Study on the Pathogenic Conditions and Mechanisms of Yunnan Tea Moire Leaf Blight

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Abstract: Tea leaf blight is a disease of high humidity and high humidity. The pathogenicity, characteristics, infection cycle, occurrence regularity and dangerous symptoms of the disease were introduced. It was pointed out that proper topography, topography and soil should be selected in agricultural management, disease-resistant varieties should be used, and health of tea plantations should be paid attention to. Strengthen the management of teahouse and select appropriate medicines as soon as possible. It provides a scientific basis to control the damage of tea clouds as soon as possible.

1. Introduction

Leaves of tea clouds withered, mainly damaging leaves, new buds, branches and fruits. When the tea plant is sick, its leaves often fall off quickly, and it looks as if the new buds are dead. Then, weaken the tree. Tea blight and trees are weak. After flowering, the tea garden is more intense and the autumn harvest seedlings are more frequent[1]. If the tea bowl is too full, it will show golden tea, and the young trees will wither. In addition to damaging tea plants, diseases can also damage toon, toon, tea and other plants. In the former Soviet Union, Japan, Sri Lanka, China, Bangladesh, China, Vietnam, Mara Thea and other countries, there are foreign countries, and China's tea producing areas and autonomous regions are also distributed. The mechanism of pathogen and disease of tea leaf blight was studied, and the synchronous fixed point integrated control technology was proposed.

2. Pathogens and Characteristics

The Cooke period of Yunnan Pinus bungeana leaf blight is short stem stage, belonging to Ascaris, Ascaris, cysticercus and black rot fungi[2]. The asexual period is Camellia sinensis, a true genus, Helicobacter pylori, black Cyclospora, black cyclosporaceae, anthrax.

2.1. Form

The small black spots on the lesion were mainly the conidial disc of the pathogen. Conidia discs are scattered under the host epidermis. At maturity, the epidermis ruptures, producing a large number of conidia. The conidial disc is round and its diameter ranges from 187.0 to 290.0 um. The base is thin and flat, forming an accessory seat with conidia and setae. Conidia, branchless, colorless, with size (9.0-19.0) um × um (3.0-3.5), and an apical conidia. Conidia are long elliptical cylinders with round ends or slightly thin ends, straight or slightly curved, single-celled, smooth, colorless, and in 1 or 2 oil balls of size (10.0-21.0) um × um (3.0-6.0). Most buds split from the middle and become twins. Hard-leaved spores are spherical to oval in shape and light brown in outer membrane. There are two or three oil balls. Rigid hair is acicular, base thicker, tip thinner gradually, dark brown 1-3 separation. (40.0-70.0) um × um (3.0-5.0). Sexual reproduction is rare and sometimes produces ascospores. Ascomycetes scattered across the banks of the disease, semi-underground in the host tissue, black, flat spherical, 160.0 - 200.0 um in diameter, membrane
and circular hole diameter 7.0 - 18.0 um. The embryo sac is rod-shaped to oval, with round apex and small stems at the base. Size (40.0-66.5) um × um (9.0-18.0). Among them, 8 ascospores were arranged in two rows. The ascospores are spindle-shaped ellipse, unitary, colorless, with a size of 10.0-18.0 um × um (3.0-6.0).

2.2. Features

The optimum temperature for the growth and development of pathogenic bacteria is 27.0-29.0 C and the highest temperature is 37.5 C. Low temperature and high temperature have strong tolerance to pathogenic bacteria. They survive for 30-60 days at low temperature of - 2-4 and the lethal temperature is 10-50 C.

2.3. Conditions of onset

The average temperature in the first half of the field is 27 to 29 degrees c, the rainfall is more than 40 mm, and the average relative humidity is over 80%. The occurrence and progress of diseases make the greatest contribution[3]. If the temperature is high and dry, the disease will develop slowly, but in the dry summer season, in the strong sunshine, the water of the tea plant can not keep balance, and the leaves become dry. If you take a shower after tanning, illness is very popular. If the weather is dry, continuous rain or sunny, the disease will develop rapidly. The peak incidence usually occurs after 10 to 15 days of rainfall. In the tea growing area, the rainy season from April to June and the autumn of September to November are the seasons of this disease. In addition, tea plantations with bad environment and poor cultivation and management are also vulnerable to the disease. Tea plantations with high groundwater level, poor drainage and shallow water and soil layers are afraid of freezing and dry water because of the development of roots[4]. Wide management, not timely planting and weeds, high planting efficiency, insufficient fertilizer and partial application of nitrogen fertilizer, close planting, vulnerable tea plants are vulnerable to disease. Vehicle tree species have different resistance to this disease. Generally speaking, Southern varieties are more susceptible than northern varieties, while large-leaved varieties are less susceptible to leaf types, and high-quality varieties are also more susceptible.

3. Harmful Symptoms

The wilting of tea clouds is mainly harmful to leaves, but other parts of the tea tree, such as new branches, branches and fruits, may also be infected.

3.1. Leaf disease

Leaf and leaf lesions mostly occur at the edge or front of the leaf. In the early stage, the lesions are yellowish-brown, semi-circular or light green and gradually enlarged. The color is dark brown or brown. A week later, the lesion grey from inside to outside. The organization is dead, the edge is yellow and green. Form irregular patches of grey, dark brown and dark brown. In the late stage of leaf blight, there are small round gray-black spots, i.e[5]. the fruit bodies of pathogens (spore mounds are reddish-brown, irregularly arranged, and the lesion parts are rotated) (Fig. 1). The lesion is yellowish brown on the back. The lesion is the vine and the whole leaf. Finally, the leaves are dried off after 25 to 50 days of symptoms (Fig. 2). Young leaf lesions are initially round brown, followed by dark brown (Fig. 3).
3.2. Disease of Branches and Fruits

Branches produce gray-brown patches, elliptical slightly concave, with small gray-black particle spots, often withered branches. Fruit lesions are yellowish-brown or gray, round, with small gray spots on the surface and sometimes cracks. When the tea plant is weak, it will produce small pathological changes, not plasticized, gray-white, scattered black spots in the front.
4. Pathogenesis

Plants are infected by pathogens during their growth and development, which is a biological pressure. Pathogens use their own secreted enzymes and toxins to achieve the purpose of infection, but plants resist it through corresponding mechanisms[6]. Disease tolerance of plants is related to their morphological structure, physiological and biochemical characteristics. Plant disease resistance factors are generally divided into three types.

4.1. Structural factors of plant disease resistance

Structural tolerance factors mainly refer to plant morphology, organ morphology, plant surface keratin and cell wall thickness and composition, whose function is to restrict and hinder the invasion of pathogens. Plant morphology that does not promote the retention of spore droplets and water droplets of pathogens can protect plants from diseases to a certain extent[7]. Many pathogens invade the host only through stomata. Xiaochun first reported on the susceptibility of corn cultivar Xuegan cake rust and the number of stomata on the leaf surface.

Gels, invaders and plugs induced by pathogenic bacteria can increase plant resistance to pathogenic bacteria. The sedation of steroids, hydroxypropyl protein and lignin can reduce the growth rate of pathogens. Local tissue necrosis near the infection site affects the movement of pathogens and the acquisition of plant nutrients.

The cuticle is the outermost layer of the plant surface. It can not only resist the invasion of pathogenic bacteria, but also inhibit the spore germination of some fungi. The waxy layer has the effect of alleviating and alleviating the disease[8]. Pathogens must attach the host to its surface and germinate to form reproductive ducts. This step usually requires the host to have a wet surface.

4.2. Biochemical factors of plant disease resistance

Biochemical factors mainly include two types, one is the inhibition of metabolic ability of pathogenic bacteria, the other is the direct killing of pathogenic bacteria. The former mainly refers to plant degrading enzyme inhibitors contained in plants, while the latter includes plant antitoxins, phenolic compounds and enzymes capable of degrading the cell wall of pathogens. In addition, many plants also produce a large number of other structural fungicides, such as hydrogen cyanide, saponins and tannins. In the process of disease resistance, the activities of phenylalanine ammonia lyase, cinnamyl alcohol dehydrogenase, polyphenol oxidase, peroxidase and other enzymes related to lignin synthesis in plants increased[9]. The activities of peroxidase dismutase, catalase and other protective enzymes in vivo also increased.

Phenols are important secondary metabolites of plants. They participate in many important physiological processes. When studying the natural resistance of plants, it was found that more phenolic substances were accumulated in the infected parts of resistant plants. The main phenolic substances are cinnamic acid, chlorogenic acid and protocatechuic acid. Many studies have shown that the content of phenolic substances in plants increases after the pathogen infects them. This phenomenon is particularly evident in disease-resistant plants. The total phenolic content of coffee trees growing in two years after rust infection increased rapidly. The content of phenolic compounds, chlorogenic acid and ferulic acid increased significantly in cotton seedlings infected with sickle stone. The content of catechin in resistant cotton seedlings was higher than that in susceptible varieties. After inoculation, the content of catechin in diseased plants was significantly higher than that in healthy plants, and the increase of catechin content in resistant varieties was significantly higher than that in susceptible varieties. Catechin inhibited mycelial growth, sporulation and spore germination of Fusarium, and inhibited the activities of most lactase and pectin lyase of Fusarium.

The gossypol content in cotton was positively correlated with the resistance of cotton varieties to Verticillium wilt. The gossypol content of resistant varieties was higher than that of resistant varieties[10]. The gossypol content of resistant varieties was higher than that of susceptible varieties. The accumulation rate of gossypol in resistant varieties was higher than that in susceptible varieties.

The accumulation of chlorogenic acid in resistant Poplar Cultivars was three times higher than that in susceptible poplar cultivars, and the increase of chlorogenic acid was consistent with that of
resistant poplar cultivars.

Lignin is a bridging molecule synthesized from a variety of styrene-propylene monomers. It is often associated with cellulose and other sugars in the cell wall. It attaches to the wall and forms corks to prevent pathogen infection and transmission. In the pathogen-host plant relationship, the accumulation of Li Guning, the host plant, is an active response to pathogenic organisms. Increase in the content of Lignin in plants infected by pathogenic bacteria is a common phenomenon. From the relationship between several biochemical substances in Huata and resistance to CFC disease, the interaction between cucumber and charcoal disease, cucumber and shellfish disease, and disease resistance showed a positive correlation between the storage rate and quantity of Riegnin. Lycopene is widely distributed in the plant kingdom of lycopene, lycopene, lycopene, oatmeal and scallion. In addition to antimicrobial pathogens, these saponins also inhibit spore germination of most fungi, but inhibit mycelial growth is very fragile.

Plant antibiotics are low molecular weight antimicrobial substances synthesized and accumulated in plants when attacked by biological or abiotic factors. It has antimicrobial effect. After the invasion of pathogens, tissue necrosis plaques were formed around the infiltrating sites, which hindered the proliferation of pathogenic bacteria and inhibited the expansion of lesions. The formation of phytalexins in non-infected cells is related to the production efficiency and storage capacity of phytalexins, which hinder the proliferation of pathogens. The storage of starch resistance in plants has the following characteristics: Resistant plants and edible plants can also accumulate plant anti-fibrin, but the formation of plant anti-fibrin in resistant plants is very fast, reaching its peak at the initial stage. At this time, plant cells can appear local necrosis or browning allergic reactions. The accumulation of starch resistance in plants is very slow. After several days of pathogen growth, the starch-resistant components of plants can reach a high concentration, but at this time, the mycelium begins to form spores. Phytobactericides do not work at all.

Plant aniline is trapped around infected cells and is not transported to other parts of the plant. Phytobactericides act as chemical barriers around infected cells to prevent further infection of pathogens.

The induction of plant defensins is nonspecific. Biological factors: Pathogenic bacteria, non-pathogenic physiological competition, can lead to the formation of starch resistance in plants.

4.3. Molecular factors of plant disease resistance

Through the interaction of resistance gene (R) and receptive gene (R), host plants exhibit molecular resistance to disease, i.e., gene resistance. R genetic factors and R genetic factors are essential genetic factors for plant normal metabolism. Those are the original functions, but the two functions are different. Once invaded by pathogenic bacteria, r plants will show resistance, and R plants will show symptoms. Host pathogens, pathogenic gene interactions of disease resistance genes, and how disease resistance genes cause disease resistance are all more complex topics. The number of microeffect genes is uncertain, the natural environment is very vague, and it is difficult to learn. Current research focuses on the identification, isolation, cloning and expression of drug resistance genes. Thus, the gene functions of disease resistance genes, disease resistance institutions and signal transduction pathway related genes were identified.

In order to fundamentally identify the mechanism of plant diseases, it is particularly important to study the pathogen infection mechanism and disease resistance mechanism of plant defense system. In the past 20 years, great progress has been made in the research of plant disease resistance molecular biology, which is mainly reflected in the deep understanding of plant disease resistance mechanism at the molecular level, and the ultimate realization of gene transformation research using genetic engineering to improve plant disease resistance.

5. Conclusion

The control of tea blight is mainly based on agricultural management and chemical control, using harmless seeds and resistant varieties. We should strengthen cultivation management, improve plant resistance and adopt comprehensive control measures centered on preventing sowing in growing
season. The main countermeasures of various control methods and the control effect of fungicides during the dissemination period, after the end of Japanese tea ceremony, during the summer Japanese tea ceremony held a dissemination, but in June, the temperature of young tea plantations increased, the frequent occurrence of leaves, spray drying protection needs to be dryer than the dry season leaves in August, high temperature drying. When the occurrence of diseases is beneficial to meteorological conditions, the average temperature is 28 degree c, rainfall > 40 mm, and the relative humidity is 80%. Spray for another 10 days or so, and then control the development of the disease. Generally, spray 2 - 3 times a year.

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