., (2023) demonstrated resistant insects showing reductions in longevity, adult duration, pre-oviposition period and fecundity. These observations indicate that resistance development may involve trade-offs between insecticide tolerance and biological performance, which is consistent with the reduced fecundity, nymphal survival and delayed development recorded in the Bargarh population.

5. CONCLUSION

The present study provides important baseline information on the insecticide resistance status of *Nilaparvata lugens*, which can serve as a foundation for resistance surveillance and the development of effective management strategies. The

findings have implications for Integrated Pest Management (IPM) programmes by supporting the selection and judicious application of insecticides that remain effective against field populations of BPH. Since insecticide resistance is a dynamic process that can change with continued selection pressure, regular and systematic monitoring of susceptibility is essential for detecting shifts in resistance levels, enabling timely management recommendations, and reducing the risk of control failures. The study further highlights the need for location-specific resistance management strategies that consider the prevailing resistance profiles of BPH populations across different rice-growing ecosystems. In addition, the development and application of reliable molecular markers for detecting resistance-associated genotypes could facilitate early identification of resistant populations, strengthen resistance surveillance, and improve the implementation of long-term resistance management programmes.

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