

# Inheritance mode and realized heritability of resistance to imidacloprid in the brown planthopper, *Nilaparvata lugens* (Stål) (Homoptera: Delphacidae)

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## Abstract

**BACKGROUND:** The brown planthopper, *Nilaparvata lugens* (Stål), is a serious pest that causes enormous losses to the rice crop in Asia. The genetic basis of imidacloprid resistance was investigated in *N. lugens*.

**RESULTS:** The resistant strain, selected for imidacloprid resistance from a field population of *N. lugens* collected from Nanjing, Jiangsu Province, China, showed a 964-fold resistance compared with the laboratory strain. Progenies of reciprocal crosses ( $F_1$  and  $F_1'$ ) showed similar dose–mortality responses ( $LC_{50}$ ) to imidacloprid, and also exhibited a similar degree of dominance ( $D$ ), 0.58 for  $F_1$  and 0.63 for  $F_1'$ . Chi-square analyses of self-bred and backcross progenies ( $F_2$ ,  $F_2'$  and BC respectively) rejected the hypothesis for a single gene control of the resistance. The estimated realized heritability ( $h^2$ ) of imidacloprid resistance was 0.1141 in the resistant strain of *N. lugens*.

**CONCLUSION:** The results showed that imidacloprid resistance in *N. lugens* was autosomal and was expressed as an incompletely dominant trait, probably controlled by multiple genes.

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**Keywords:** *Nilaparvata lugens*; imidacloprid; resistance inheritance; realized heritability

## 1 INTRODUCTION

The brown planthopper, *Nilaparvata lugens* (Stål), is a monophagous pest that is capable of causing enormous and serious yield loss to the rice crop in Asia.<sup>1,2</sup> In recent years, *N. lugens* outbreaks have been more common in Asian countries because the insect has developed medium to high levels of resistance to major insecticides including organochlorines, organophosphates, carbamates, insect growth regulators and neonicotinoids.<sup>3,4</sup>

Imidacloprid, the first member of the neonicotinyl insecticides, was registered for controlling *N. lugens* on rice in the early 1990s. It quickly became the primary insecticide in many rice-growing areas in China because of its systemic nature and high efficacy against sucking insects.<sup>3</sup> However, farmers have begun to switch to other insecticides since 2005 because of decreased efficacy of imidacloprid against *N. lugens*.<sup>4</sup> In an effort to understand the resistance mechanisms, researchers found that piperonyl butoxide (PBO) could synergize imidacloprid toxicity in green peach aphid (*Myzus persicae* Sulzer), cat flea (*Ctenocephalides felis* Bche), housefly (*Musca domestica* L.) and tobacco whitefly (*Bemisia tabaci* Genn.), suggesting that cytochrome-P450-mediated detoxification might be an important biochemical mechanism for imidacloprid resistance.<sup>5–8</sup> In addition, a study revealed a point mutation (Y151S) in two nicotinic acetylcholine receptor (nAChR) subunits, which was

potentially associated with imidacloprid resistance in a laboratory selected strain of *N. lugens*.<sup>9</sup>

The success of insecticide resistance management strategies depends on a variety of factors, including the clarification of the mode of resistance inheritance.<sup>10</sup> An improved understanding of the genetics of resistance enhances the ability to design and apply resistance management programs,<sup>11</sup> such as facilitating the formulation of strategies to slow down the development of imidacloprid resistance in *N. lugens*. Information about the mode of inheritance of resistance to an insecticide can improve resistance detection,

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monitoring, risk assessment, modeling and management of the resistance.<sup>12,13</sup> Therefore, the genetic basis of resistance to some insecticides was examined in many *N. lugens* populations. Results showed that the inheritance of resistance to methamidophos, malathion and isoprocarb was controlled by an incompletely dominant autosomal gene,<sup>14–16</sup> while resistance to buprofezin was inherited as an incompletely recessive autosomal trait.<sup>17</sup> Because imidacloprid is a relatively new insecticide, the genetic basis of resistance in *N. lugens* has not yet been documented.

This study was designed to elucidate the mode of inheritance and estimate the realized heritability of imidacloprid resistance in *N. lugens*. Inheritance patterns were examined to determine the degree of dominance of the resistance, possible sex linkages and the monogenic or polygenic nature of the resistance.

## 2 MATERIALS AND METHODS

### 2.1 Insects

Two strains of *N. lugens* were used in the study. The susceptible strain (S) was collected in 1995 from a rice field near Hangzhou, Zhejiang Province, China, and reared continuously in the laboratory without exposure to insecticides for approximately 120 generations. Rearing conditions were  $27 \pm 1$  °C and a photoperiod of 16:8 h (light:dark).

The resistant strain (R) was collected in 2005 from a field population near Nanjing, Jiangsu Province, China. This population had developed ~200-fold resistance to imidacloprid, and was used as the parent generation (G0 of the resistant strain) in this study for imidacloprid selections. The third-instar nymphs of every generation were treated with imidacloprid, and the study lasted a total of 23 generations.

### 2.2 Insecticide

Imidacloprid (95.3% TC) was supplied by Changlong Chemical Industrial Group Co. Ltd (Changzhou, Jiangsu, China). This was formulated as a 25 g L<sup>-1</sup> EC containing 100 g L<sup>-1</sup> Triton X-100 in acetone as solvent for the laboratory assays.

### 2.3 Bioassay

Bioassays were carried out by using a rice-stem dipping method to assess the resistance of third-instar nymphs of *N. lugens* according to Zhuang *et al.*<sup>18,19</sup> Rice plants at the tillering to booting stages were pulled out and washed thoroughly. Rice stems (about 10 cm length) with roots were cut and air dried to remove excess water. Three rice stems were grouped and dipped into the appropriate insecticide test solution for 30 s. Three replicates (groups of three rice stems) were used per dose. Imidacloprid EC was diluted in distilled water, and a set of serial concentrations (5–6 doses plus distilled water only as a control) were prepared for each trial. Owing to variable resistance levels in test colonies, imidacloprid concentrations were adjusted accordingly to generate a desirable range of mortality (10–90%) in treated planthoppers. The test concentrations ranged from 0.03125 to 1.0 mg L<sup>-1</sup> for the susceptible strain and from 7.8125 to 250.0 mg L<sup>-1</sup> for the resistant strain. After the rice stems had been air dried for approximately 1 h, moistened cotton was used to wrap the rice roots. Treated rice stems (three per replicate) were then placed into a 500 mL plastic cup. Twenty third-instar nymphs of *N. lugens* were introduced into each plastic cup using a vacuum device. The cups were covered with sponge to ensure air circulation. The treated insects were maintained at a temperature

of  $27 \pm 1$  °C and a photoperiod of 16:8 h (light:dark). Mortality was recorded after 96 h for imidacloprid treatments. The nymphs were considered dead if they did not move after a gentle prod with a fine bristle.

### 2.4 Genetic crosses

After being selected for 23 generations with imidacloprid, the resistant strain exhibited a high level of resistance (964-fold based on LC<sub>50</sub> values) compared with the susceptible strain which had been maintained in laboratory for 120 generations. These two strains were considered as homologous in their genetic composition.<sup>20</sup> To determine resistance inheritance to imidacloprid, virginity of *N. lugens* for mass mating was ensured by separating males and females within 24 h of nymph eclosion. Reciprocal crosses were made by mass mating between the S and R strains to produce two lines: F<sub>1</sub> (S♀ × R♂) and F<sub>1</sub>' (S♂ × R♀). A backcross line (BC) was generated by mating F<sub>1</sub>♀ with S♂. Two F<sub>2</sub> lines (F<sub>2</sub> and F<sub>2</sub>') were obtained through inbreeding of the progenies of the two reciprocal crosses (F<sub>1</sub>♀ × F<sub>1</sub>♂ and F<sub>1</sub>'♀ × F<sub>1</sub>'♂).

### 2.5 Analysis of mortality data

Mortality was corrected by using Abbott's formula.<sup>21</sup> Probit analysis was conducted using the POLO-PC program. The median lethal concentrations (LC<sub>50</sub>) were calculated, and any two LC<sub>50</sub> values were considered significantly different if their corresponding 95% confidence limits (CLs) did not overlap. Resistance ratios (RRs) were determined by dividing the LC<sub>50</sub> values of the resistant strain by the LC<sub>50</sub> value of the susceptible strain.

### 2.6 Analysis of resistance inheritance

By using the method of Stone<sup>22</sup> and Bourguet *et al.*,<sup>23</sup> the degrees of dominance (*D*) of imidacloprid resistance were calculated:

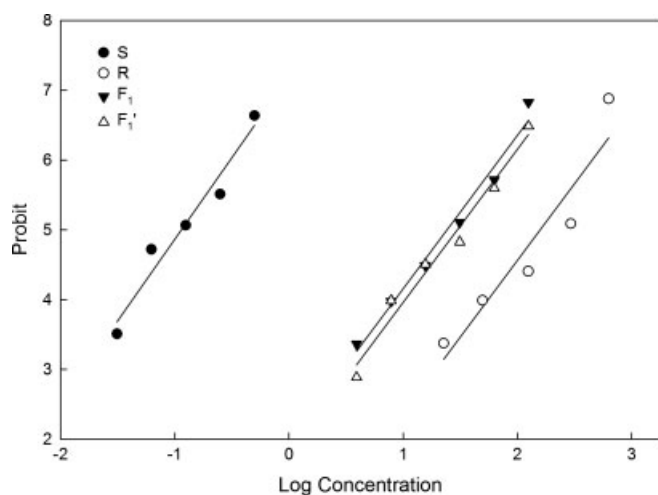
$$D = (2x_F - x_R - x_S) / (x_R - x_S)$$

where  $x_F$ ,  $x_R$  and  $x_S$  are the logarithms of LC<sub>50</sub> values for the reciprocal progeny (F<sub>1</sub> or F<sub>1</sub>'), the resistant strain and the susceptible strain respectively. The degree of dominance values ranged from -1 (completely recessive resistance) to 1 (completely dominant resistance).<sup>22,23</sup> The null hypothesis of monogenic resistance was tested on the basis of chi-square goodness-of-fit between the observed mortality and the theoretical expectation according to Sokal and Rohlf:<sup>24</sup>

$$\chi_i^2 = (N_i - pn_i)^2 / pqn_i$$

where  $N_i$  is the observed number of deaths at a given dose,  $n_i$  is the expected number of deaths at a given dose,  $p$  is the expected mortality estimated as described by Georghiou<sup>25</sup> and  $q = 1 - p$ . The null hypothesis was then tested by comparing the sum  $\chi^2 = \sum N_i \chi_i^2$  with values from the chi-square table with  $N$  degrees of freedom. The null hypothesis was rejected if this test indicated that the observed mortality was significantly different from the expected mortality.

Log dose–probit lines of backcross and self-bred progenies were also used to estimate the number of factors responsible for resistance. According to Tsukamoto,<sup>26</sup> log dose–probit lines of the resistant strain, the susceptible strain and their reciprocal progenies do not overlap if the resistance is controlled by a single gene. In addition, the log dose–probit lines show plateaus at mortality levels of around 25 and 75% for the self-bred progenies, and of around 50% for the backcross progenies.



**Figure 1.** Log dose–probit lines for S and R strains and  $F_1$  and  $F_1'$  progenies of *Nilaparvata lugens* to imidacloprid.

### 2.7 Estimation of realized heritability

To assess risk of imidacloprid selection on resistance development, realized heritability ( $h^2$ ) was estimated by using the method described by Tabashnik<sup>27</sup> as  $h^2 = R/S$  (where  $R$  is the response to selection and  $S$  is the selection differential). Response to selection ( $R$ ) was estimated as

$$R = [\log(\text{final LC}_{50}) - \log(\text{initial LC}_{50})]/n$$

where the final  $\text{LC}_{50}$  is the  $\text{LC}_{50}$  of offspring after  $n$  generations of selection, the initial  $\text{LC}_{50}$  is the  $\text{LC}_{50}$  of the parental generation before the selections start and  $n$  is the number of generations selected. The selection differential ( $S$ ) was estimated as

$$S = i \times \sigma_p$$

where  $i$  is the intensity of selection and  $\sigma_p$  is the phenotypic standard deviation. The intensity of selection ( $i$ ) was estimated as

$$i = 1.583 - 0.0193336p + 0.0000428p^2 + 3.65194/p$$

where  $p$  is the average percentage of surviving rate.<sup>28</sup> The phenotypic standard deviation ( $\sigma_p$ ) was estimated as

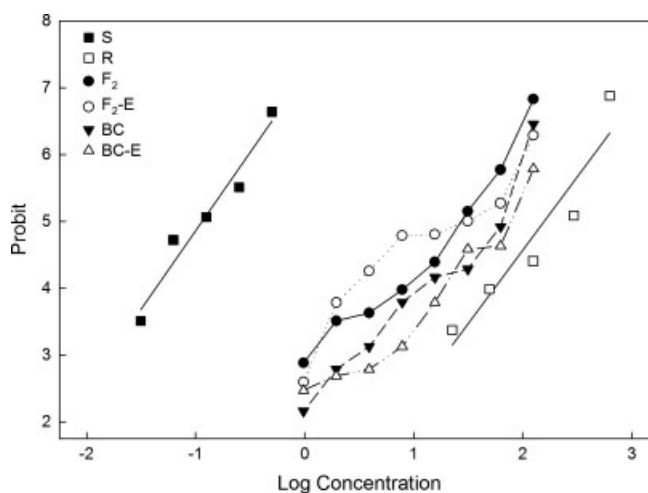
$$\sigma_p = [1/2(\text{initial slope} + \text{final slope})]^{-1}$$

where the initial slope is the slope of the probit regression lines from the parental generation before selection, and the final slope is the slope of the probit regression lines from offspring after  $n$  generations of selection.

## 3 RESULTS

### 3.1 Inheritance of imidacloprid resistance

Dose–mortality responses to imidacloprid in susceptible and resistant strains and in  $F_1$  progenies of reciprocal crosses ( $F_1$  and  $F_1'$ ) were characterized by nearly straight lines (Fig. 1), suggesting that the S and R strains were likely to be homogeneous for susceptibility and resistance to the insecticide. The results of bioassays for reciprocal cross progenies showed no significant differences in the  $\text{LC}_{50}$  values between  $F_1$  ( $R_{\text{♀}} \times S_{\text{♂}}$ ) and  $F_1'$



**Figure 2.** Log dose–probit lines for S and R strains and  $F_2$  and BC progenies of *Nilaparvata lugens* to imidacloprid.  $F_2$ -E and BC-E are the expected values.

( $R_{\text{♂}} \times S_{\text{♀}}$ ) (Table 1), indicating that the resistance to imidacloprid was inherited autosomally in *N. lugens*. The degree of dominance ( $D$ ) was 0.58 and 0.63 for  $F_1$  and  $F_1'$  respectively. These results suggested that imidacloprid resistance in *N. lugens* was expressed as an incompletely dominant trait.

To test whether imidacloprid resistance in *N. lugens* was monogenically inherited, expected mortalities were calculated and were compared with observed mortalities. Results of the goodness-of-fit chi-square test showed that the observed mortalities of all crosses were significantly different from the expected mortality ( $\chi^2 = 55.17, 43.12$  and  $284.15$  for  $F_2, F_2'$  and BC respectively, significantly higher than the value of 14.1,  $df = 7, P < 0.05$ , in the chi-square table), rejecting the hypothesis of monogenic mode of inheritance. This estimate supported the conclusion that imidacloprid resistance was conferred by more than one gene.

The observed and expected probabilities for the backcross progenies (BC) and the inbred progenies ( $F_2$  and  $F_2'$ ) were plotted against imidacloprid concentrations (Figs 2 and 3). Based on the patterns of the dose responses, the resistance in *N. lugens* typically matched the polygenic inheritance characteristic according to the method of Tsukamoto.<sup>26</sup> The observed mortality (log dose–probit lines) of the backcross progenies (BC) showed no plateau at 50% mortality (i.e. probit = 5.0), and the observed mortality (log dose–probit lines) of the  $F_2$  progenies from inbreeding ( $F_2$  and  $F_2'$ ) showed no plateaus at 25% and 75% mortality levels (i.e. probit 4.33 and 5.67 respectively).

### 3.2 Realized heritability

After the R strain of *N. lugens* was selected with imidacloprid for 23 generations, the  $\text{LC}_{50}$  value was increased from 16.01 (13.21–18.73)  $\text{mg L}^{-1}$  to 106.05 (88.06–131.23)  $\text{mg L}^{-1}$ , and the slope of the log dose–probit line increased from 1.9664 to 2.0604 in the R strain. Therefore, the estimated realized heritability ( $h^2$ ) was 0.1141 to imidacloprid in *N. lugens* (Table 2).

## 4 DISCUSSION

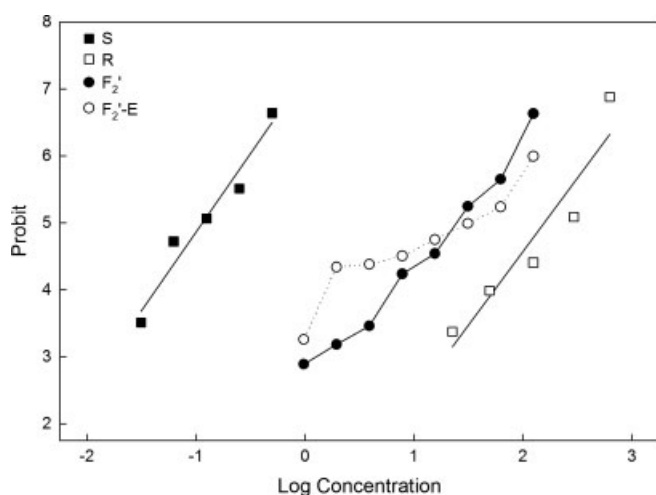
Inheritance of imidacloprid resistance has not previously been studied in *N. lugens*. In the Colorado potato beetle, *Leptinotarsa decemlineata* (Say), resistance to imidacloprid was inherited as

**Table 1.** Probit analysis of dose–mortality responses ( $LC_{50}$ ) to imidacloprid, resistance ratios (RRs) and degree of dominance ( $D$ ) in resistant (R) and susceptible (S) strains and their progenies from reciprocal crosses ( $F_1$  and  $F_1'$ ) of *Nilaparvata lugens*

Strain	$n$	Slope ( $\pm SE$ )	$LC_{50}$ (95% CL) ( $mg L^{-1}$ )	RR	$D$	$\chi^2$ (df)	Probability
S	420	2.16 ( $\pm 0.31$ )	0.11 (0.09–0.14)	1.0		6.6 (3)	0.09
R	420	2.06 ( $\pm 0.47$ )	106.05 (88.06–131.23)	964.09		1.1 (3)	0.78
$F_1$ (S $\times$ R)	420	2.13 ( $\pm 0.52$ )	25.68 (21.64–30.57)	233.45	0.58	2.2 (4)	0.70
$F_1'$ (S' $\times$ R')	420	2.06 ( $\pm 0.48$ )	30.19 (25.35–36.24)	274.45	0.63	3.6 (4)	0.47

**Table 2.** Estimation of realized heritability ( $h^2$ ) of imidacloprid resistance in *Nilaparvata lugens*

No. of generations selected ( $n$ )	Estimate of mean response per generation			Estimate of mean selective differential per generation						
	Initial $LC_{50}$ (log)	Final $LC_{50}$ (log)	Response to selection ( $R$ )	$p$	$i$	Initial slope	Final slope	$\sigma_p$	Selection differential ( $S$ )	$H^2$
23	1.2044	2.0255	0.0357	50.19	0.7606	1.9664	2.0604	0.4115	0.3130	0.1141



**Figure 3.** Log dose–probit lines for S and R strains and  $F_2'$  progeny of *Nilaparvata lugens* to imidacloprid.  $F_2'$ -E indicates the expected values.

an incompletely recessive trait.<sup>29</sup> However, it was found that the inheritance of imidacloprid resistance in *N. lugens* was different from that of *L. decemlineata*. No significant difference was found in the comparison of the  $LC_{50}$  values for the reciprocal cross progenies  $F_1$  and  $F_1'$ . The present bioassay data also showed a positive degree of dominance for the  $F_1$  and  $F_1'$ , i.e. 0.58 and 0.63 respectively. These results suggested that the resistance to imidacloprid was autosomally inherited as an incompletely dominant trait in the R strain of *N. lugens* (Table 1).

In this study it was found that *N. lugens* increased resistance ratios to imidacloprid from 200-fold in the starting generation to 964-fold after the resistant strain was further selected with imidacloprid in the laboratory for 23 generations. The estimated  $h^2$  was 0.1141 for 23 generations of selection with imidacloprid, suggesting that *N. lugens* could develop a certain level of resistance to imidacloprid when the target insect received constant treatment in the laboratory. According to Tabashnik,<sup>27</sup> the number of generations required for a tenfold increase in  $LC_{50}$  value of the resistant strain was estimated to be 29 generations if a field population received prolonged and uniform exposure to imidacloprid and 50.2% of individuals survived the selection

(i.e.  $p = 0.502$ ) in each generation. The estimated  $h^2$  value from laboratory selection experiments could be higher than in the field owing to the reduced environment variation.<sup>30</sup> Although laboratory experiments do not completely reflect field conditions, the estimated  $h^2$  value provides evidence for the potential of further increase of the resistance in *N. lugens*.<sup>27</sup>

Because understanding the resistance inheritance is important for predicting the continuing and effective use of a chemical for a particular pest control,<sup>31</sup> the mode of resistance inheritance in *N. lugens* through laboratory selection with imidacloprid was examined. The data generated from this study could lead to a better understanding of the rate of resistance development by use of the information on the number of genes involved, and the degree of dominance. Computer models suggested that resistance controlled by two or more genes would develop more slowly than that determined by a single gene.<sup>32–34</sup> The degree of dominance of resistance alleles may play a significant role in the expression and distribution of the resistance gene.<sup>11</sup> If insecticide resistance is controlled by a dominant gene, it will make chemical control more difficult since heterozygotes are also resistant.<sup>11</sup> The resistance with dominant alleles may develop faster than the resistance inherited as a recessive trait, because resistant genotypes, including heterozygotes, might have a higher chance of surviving insecticide treatment, and then tend to increase (R:S = 3:1 for dominant versus 1:3 for recessive) and spread faster in field populations.<sup>11,35,36</sup> Although the resistance to imidacloprid in *N. lugens* is not completely dominant, caution must be taken in resistance management because the heterozygotes can tolerate a significantly higher dose of imidacloprid than the susceptible insects (Table 1 and Fig. 1).

The rapid development of resistance to imidacloprid in *N. lugens* might be associated with many biological and physiological characteristics of the insect, such as a short generation period, long-distance migratory ability and monophagy. These intrinsic features of the insect are hard to manipulate or control directly by human intervention. However, alteration of agronomic practices, such as the timing and placement of crops, could help to improve insecticide efficacy and host plant resistance/tolerance or develop integrated approaches for suppressing *N. lugens* abundance and minimize the need for continuous reliance on insecticide

applications.<sup>37</sup> A comprehensive and systematic strategy for delaying resistance development and managing resistance is therefore necessary. The present data, including inheritance analyses and estimation of realized heritability and resistance selection, along with resistance monitoring and preliminary examination of the resistance mechanism,<sup>3,38</sup> might be valuable for formulating resistance management strategies.

A rotational scheme for insecticides with different modes of action and resistance mechanisms including cross-resistance<sup>39,40</sup> should be adopted for control of *N. lugens*. This pest management strategy could reduce selection pressure and result in recovery of susceptibility (data not shown). Besides avoiding frequent use of a single insecticide during a growing season, other strategies could include timely application to achieve the best efficacy, and improving application techniques to maintain long-term effectiveness. The primary goal of these practices is to alleviate selection pressure and to slow down resistance development.

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