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Research Overview on Occurrence and Control of *Tetranychus urticae* Koch in China

Yang HAN, Tingmi YANG, Jingcheng WU, Qiuling PANG, Xiaowen LIANG, Binghai LOU *

Guangxi Academy of Specialty Crops, Guangxi Laboratory of Germplasm Innovation and Utilization of Specialty Commercial Crops in North Guangxi/Guangxi Citrus Breeding and Cultivation Technology Innovation Center, Guilin 541004, China

Abstract This paper elaborates the occurrence factors and damage characteristics of *Tetranychus urticae* Koch in China, and emphatically summarizes three main control strategies of *T. urticae*, namely agricultural control, chemical control and biological control, as well as research progress in its resistance mechanisms. The problems existing in controlling *T. urticae* and its resistance management strategies are put forward, to provide a theoretical basis for the resistance management and comprehensive control of *T. urticae*.

Key words *Tetranychus urticae* Koch; Insecticide resistance; Chemical control

1 Introduction

Tetranychus urticae Koch, belonging to *Tetranychus*, Tetranychidae, is a worldwide pest mite mainly distributed in temperate and subtropical regions around the world[1]. There are more than 200 species of host plants of *T. urticae*, and it can seriously reduce the production of field crops, fruit trees and vegetables[2]. *T. urticae* has fast reproduction rate and severe overlapping generations due to short generation cycle. It mainly adopts the reproductive mode of sexual reproduction, but also can camp parthenogenesis, and its parthenogenesis offspring are all male mites.

2 Occurrence

The generations and peak periods of *T. urticae* vary among different regions and climatic conditions. Generally, it occurs 10 – 20 generations a year (increasing gradually from north to south), and their overwintering sites and insect states are also different among regions. In North China, it survives the winter as female adult mites in weeds, litters and fallen leaves. In central China, most of them overwinter in weeds in various insect states[3]. When the temperature reaches more than 10 ℃ in March and April of the next spring, *T. urticae* begins to reproduce massively. The optimum temperature for reproduction is 25 – 30 ℃ and the optimum relative humidity is 35% – 55%. Therefore, it is easy to break out in June and July when the temperature is high and the relative humidity is low, causing serious harms[4]. When the temperature is higher than 30 ℃ and the relative humidity is greater than 70%, it is not conducive to its growth and development, and the number of insects decreases in summer when the temperature and humidity are high. In addition, heavy rainfall in summer also inhibits the occurrence of *T. urticae*[5].

The damage of *T. urticae* in China was first reported in the 1980s. In 1983, *T. urticae* was found to damage flowers and plants such as *Salvia splendens* in Beijing[6]. Later, *T. urticae* was found successively in Tianshui in Gansu Province, Zhaoyuan and Linyi in Shandong Province, and Changli in Hebei Province, causing serious damage. Since 1990, *T. urticae* has caused serious damage in apple producing areas in China, and the damage has further spread and aggravated, resulting in huge economic losses[7]. In 1991, Pu Chunshu *et al.*[8] carried out a preliminary investigation on *T. urticae* in Tianshui area of Gansu Province, and the results showed that *T. urticae* caused damage by breeding on more than 50 species of fruit trees, vegetables, field crops, flowers and weeds around apple orchards. The census results of Meng Wei *et al.*[9] showed that in 1997, there were different degrees of *T. urticae* occurrence in orchards in southern and western Liaoning Province. In recent years, Landeros *et al.*[10] and Gough[11] reported the damage of *T. urticae* in Mexico, Australia, India, Japan and other countries.

3 Distribution and damage

T. urticae mainly distributes in the northern United States, Australia, France, Italy, South Africa, New Zealand, Japan and other places abroad, and it is a worldwide pest mite[12]. Since the first report on the harm of *T. urticae* in China in 1983, it has been found throughout the country, especially in northern China, where there are many host species[13].

T. urticae mainly causes damage by nymph and adult mites gathering on the back of leaves to absorb sap. After being fed by *T. urticae*, the leaves will first appear pale spots on both sides of the main vein. With the aggravation of the damage, the leaves will turn grayish white, making the plants brittle and hard, and inhibiting the normal photosynthesis[14]. In addition, *T. urticae* can also release toxins or growth regulating substances, causing plant growth imbalance[15].

T. urticae is featured by small individual size, strong adapta-

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* Corresponding author. E-mail: loubinhai000000@163.com

bility, fast reproduction rate, strong stress resistance, and mixed feeding habits[16], and there are many host species. It has been reported that more than 200 species of plants belonging to more than 50 families are harmed by *T. urticae*, which is a kind of polyphagous mite, damaging many fruit trees such as apple, pear, peach and apricot, as well as many crops such as cotton, bean and corn[17]. In recent years, the mite has spread and harmed in the northern fruit area, with a trend of gradual development.

4 Control

Because of its small size, rapid reproduction rate, short generation cycle, both bisexual and parthenogenetic reproduction, it is particularly difficult to control *T. urticae*. At present, chemical control is still the main method for field control of *T. urticae*, and comprehensive control measures combining biological control and physical control are adopted simultaneously[18]. However, in the future, biological control should be the dominant method to gradually restore the damaged ecosystem and give full play to the control role of natural enemies on *T. urticae*[19].

4.1 Agricultural control Agricultural control of *T. urticae* is mainly to reduce the number and alleviate the damage of *T. urticae* to various crops through agricultural operations[20]. First of all, weeds should be uprooted on the edge of the field regularly, and the residual plants and leaves should be removed to reduce the environment and places suitable for the survival of *T. urticae*. The overwintering sites of harmful mites are destroyed and overwintering female adult mites are eliminated by timely pruning branches and leaves during spring and autumn ploughing, cutting short tillering on branches, timely cleaning up the litters on the edge of the field, scraping off the rough barks of branches and warping the seams, removing dead branches, fallen leaves and weeds in the garden and burning them centrally, so as to reduce the initial quantity of mites in the following year and control the number of *T. urticae* population from the source[21]. In addition, crop rotation should be carried out in the field. It is easy for *T. urticae* to multiply in large numbers when planting a single crop for a long time. Regular rotation and tillage can also control the population of *T. urticae* by destroying their living environment and sites, making it difficult for *T. urticae* to break out in large quantity[22]. Finally, starting from the optimum temperature and humidity, too low or too high temperature and too high relative humidity is not conducive to the growth, development and reproduction of *T. urticae*. Therefore, in the actual production process, it can effectively inhibit the growth, development and reproduction of *T. urticae* by more watering and irrigation to maintain the relative humidity in the environment. Meanwhile, the water and fertilizer management of crops should be strengthened to ensure the healthy and robust growth of crops, which can also reduce the harm of *T. urticae*[23].

4.2 Chemical control Chemical control refers to the control by chemical agents, featured by quick effect, high efficiency, convenient use and low economic cost, and it is the main means for the prevention and control of *T. urticae* at the present stage[24].

Acaricides developed from calcium arsenate and sulphur in the earliest era of inorganic acaricides to organic synthetic acaricides dominated by nerve agents in the middle 20th century, but organophosphorus insecticides not only prevented and controlled mites but also killed their natural enemies in large quantity[25]. Since the 1970s, acaricides had been developed to inhibit respiratory metabolism of mites. In the 1990s, abamectin, the most representative insecticidal and acaricidal agent, was developed, and at the same time, the fungicide fluazinam was also used to control all kinds of harmful mites due to its acaricidal activity[26]. In the 21st century, many acaricides with heterocyclic structure have been developed, and these acaricides have higher activity and longer duration, such as etoxazole, a mite growth regulator that kills both phytophagous mites and mite eggs, and bifenazate, which has no cross-resistance with existing acaricides. In recent years, acaricides have been developed to the direction of heterocyclic acaricides that inhibit the respiratory metabolism of mites[27]. However, due to the large and frequent use of various acaricides, *T. urticae* has gradually developed different degrees of resistance to various acaricides[28]. Therefore, while paying attention to the development of new agents, chemical control should also pay close attention to the dynamic change of the resistance of harmful mites to existing acaricides. Acaricides should be used scientifically and rationally, rotated and mixed. In areas where there is a high level of resistance to certain acaricides, the use of such acaricides should be suspended to avoid further development of resistance and extend the service life of the agents[29].

In China, there are also many reports on the use of acaricides to prevent and control *T. urticae*. Acaricides have such functions as contact toxicity, stomach toxicity, systematic absorption and fumigation on *T. urticae*. Most commonly used acaricides mainly have contact and stomach toxicity, which can affect harmful mites through direct contact. Dai Aimei *et al.*[30] found that the control effect of a new acaricide, 30% cyetpyrafen SC 2 000 times dilution, on *T. urticae* reached 66.21% at 1 d post administration, and the control effect remained above 85% at 14 d post administration, showing good durability. Hong Yingxue *et al.*[31] showed that most of the tested chemicals had unsatisfactory control effects on *T. urticae* in Aksu and Shangqiu areas. This also reminds us that in actual production, it is necessary to seize the best application period and not rely on a certain type of agents, and should be mixed reasonably and used agents alternately with different mechanisms of action.

4.3 Biological control Biological control is the safest and most environmentally friendly means of control, but due to slow effect, high economic cost and unstable control effect, biological control is still in the development stage. Biological methods for the prevention and control of *T. urticae* have also been reported in China. Since 1983, when *T. urticae* was found to damage *S. splendens* in China, Dong Huifang *et al.* [32] began to study the application of *Phytoseiulus persimilis* to control *T. urticae* on flowers. The test results showed that the application of *P. persimilis* was effective in preventing and controlling *T. urticae* for a long time. Sun Yuehua *et al.* [33] found that *Amblyseius fallcis* had a large predation on *T. urticae*, and the population growth rate of this predatory mite was fast, which had a good development prospect. Qiu Xiaohong *et al.* [34] found that predator *Amblyseius cucumeris* could be used to prevent and control *T. urticae*, but more studies on the use of natural enemies for predation are still in the experimental stage. Zhang Xiaona *et al.* [35] also reported that plant-derived products, namely nutrients and secondary metabolites produced by different host plants of *T. urticae*, could be used to affect the normal growth and development of the mite. It can also achieve the purpose of preventing and controlling *T. urticae* by microbial poisoning. There are also many research reports on the biological control of harmful mites abroad. From 1987 to 1988, James *et al.* [36] monitored the populations of *T. urticae* and *Amblyseius victoriensis*, a predatory mite, in peach orchard in southern New South Wales, Australia, and found that *A. victoriensis* had a strong predatory effect on *T. urticae*. Garcia-Mari *et al.* [37] found that *Neoseiulus califonicus* preyed on phytophagous mites in strawberry gardens in Valencia, Spain, and can be used to control *T. urticae*.

Although the biological control of *T. urticae* has made good progress, most of the biological control methods are still in the experimental stage, and there are still some problems in control measures. For example, *P. persimilis* has high requirements on temperature and relative humidity conditions. Only when the relative humidity reaches 90% can all eggs hatch, while all eggs can not hatch when the humidity is less than 50% [38].

5 Status of resistance and resistance mechanism

5.1 Status of drug resistance of *T. urticae* It has only been a few decades since 1983, when China first discovered the harm caused by *T. urticae*. However, at present, *T. urticae* has occurred in most parts of China, with extremely fast development and diffusion[39]. At present, *T. urticae* is mainly controlled by chemical methods, and its resistance to various chemical agents is also developing very fast. At present, many efforts have been dedicated to the resistance of *T. urticae* at home and abroad. As early as the early 1990s, Tian *et al.* [40] found that the population of *T. urticae* in California pear garden had medium and high levels of resistance to cyhexatin and fenbutatin oxide. Sato *et al.* [41] reported that the *T. urticae* population in Sao Paulo, Brazil had developed more than 600 times resistance to avermectin. Khajehali *et al.* [42] found that the resistance ratios of *T. urticae* to chlorpyrifos and dimethoate in Belgium were 586 and 743 times, respectively. Subsequently, it was found in 2014 that the MR-VP *T. urticae* population in Belgium developed cross-resistance to cyenopyrafen and cyflumetofen. Wang Kaiyun *et al.* [43] figured out that the *T. urticae* population in Yantai, Shandong Province had developed a moderate level of resistance to pyridaben. Zhao Weidong *et al.* [44] showed that the *T. urticae* population in Yantai and Shouguang areas of Shandong Province had developed a moderate level of resistance to isocarbophos, fenpropathrin and pyridaben. Liu Qingjuan *et al.* [45] measured the resistance of *T. urticae* to 7 kinds of acaricides by slide dip method, and found that the tested *T. urticae* population had a high level of resistance to pyridaben, with a resistance ratio of 105.47 times, and showed a resistance ratio of 5.45 times to fenpropathrin. In 2016, Wang Ling[46] monitored the resistance level of different populations of *T. urticae* to insecticides by means of drug tube leaf dipping method, and the results showed that all the 6 populations monitored had a high level of resistance to avermectin, and the population in Yuncheng, Shanxi had a medium level of resistance to bifenazate. In 2019, Xu Dandan[47] monitored the resistance of *T. urticae* populations by agar leaf leaching method, and found that the populations in Harbin, Heilongjiang, and Jiyang, Hainan had extremely high levels of resistance to abamectin (up to 1 809.51 times), and most of the populations had moderate levels of resistance to cyflumetofen and cyenopyrafen.

5.2 Resistance mechanism With the increasing resistance of *T. urticae* to various acaricides, more and more scholars are committed to studying the mechanism of resistance of *T. urticae*. At present, the mechanism of resistance can be divided into three aspects: reduced penetration rate, enhanced metabolic ability of detoxification enzymes and insensitive target.

5.2.1 Reduced penetration. At present, most of the commonly used acaricides on the market play a control effect by killing harmful mites through contact. To be effective, the drug must pass through the epidermis of mites, whereas the reduction of penetration effect on the one hand is that the penetration rate of the epidermis is reduced, so that the drug dose reaching the target site in the same time will be reduced, thus improving the resistance of the harmful mite. Insects can thicken the epidermis by increasing the synthesis of epidermal proteins, thereby reducing the penetration rate of agents[48]. On the other hand, the sensitivity of target organs of insects to agents decrease, which will reduce the absorption of agents, thus enhancing the resistance of pests.

5.2.2 Metabolism of detoxification enzymes. The rise of some enzymes in the insect body leads to the enhancement of insect metabolism, which accelerates the detoxification metabolism of pests to insecticides. Insect detoxification enzyme is a heterogeneous enzyme system capable of metabolizing a large number of endogenous or exogenous substrates. The main enzymes involved in insect metabolic resistance include mixed function oxidases (MFOs), carboxylesterascs (CarE) and glutathione-S-transferases (GSTs), which reduce pesticide toxicity by enhancing the transformation and degradation of pesticides or protect themselves by blocking[49]. Carboxylesterase is one of the important detoxification enzymes in insects, and its enhanced detoxification activity is an important mechanism of mite resistance to organophosphorus and carbamate insecticides. He Lin *et al*.[50] found that the GSTs of *Tetranychus cinnabarinus* in abamectin resistant strains was 3.4 times of that in sensitive strains, suggesting that significantly increased detoxification enzyme activity is an important cause of abamectin resistance in *T. cinnabarinus*. Liu Qingjuan *et al*.[51] put forward that the increased activity of GSTs is one of the reasons for the resistance of *T. urticae* to fenpropathrin. Yang Ming[52] tested the synergistic effects of multifunctional oxidase inhibitor PBO, oxidase and esterase inhibitor SV, and esterase inhibitor DE on bifenthrin by using the sensitive and resistant strains of *T. urticae*, and the results showed that the increased activity of multifunctional oxidase inhibitor PBO is the main mechanism of resistance to bifenthrin in *T. urticae*.

5.2.3 Decreased target sensitivity. Acetylcholinesterase is the target of organophosphorus and carbamate insecticides, and the target site of DDT and pyrethroids is voltage-gated sodium ion channel. When the sensitivity of acetylcholinesterase and voltage-gated sodium ion channel to various agents decreases, the effect of these agents on pests will decrease, and the resistance of pests will increase[53]. The decreased sensitivity of the target on the one hand is attributed to that mutation of target has changed its structure, thus reducing the binding ability of the pesticide and the target. By sequencing the mitochondrial genome (mtDNA) of sensitive and resistant populations to bifenazate, Van *et al*.[54] proved that diphenylhydrazine resistance was inherited maternally, and mutation sites G126S, I136T, S141F, D161G and P262T mutation were found on cytochrome b (cytb) cd1 pocket and ef helix, which were confirmed to be associated with resistance to bifenazate. Subsequently, Van *et al*.[55] confirmed that cytb was indeed the target site of bifenazate. Through high-throughput gene sequencing and other technologies, Van *et al*.[56] also revealed that the resistance of *T. urticae* to etoxazole was monogenic and recessive, and identified the target site of etoxazole as chitinase synthetase 1(CHS1). Demaeght *et al*.[57] also reported that I1017F mutation on CHS1 gene was associated with etoxazole resistance, and produced cross-resistance with clofentezine and hexythiazox. On the other hand, the target expression had changed, which affected the binding affinity of the target with insecticide[58]. Meng Hesheng *et al*.[59] declared that the increase of acetylcholinesterase activity in *Panonychus citri* is one of the main reasons for its resistance to pyridaben. Khajehali *et al*.[60] found that the mutations of A201S, T280A and F331W are the main reasons for the resistance of *T. urticae* to organophosphorus insecticides. Meantime, Kwon *et al*.[61-62] analyzed the correlation between the multiple of resistance to monocrotophos and the frequency of gene mutation, and found that G119S played an important role in resistance of *T. urticae*. Subsequent *in vitro* functional verification demonstrated that single mutations of F331W and G328A could lead to moderate resistance to monocrotophos, and the cumulative effect of these two mutations with A280T and G119S could lead to higher resistance.

6 Problems and prospects

As a worldwide pest mite, *T. urticae* has been harmed in China for nearly 40 years, causing huge economic losses to economic crops such as vegetables and flowers. For a long time, the use of chemical agents is the main way to control the damage of *T. urticae*. However, due to its rapid reproduction, short generation cycle, strong stress resistance, and long-term continuous single, frequent and unreasonable use of chemical agents, *T. urticae* has produced different degrees of resistance to common acaricides. Due to the differences in genetic background and application level of hosts in different regions, the development laws of *T. urticae* resistance to acaricides are also different. In order to solve the problem of increasing resistance of *T. urticae*, the development of resistance can be delayed by the following measures.

(i) Strengthening the monitoring of resistance of *T. urticae*. By monitoring the resistance of *T. urticae*, people can know the current status of its resistance to common acaricides promptly, which is very important for resistance control. It is necessary to strengthen the monitoring of resistance of Chinese vegetables, flowers and fruit trees to *T. urticae*, and clarify the resistance status of these *T. urticae* populations to commonly used acaricides, which can provide scientific guidance for its control and has important practical significance for ensuring the quality production of vegetables and flowers[63].

(ii) Risk assessment of the resistance of *T. urticae* to novel acaricides. It is necessary to evaluate the resistance risk of new acaricides before they are widely used. New acaricides can be used more efficiently and consistently by clarifying the resistance risk of *T. urticae* to these acaricides. The resistance risk assessment can provide a theoretical basis for the formulation of a reasonable resistance management strategy, and lay a foundation for future research on the genetic mechanism of resistance.

(iii) Strengthening the study on the resistance mechanism of *T. urticae* to insecticides. Bioassay of synergist and enzyme activity test can be used to determine the metabolic resistance of detoxification metabolites in *T. urticae* to various insecticides. Transcriptomic sequencing technology can be used to compare the differences in expression patterns of detoxification enzyme genes at the molecular level, and to understand the changes in detoxification metabolism of *T. urticae* after producing resistance to various insecticides. It is of great significance for field resistance management and development of new insecticides by exploring the resistance mechanism of *T. urticae* to various insecticides[64].

(iv) Strengthening the green and biological control of *T. urticae*, and establishing a comprehensive control system. The prevention and control of *T. urticae* should focus on more efficient and environmentally friendly green prevention and control, strengthen the research and development of its biological control, and develop biological pesticides with lower resistance risk to ensure the population of its natural enemies. From the prediction and early warning of *T. urticae* to the green and efficient prevention and control, a mature control system has been established to ensure the sustainability of the prevention and control of *T. urticae* in China.

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