Modeling the Relationship Between Development Rate and Temperature in Tetranychus truncatus and Risk Assessment of Resistance to Pyridaben: Postprint

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Abstract

To clarify the effect of temperature on the developmental rate of pyridaben-resistant (Py-R) and susceptible (SS) populations of Tetranychus truncatus and to assess the resistance risk of T. truncatus to pyridaben, this study fitted the relationship curves between generation developmental rate and temperature for the two populations using the Wang-Lan-Ding model under six temperature gradients of 16 °C, 20 °C, 24 °C, 28 °C, 32 °C, and 36 °C, and estimated the realized heritability of resistance to pyridaben in T. truncatus and predicted the resistance risk to pyridaben under different selection pressures using the threshold trait analysis method in quantitative genetics. The results showed that, based on the fitted Wang-Lan-Ding model, the lower and upper critical temperatures for generation development of the susceptible SS population of T. truncatus were 10.05 °C and 39.24 °C, respectively, and those for the resistant Py-R population were 13.45 °C and 41.89 °C, respectively; the upper critical temperature values for all mite stages in the resistant population were significantly greater than those in the susceptible population, indicating that the pyridaben-resistant population of T. truncatus had greater tolerance and adaptability to high temperatures than the susceptible population. The estimated realized heritability (h^2) of resistance to pyridaben in T. truncatus was 0.11, and the h^2 values during the early and middle stages of resistance selection were 0.12 and 0.18, respectively, which were greater than that in the later stage (0.08); at the end of selection, the h^2 value increased again to 0.14. Under laboratory selection conditions ($h^2 = 0.11$), when the selection pressure (i.e., mortality rate) was 50%–90%, a 10-fold increase in resistance was predicted to require only 10–23 generations; whereas under field selection pressure ($h^2 = 0.05$), under the same conditions, a 10-fold increase in resistance would require only 21–46 generations. Therefore, T. truncatus poses a certain resistance risk to

pyridaben, and rotation with other insecticides without cross-resistance, along with reduced selection pressure, could be employed to delay the development of resistance.

Full Text

Simulation of Developmental Rate in Relation to Temperature and Assessment of Resistance Risk to Pyridaben in *Tetranychus truncatus* Ehara

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Abstract

To clarify the effects of temperature on the developmental rate of pyridaben-resistant (Py-R) and susceptible (SS) populations of *Tetranychus truncatus* Ehara and to assess the resistance risk of this mite to pyridaben, we fitted the relationship between generation developmental rate and temperature using the Wang-Lan-Ding model across six temperature gradients (16 °C, 20 °C, 24 °C, 28 °C, 32 °C, and 36 °C). We also estimated the realized heritability of resistance to pyridaben and predicted resistance risk under different selection pressures using threshold trait analysis from quantitative genetics. The fitted Wang-Lan-Ding models revealed that the minimum and maximum critical temperatures for generation development were 10.05 °C and 39.24 °C for the susceptible SS population, and 13.45 °C and 41.89 °C for the resistant Py-R population, respectively. The maximum critical temperature for each developmental stage was significantly higher in the resistant population, indicating greater tolerance and adaptability to high temperatures compared with the susceptible population. The realized heritability (h^2) of resistance to pyridaben was estimated at 0.11, with h^2 values of 0.12 and 0.18 during the early and middle stages of selection, respectively—both higher than the late-stage value of 0.08—while h^2 rebounded to 0.14 at the final selection stage. Under laboratory selection conditions (h^2 = 0.11) with mortality rates of 50%–90%, only 10–23 generations were required for a 10-fold increase in resistance. Under field selection pressure (h^2 = 0.05), the same level of resistance development required 21–46 generations. Therefore, *T. truncatus* poses a certain resistance risk to pyridaben, and management strategies should include rotation with insecticides lacking cross-resistance and reduction of selection pressure to delay resistance evolution.

Keywords: *Tetranychus truncatus*; developmental rate; temperature; pyridaben; resistance risk

Introduction

In recent years, *Tetranychus truncatus* Ehara has become one of the primary pests in the Hexi region of Gansu Province. The expansion and concentrated cultivation of seed corn, combined with high-temperature and drought conditions, have created favorable environments for severe mite infestations. Previous reports indicate that the dominant mite species attacking crops in this region include *T. truncatus*, *T. cinnabarinus* Boisdural, and *T. urticae* Koch, with *T. truncatus* being the predominant species in corn fields [1–2]. This mite has a broad host range, damaging not only corn but also cotton, sugar beet, soybean, eggplant, and various weeds. Prolonged reliance on chemical control has led to the development of resistance to numerous acaricides and insecticides, including methamidophos, malathion, dicofol, and fenpropathrin [3–5], complicating management efforts.

Temperature represents the most significant ecological factor influencing insect development and population dynamics. However, most previous studies on mite resistance have evaluated biological fitness under different temperature conditions to assess population responses to environmental stress. For instance, He Lin et al. [6], Chen Wenbo et al. [7], and Fu Haibo et al. [8] reported that resistant and susceptible mite populations exhibit different fitness advantages or disadvantages across temperature gradients, yet these studies failed to explain the fundamental mechanisms enabling long-term survival of resistant populations.

If temperature is assumed to be the sole limiting factor for organismal development, developmental risk primarily originates from extreme low- and high-temperature zones. Populations that have survived and reproduced to the present day have presumably avoided these risks through ecological strategies that minimize exposure to limiting developmental rates at temperature extremes [9]. Zhou Yihong et al. [10] used Logistic and Wang-Lan-Ding models to characterize the relationship between generation developmental rate (V) and temperature (T) in *Liriomyza sativae* and *L. huidobrensis*, revealing differences in their optimal temperature ranges that explained why *L. sativae* is more widely distributed and causes more severe damage, while *L. huidobrensis* primarily outbreaks in moderate or relatively cool climates. Similarly, resistant mites persist in natural environments by presumably adopting ecological strategies that minimize risk and avoid limiting developmental rates at temperature extremes. By modeling the relationship between developmental rate and temperature, we can predict differences in risk developmental rates between resistant and susceptible populations, thereby identifying the extreme temperature ranges that support resistant populations and providing a theoretical basis for resistance management.

Furthermore, the evolution of pesticide resistance is primarily determined by resistance genes that are heritable across generations. The degree of inheritance is closely related to resistance heritability [11], and in many cases, resistance is a

quantitative trait controlled by one major gene and several minor genes. Quantitative genetic methods can therefore predict the rate and potential scope of resistance evolution. For example, Holloway [12] estimated the heritability (h^2 = 0.474 ± 0.294) of knockdown time to primiphos-methyl in the rice weevil (*Sitophilus oryzae*), indicating that approximately 47% of phenotypic variance in resistance was heritable. Meng Xiangqing et al. [13] found that the narrow-sense heritability of resistance to fenvalerate in the cotton bollworm (*Helicoverpa armigera*) (0.4625 ± 0.1578) was greater than that to cyhalothrin (0.2476 ± 0.0248), suggesting faster resistance development to fenvalerate. However, few studies have reported on the realized heritability and resistance risk assessment in mites, with only He Lin [14] and He Hengguo [15] investigating these parameters in *T. cinnabarinus* and *Panonychus citri*, respectively.

Pyridaben belongs to the mitochondrial electron transport inhibitor (METI) class of acaricides, which inhibit electron transfer at the coenzyme Q0 site. It is currently one of the primary acaricides used in China [16]. Due to its novel structure, unique mode of action, and broad-spectrum efficacy against spider mites, pyridaben has been widely applied in field conditions. However, prolonged and extensive use of this single compound has led to the development of resistance in *T. truncatus*. Therefore, estimating the realized heritability of resistance and assessing the resistance risk to pyridaben can provide a theoretical foundation for its rational use and application prospects in the field.

Materials and Methods

1.1 Test Mites

The susceptible population (SS) of *T. truncatus* was collected from experimental corn fields at Gansu Agricultural University that had never been exposed to pesticides. This population was maintained on potted bean seedlings in a climate-controlled rearing room for over 90 generations without any chemical exposure. The pyridaben-resistant population (Py-R) was selected in the insect laboratory of the College of Prataculture at Gansu Agricultural University, with a resistance ratio of 955.25 [17]. Rearing conditions were maintained at (24 ± 1) °C, 60% ± 5% relative humidity, and a photoperiod of L:D = 16 h:8 h.

1.2 Measurement of Developmental Periods and Development Rate-Temperature Relationships

Experiments were conducted in illuminated incubators using potted bean seedlings as host plants. Six temperature gradients were established: 16 °C, 20 °C, 24 °C, 28 °C, 32 °C, and 36 °C, with no pesticide application during the rearing period. Both susceptible and resistant populations were reared with 60 individuals each. A single female adult was placed on each bean leaf and removed after oviposition, leaving one fresh egg per leaf. The developmental period and survival status of individual mites were recorded daily until death.

Based on previous results regarding the effects of temperature on biological parameters of resistant and susceptible populations [18], developmental duration (N) was converted to developmental rate (V). The Wang-Lan-Ding model [9] was fitted to characterize the relationship between developmental rate (V) and temperature (T) for each developmental stage using the Marquardt damping least-squares iteration method [19]. The model formula is:

$$V = \frac{K}{1 + \exp\left(\frac{r(T-T_0)}{T-T_L} + \frac{r(T_0-T)}{T_H-T}\right)}$$

Derived from the Logistic model using asymptotic matching, this model is applicable across the entire developmental temperature range. In the formula, K represents the potential saturated developmental rate at high temperatures; r is the exponential growth rate of developmental rate with temperature; T_L and T_H are the minimum and maximum critical developmental temperatures, respectively; T is the experimental temperature; T_0 is the optimal developmental temperature; and represents the width of the boundary layer, with its relative magnitude reflecting different tolerance levels to extreme temperatures. Parameter estimation followed the methods described in reference [20].

1.3 Test Chemical and Resistance Selection

The test chemical was 15% pyridaben emulsifiable concentrate (Jiangsu Lanfeng Biochemical Co., Ltd.). A subset of individuals from the susceptible population was selected and continuously treated with pyridaben for 49 generations after propagation. Toxicity bioassays were conducted every 3–5 sprays using seven concentration gradients, with 30 mites per concentration and three replicates, to calculate the median lethal concentration (LC) and monitor resistance development trends [17].

1.4 Data Analysis

All data analyses were performed using SPSS 19.0 and Microsoft Excel. Realized heritability (h^2) and resistance risk were calculated using the following formulas.

1.4.1 Estimation of Realized Heritability (h^2) Realized heritability was estimated using the threshold trait analysis method of Tabashnik [21]:

$$R = \frac{\lg(\text{final } LC_{50}) - \lg(\text{initial } LC_{50})}{n} \quad (3)$$

$$S = i \times \delta_p \quad (4)$$

$$i = 1.583 - 0.0193336P + 0.0000428P^2 + \frac{3.65194}{P} \quad (10\% \le P \le 80\%) \quad (5)$$

$$\delta_p = \frac{1}{(b_1 + b_n)/2} \quad (6)$$

$$h^2 = \frac{R}{S}$$

where h^2 is the realized heritability; R is the selection response, representing the difference in mean phenotypic value between offspring and parent generations; S is the selection differential, representing the deviation of the parental mean phenotypic value from the population mean; n is the number of selection generations; i is the selection intensity; P = 1 - mean corrected mortality; _p is the standard deviation; b is the slope of the probit regression line for the parental generation; and b_n is the slope for the offspring after n generations of selection.

1.4.2 Resistance Risk Assessment From the formula R = [lg(final LC) - lg(initial LC)]/n, we derive R = lg(final LC /initial LC)/n. Based on realized heritability (h^2), the number of generations required for resistance to increase by x-fold can be predicted as G = lgx/(h^2S). Following reference [14], when selection produces a 10-fold increase in resistance (final LC /initial LC = 10), the required number of selection generations is G = lg(10/R).

Results and Analysis

2.1 Simulation of Developmental Rate-Temperature Relationships in Susceptible and Resistant Populations

The Wang-Lan-Ding model was fitted to characterize the relationship between developmental rate (V) and temperature (T) for eggs, larvae, nymphs, and complete generations in both susceptible (SS) and pyridaben-resistant (Py-R) populations of *T. truncatus*. Model parameters are presented in Table 1 , and fitted curves are shown in Figure 1 [Figure 1: see original paper] (nymph parameters could not be simulated and are therefore omitted).

The results indicate that the minimum and maximum critical temperatures for generation development in the susceptible SS population were 10.05 °C and 39.24 °C, respectively, with an optimal developmental temperature of 25.73 °C. For the resistant Py-R population, the corresponding values were 13.45 °C and 41.89 °C, with an optimal temperature of 26.04 °C. The maximum critical temperature for each developmental stage was significantly higher in the resistant population, demonstrating that the pyridaben-resistant population possesses substantially greater adaptability to high temperatures than the susceptible population. However, the resistant population also required a significantly higher minimum temperature for development, indicating weaker adaptability to low temperatures. These modeling results are consistent with experimental observations.

2.2 Realized Heritability of Resistance to Pyridaben

As shown in Table 2 , the realized heritability of resistance to pyridaben across the entire selection period (F –F) was 0.11. During the early selection phase (F –F), h^2 was 0.12; during the middle phase (F –F), it reached 0.18, the highest value observed; and during the late phase (F –F), it declined significantly to 0.08. At the final selection stage (F –F), h^2 rebounded to 0.14. These data demonstrate that field populations of *T. truncatus* harbor resistance genes, and the substantial proportion of phenotypic variance attributable to genetic variation (h^2) facilitates rapid resistance development. However, as selection progressed, susceptible genes were gradually eliminated and resistance genes stabilized within the population, causing the proportion of genetic variation in phenotypic expression to decline and slowing resistance development, as reflected by the decreased h^2.

Realized heritability reflects the degree to which a trait is transmitted from one generation to the next. Among the many factors influencing phenotypic value, only additive genetic value is fixed and heritable, and thus only this component affects traits in subsequent generations. Narrow-sense heritability (h^2) is therefore defined as the ratio of additive genetic variance to total phenotypic variance. As indicated in Table 3 , higher selection pressure and greater realized heritability result in faster resistance development.

2.3 Resistance Risk Assessment of *T. truncatus* to Pyridaben

Assuming pyridaben mortality rates of 50%, 60%, 70%, 80%, and 90%, we predicted the number of generations required for a 10-fold increase in resistance (assuming a probit regression slope of 2.0, i.e., _p = 0.5, which approximates the slopes observed before and after selection) [22–23]. Table 3 shows that under selection pressures corresponding to 50%–90% mortality (selection intensity P = 10%–50%), only 10–23 generations are needed for resistance to increase 10-fold. Given that laboratory selection environments are relatively stable and exhibit lower environmental variance than field conditions, the estimated heritability from laboratory selection ($h^2 = 0.11$) may overestimate actual field values. Assuming field heritability is half the laboratory value ($h^2 = 0.05$), the same level of resistance would still require only 21–46 generations, indicating a substantial resistance risk.

Conclusion and Discussion

This study fitted the Wang-Lan-Ding model to characterize the relationship between generation developmental rate and temperature in susceptible and resistant populations of *T. truncatus*. The results demonstrate that the minimum and maximum critical temperatures for generation development were 10.05 °C and 39.24 °C for the susceptible SS population, and 13.46 °C and 41.89 °C for the resistant Py-R population. In the central Hexi Corridor of Gansu Province (primarily Zhangye City), the annual mean temperature ranges from 6.0 °C to 14.3

°C, with extreme maximum temperatures reaching 40.0 °C. These conditions align with the extreme temperature ranges simulated for both populations and corroborate previous findings that the pyridaben-resistant population exhibits biological fitness advantages at high temperatures [18]. The enhanced adaptability of field-resistant populations to high temperatures, coupled with superior survival and reproductive capacity compared with susceptible populations, explains why *T. truncatus* frequently outbreaks during hot, dry seasons and proves difficult to control.

He Lin [14] previously applied the Wang-Lan-Ding model to characterize developmental rate–temperature relationships in susceptible and fenpropathrin- and abamectin-resistant strains of *T. cinnabarinus*. That study found no difference in maximum critical temperature between the fenpropathrin-resistant strain (36.1 °C) and the susceptible strain (36.0 °C), whereas the abamectin-resistant strain exhibited a substantially higher maximum critical temperature (42.4 °C) and greater adaptability to high temperatures. These results suggest a potential link between the development of pesticide resistance and enhanced tolerance to high temperatures in spider mites. We hypothesize that after developing resistance, mites adopt ecological strategies that minimize risk by avoiding limiting developmental rates, thereby increasing their adaptability to high temperatures.

The specific mechanisms underlying enhanced heat tolerance in resistant insects remain unclear, though research has focused on heat shock proteins and changes in protective enzyme activities (SOD, CAT, POD) [24-25]. Whether resistance to pyridaben in *T. truncatus* is associated with increased expression of heat shock proteins or altered protective enzyme activities, thereby conferring improved high-temperature adaptability, warrants future investigation.

Using threshold trait analysis, we estimated the realized heritability of pyridaben resistance in *T. truncatus* at $h^2 = 0.11$, indicating a significant resistance risk. However, heritability estimates from laboratory selection may differ from field conditions. The realized heritability value can be influenced by several factors: uneven pesticide exposure among test individuals, differential sublethal effects in survivors, and unequal selection between sexes, all of which may lead to under- or overestimation. Additionally, laboratory selection under constant temperature and photoperiod conditions tends to underestimate environmental variance. In field settings, resistance development may be delayed by emigration of resistant individuals, immigration of susceptible individuals, environmental variation, alternating or rotating pesticide use, and gene mutation, potentially resulting in lower realized heritability [26]. Consequently, multiple factors can lead to under- or overestimation of heritability.

Assuming field heritability is half the laboratory value ($h^2 = 0.05$) and selection pressure corresponds to 50%-90% mortality (selection intensity P = 10%-50%), a 10-fold increase in resistance would require 21–46 generations—approximately double the laboratory estimate of 10–23 generations. Although laboratory results contain inherent errors and cannot be directly applied to field conditions,

they provide valuable guidance for timely resistance monitoring and proactive resistance management strategies. Based on these findings, *T. truncatus* exhibits a high risk of developing resistance to pyridaben. We recommend limiting the number of pyridaben applications in field management and implementing rotation with insecticides lacking cross-resistance to reduce selection pressure and delay resistance evolution.

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Note: Figure translations are in progress. See original paper for figures.