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Anthracnose of Persimmon Caused by *Colletotrichum gloeosporioides* in China

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ABSTRACT

Persimmon (*Diospyros kaki* Thunb.) is native to China and has been widely cultivated for a long time in China. *Colletotrichum gloeosporioides* is the major pathogen of persimmon crop in almost all areas where it is grown extensively, causing economically important losses. *C. gloeosporioides* may overwinter as mycelium in or on diseased tissues of a tree. Conidia are water-borne and spread by splashed rain so infection is usually highest during the wettest periods of the growing season. *C. gloeosporioides* exhibits an infection strategy of intracellular colonization in the infection process and its infection vesicles and primary hyphae are surrounded by an interfacial matrix that separates the fungal cell wall from the invaginated host plasma membrane. Current management strategies for this fungus comprise the exploitation of cultivar resistance, and cultural and chemical means. This review focuses on some progress made in China, and also describes our own work on the symptoms, origin of inoculum and hemibiotrophic pathogen, *C. gloeosporioides* on persimmon.

Keywords: *Colletotrichum gloeosporioides*, disease management, fungal–plant interactions, persimmon anthracnose, symptoms

CONTENTS

INTRODUCTION

Persimmon is the common name for some trees of the genus *Diospyros* in the *Ebenaceae* family. There are about 400 species of persimmon. Persimmon species important for their fruit are *Diospyros kaki*, *D. oleifera*, and *D. lotus*. However, the most widely cultivated species in Asian countries with increasing popularity worldwide is *D. kaki*, persimmon or oriental persimmon, called 'shizi' in Chinese. This species is native to China, and from here it spread to Korea, Japan and different parts of the world. Persimmon (*D. kaki*) has been cultivated in China for over 2500 years. Its propagation history by cleft-grafting was traced back to late years of the Northern Wei Dynasty (386-534) in the earliest surviving Chinese agricultural encyclopedia Qimin Yaoshu (Essential Techniques for the Peasantry) (Shi 1957). It is found wild at altitudes up to 6000-8000 ft (1830-2500 m) and it is cultivated from Liaoning province southward to Guangdong. The long history and diversified climates enable China to harbor the most persimmon varieties and more than 2000 different cultivars exist and more than 963 cultivars have been cultivated (Wang *et al*. 1997; Dalong *et al*. 2006).

Persimmon was grown widely for its delicious and attractive fruits. In China, it was planted in home yards and in or around the crop fields for the need of great self-sufficiency under the fundamental framework of Chinese small-farming economy in the early days. Great attention was not paid to the commercial value of persimmon until the early 1990's. With China's rapid economic development, consumption and trade of persimmon increased rapidly. Although a minor crop, persimmon provides some source of income for a few fruit growers. In order to meet increasing market demand, local government encouraged growers to grow local specific persimmon cultivars as specialty products, or high quality cultivars introduced from other countries. Persimmon seedlings were propagated at a large scale and many persimmon orchards were established, promoting the development of the persimmon industry. In 2005, persimmon production was 1,837,000 t in China (FAO 2006). However, persimmon cultivation on a large scale resulted in the severe occurrence of anthracnose disease.

The anthracnose of persimmon is a destructive disease. It occurred severely in persimmon nurseries and in the field, causing serious economic losses to growers and became a crucial problem in the development of the persimmon industry in China. To control the disease, research was conducted for many years. In this paper, some of those research results on anthracnose of persimmon in China are reviewed.

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Invited Mini-Review

PERSIMMON ANTHRACNOSE AND ITS PATHOGEN

Anthracnose of persimmon is an archaic disease in China. It was first described in Taiwan and its pathogen was identified as *Gloeosporium kaki* Hori (Sawada 1933). From then on, the anthracnose disease was reported in Sichuan (1941), Yunnan (1941), Henan (1950), Jiangxi (1955), Hebei (1960), Shandong (1960), Jiangsu (1960), Zhejiang (1960) and Guangxi provinces (1960) (Dai 1979). However, anthracnose fungus was still described under the name of *G. kaki* (Dai 1979). Until 1975, anthracnose fungus name was formally transferred and accepted as *Colletotrichum gloeosporioides* Penz. (Teaching Materials Writing Group of Plant Pathology 1975). In the early 1990's, the persimmon industry developed rapidly in areas where persimmon trees were grown widely. Some persimmon orchards were established and some sweet persimmon cultivars were introduced from Japan as well (Jia *et al*. 1997; Luo 2002). Anthracnose of persimmon took place not only in provinces where they were recorded previously, but also in other provinces, such as Fujian, Hubei, Shaanxi and Shanxi provinces (Xu *et al*. 1999; Luo 2002).

The life cycle of *Collectotrichum* species comprises a sexual and asexual stage. In general terms, the sexual stage accounts for the genetic variability and the asexual stage is responsible for the dispersal of the fungus. Whereas *C. gloeosporioides* is considered a cumulative species and its teleomorph belongs to *Glomerella cingulata* (Stonem.) Spauld. & Schrenk, which affects tropical, subtropical, and temperate fruits, such as almond, apple, avocado, mango, pecan, nectarine, and peach (Freeman and Shabi 1996). Depending on morphological criterion, all anthracnose fungus from persimmon trees were identified as *C. gloeosporioides* (Jia *et al*. 1997; Johnston and Jones 1997; Luo 2002; Zhang *et al*. 2005), but its sexual stage has never been found in nature and culture. In *Colletotrichum*, morphological characteristics are variable in culture and there is an overlap of phenotypes, which has not always made these criteria reliable. DNA sequence comparisons have been used to examine a number of *Colletotrichum* species, and sequence of the internal transcribed spacer regions (ITS) of the ribosomal DNA have proven to be particularly useful for delimiting members of this genus (Sherriff *et al*. 1994, 1995; Bailey *et al*. 1996; Sreenivasaprasad *et al*. 1996). Depending on sequence analysis of ITS regions of ribosomal DNA, anthracnose fungus infecting persimmon trees isolates isolated from persimmon was confirmed further to be *C. gloeosporioides* (Teleomorph: *Glomerella cingulata*) (Zhang *et al*. 2005).

DISEASE SYMPTOMS

Anthracnose fungus may attack the newly-formed twigs, leaves (leaf petioles and vein) and fruits of most susceptible persimmon. The first symptoms on new twigs are darkish, oval or elliptic spots, which are little, needle-point in size. The little spot enlarges and develops into a dark brown lesion (**Fig. 1A**), with a sharp line of demarcation between diseased and symptomless tissue. Pinkish-orange areas are

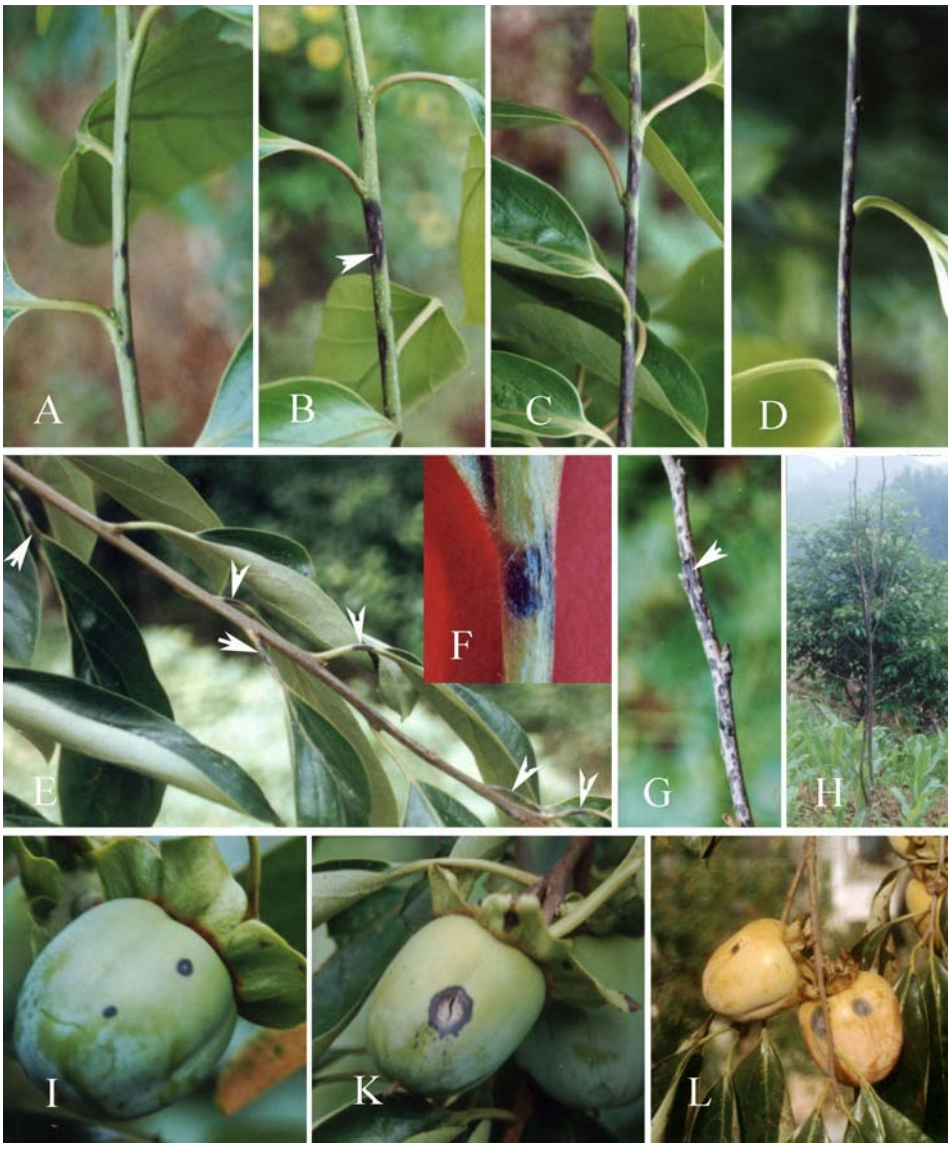

Fig. 1 Symptom of persimmon anthracnose on 'Wuheshi' (*Diospyros kaki* cv. Wuheshi). (**A-D**) Disease lesions on newly twigs. (**A**) Single spots. (**B**) Lesions coalescing a big lesion (arrows). Note that there are conidia masses (arrows) within the lesion. (**C-D**) Lesions coalescing one whole on twigs. Note that leaf petioles were not infected, and some of leaves had shed off. (**E**) Lesions (arrows) on leaf petioles. (**F**) A magnification view of the lesions in (**E**). (**G**) Anthracnose lesions and twig canker. (**H**) Persimmon tree killed by the anthracnose fungus. (**I-L**) Fruit lesions on premature and near mature persimmon fruits. (**I**) Purple dark lesions on a premature fruit. (**K**) A large lesion on premature fruit. (**L**) Dark brown lesions on near mature fruits.

formed by the conidial masses that cover the lesion center and are frequently produced in a concentric ring pattern. Several lesions may coalesce to form a large lesion (**Fig. 1B**), which continue to elongate until the entire twigs are affected (**Fig. 1C, 1D**). Meanwhile, the bases of petioles are infected and the leaves defoliate soon (**Fig. 1D**). Fungus in a large lesion may extend into the xylem resulting in collapse and a longitudinal crack, finally forming canker on a twig (**Fig. 1G**). When a new twig is girdled or infected entirely, twig blight occurs in the current season. If the amount of twig blight occuring on a tree is significant, the entire tree can be killed within two or three years (**Fig. 1H**).

Lesions on petioles and leaf veins are later than that on new twigs. Symptoms also may appear as little, round or ovoid, sunken, purple to dark brown (**Fig. 1E, 1F**), frequently being 2-4 mm in diameter (**Fig. 1F**). When a petiole is seriously infected, the leaf may continue to live and remain green for an extended period of time but easily drops off with wind.

The first symptoms of persimmon fruit anthracnose appear as circular spots 3 to 8 mm in diameter, sharply defined, occasionally slightly depressed and purple to dark purple in color on the premature fruits (**Fig. 1I**). As the disease progresses, sometimes fruit lesions reach about 20 mm in diameter. The center of the lesions becomes tan to light brown to almost gray white over time, while the broad margins remain dark purple. Under dry conditions, the diseased lesions are sunken, and the longitudinal gap often occurs through its center (**Fig. 1K**). When persimmon fruits are infected near maturity, the diseased lesions often are brown or dark brown (**Fig. 1L**) and pinkish-orange conidial masses are produced in the lesion center. Most of the seriously infected fruits drop in an unripe state. Earlier infection causes earlier fruit drop. Anthracnose of sweet persimmon also occurs in market shelves and storage warehouses, resulting in fruit rot (Jia *et al*. 1997; Zhang *et al*. 2005).

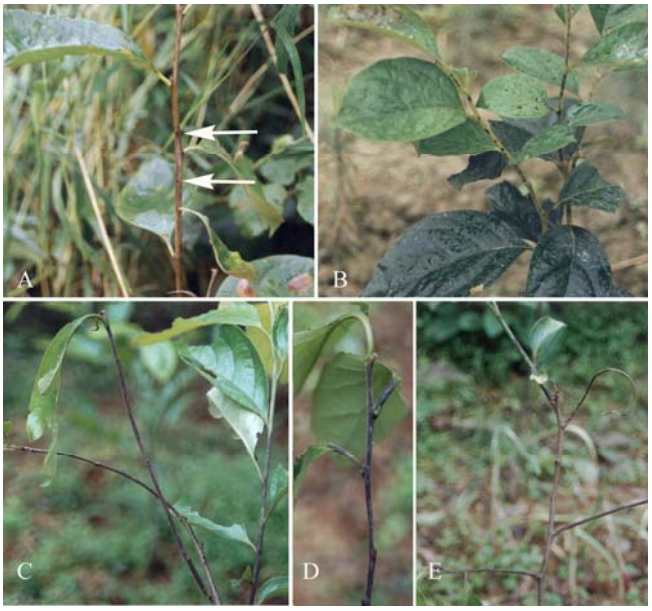

Fig. 2 Symptoms of persimmon anthracnose causing by *Colletotrichum gloeosporioides* on 'wild persimmons' and 'Wuheshi'. (**A**) Lesions (arrows) on wild persimmons in the shrubby hillsides. (**B**) Lesions on twigs a persimmon seedling in the nursery. The twigs developed from the scions of 'Wuheshi' that were grafted onto the rootstocks of 'wild persimmons'. (**C-E**) Symptoms of anthracnose on one-year-old persimmon plants transplanted to field. (**C**) Symptoms of diseased twigs. Note that twigs infected became dark brown and leaves almost completely shed off on two twigs; but the petioles were not infected on another a twig. (**D**) Lesions on a twig developing to petiole. (**E**) Symptoms of twig blight.

ORIGIN OF INOCULUM

The origin of inoculation that initiates the first infection in a persimmon nursery and in fields is unknown in most regions. In disease-free regions, it was considered in general that the inoculum resource came from the rootstock or grafted scions carrying the pathogen in a nursery, while in the fields it came from transplanting seedlings (Zhang and Xu 2003).

A good example illustrating that the initial inoculum resource of persimmon anthracnose occurred in the Chunan area, Zhejiang province was originated from 'wild persimmon' (*Diospyros kaki* var. sylvestris Makino). The Chunan area is located in a mountainous area and 'Wuheshi' is a locally important cultivar, which has been cultivated for more than 600 years, as described in written records (Zhang *et al*. 2003). Thus far, more than 200 year-old persimmon trees still can be seen in several villages. In the previous cultivation of 'Wuheshi', growers were used to propagating persimmon seedlings by means of root suckers. There is no persimmon anthracnose disease on record, while local growers are almost ignorant about it. In 1992, 666 ha of persimmon orchards were established by the local government to support the development of the persimmon industry. Subsequently, anthracnose of persimmon occurred severely in each year. Up to 1996, about half of the persimmon trees died from the disease.

The investigation indicated that the initial inocula came from the rootstocks carrying the pathogen. Due to the limitation of root sucker propagation at a large scale, growers had to choose another means of propagation. Alternatively, clonal stocks were reproduced by whip grafts and the close relative, wild persimmon, was used as the rootstock. This was because wild persimmons grow widely in shrubby hillsides, and are an available, rich genetic resource. In addition, this rootstock significantly contributed to adversity-resistance and survives. However, the wild persimmon in shrubby hillsides is susceptible to anthracnose disease but seldom is severe (**Fig. 2A**). Once the wild persimmon carrying the pathogen was used as the rootstock the pathogen would spread to the grafted newly twigs of 'Wuheshi', resulting in the occurrence of disease in the nursery (**Fig. 2B**; Zhang and Xu 2005). Once infected seedlings were transplanted to the field, the disease would develop subsequently (**Fig. 2C-E**).

INFECTION PROCESS

The initial stages of infection process of *C. gloeosporioides* on 'Wuheshi' are the same as that of other *Colletotrichum* species. Conidia adhere to, and germinate on, plant surfaces, produce germ tubes, and then go on to form appressoria which penetrate the cuticle directly (Zhang and Hu 2004). Successful penetration was followed by the formation of globular infection vesicles and filamentous primary hyphae, which grow between plant plasma membranes and plant cell walls without penetrating host protoplasts, After colonizing one or more host cells, the primary hyphae, which are biotrophic, subsequently give rise to secondary necrotrophic hyphae (Zhang *et al*. 2003, 2005). In these respects, the initial infection process of *C. gloeosporioides* is similar to that of *C. lindemuthianum* on bean and *C. sublineolum* on (O'Connell *et al*. 1985; Wharton *et al*. 2001).

During the biotrophic stage of *C. gloeosporioides* in persimmon, infection vesicles and primary hyphae are surrounded by an interfacial matrix that separates the fungal cell wall from the invaginated host plasma membrane (**Fig. 3A**), closely resembling that of *C. lindemuthianum* on bean. In contrast, on the interaction interface between persimmon host- primary hyphae, the fungal cell wall is deposited by a layer of irregular electron-opaque material and host plasma membrane is wavy (**Fig. 3B**) (Zhang *et al*. 2005). Otherwise, in the process of penetrating the host cell wall, the primary hyphae become greatly constricted from cell to cell, as described in *C. sublineolum* (Wharton *et al*. 2001). Primary hyphae also were able to penetrate the thickened, lignified walls of sclerenchyma cells but used a specific way. The primary hyphae firstly form a funnel-shaped hyphal cone

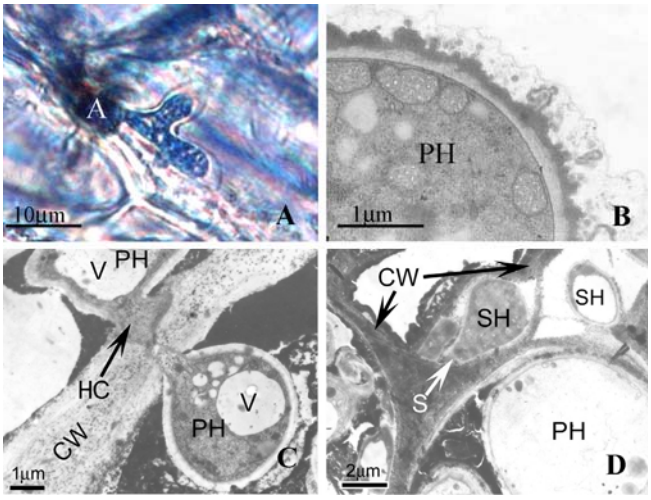

Fig. 3 Infection of *Diospyros kaki* cv. Wuheshi by *Colletotrichum gloeosporioides*. A. Appressorium (**A**) penetrating a epidermal cell to form a large primary hypha. Stained with lactophenol-Aniline blue. (**B**) Interface of primary hyphae (PH)-plasma membrane interaction. Note the plasma membrane was intact, smooth, with pleated sheet. (**C**) A primary hyphae (PH) penetrating the thickened cell wall between two host cells. Note the primary hyphae (PH) first formed a funnel-shaped hyphal cone (HC) within the host cell wall (CW) and then penetrated the cell wall to form swollen primary hyphae. (**D**) Secondary hyphae rupturing the host cell wall (CW). Note the top cell of secondary hyphae within the cell wall swollen to cause host cell to splinter. (**B**) Reprinted with kind permission from **Zhang J-Z, Hu D-W, Xu T** (2005) Ultrastructure of infection of persimmon petiole by *Collectotrichum gloeosporioides*. *Acta Phytopathologica Sinica* **35 (5)**, 434-441 with kind permission of Beijing Bureau for Distribution of Newspapers and Journals, ©2005. (**C, D**) Reprinted with kind permission from **Zhang J-Z, Hu D-W, Xu T** (2003) Studies on cytology of the infection of persimmon by *Colletotrichum gloeosporioides*. *Mycosystema* **22 (4)**, 645-652 with kind permission of Science Press, ©2003.

within the host cell wall at points of penetration and a narrower hyphae (0.3 to 0.5 μm in diameter) was produced from its button, and then penetrated the cell wall (**Fig. 3C**) (Zhang *et al*. 2003). Obviously, the morphology and function of the narrower hyphae are homologous to that of an infected peg produced from an appresorium. In the necrotrophic stage, the mechanical pressure may also play an important role in development of secondary hyphae except that the cell wall degraded enzyme. The observations showed that after invading the host cell wall, secondary hyphae produced a septum near its top and the global top cell swelled more than 2-3 times in diameter, causing the host cell wall to rupture (**Fig. 3D**) (Zhang *et al*. 2003).

In addition, for understanding the mechanisms of interaction between pathogen and host, a genomic library of *C. gloeosporioides* from persimmon cv. 'Wuhesh' was constructed with SuperCos1 vector, and cloning of pathogenesis related genes of *C. gloeosporioides* was also conducted by *Agrobacterium tumefaciens*-mediated transformation (Sun 2008). A few mutants have been shown to be related to pathogenicity and corresponding gene fragments have been cloned (Sun 2008).

DISEASE DEVELOPMENT

Anthracnose fungus overwinters as mycelia in twig lesions and cankers or blighted twigs within the tree canopy. However, in dead twigs, the fungus survives only in infected bark tissues adjacent to the living tissue parts (Zhang and Xu 2003). In spring, when rainy weather occurs, conidia form from the overwintering mycelium as the primary inocula.

Conidia are primarily dispersed by rain splash and runoff within the tree canopy and the distance to which they may disseminate varies depending on wind direction and the size of the tree canopy. Spread can occur over short distances for wet conidia and range from 1.5-3 m on unfruited trees (less than 5-year-old trees). The primary inocula land on the surface of the newly expanding twig and cause the first infection of the season. After a short time lesions form, and a new generation of conidia, i.e., the secondary inocula, reproduce. In the Chunan area of Zhejiang province, the symptoms often appear by 4-5 days after rainy or continuously overcast and rainy days, occasionally over 10 days. For example, the lesions appeared after 10 days in continuously overcast and rainy days from April 15 to May 9, 2002, and after 4 days from May 3 to May 9, 2001 and after 5 days from April 29 to May 9, 2001, respectively. The time in which lesions and secondary inocula are produced is dependent on the biology of the pathogen and its host and the duration of environmental conditions needed for infection. In the field, the secondary inocula were observed after 5-6 days under favorable environmental conditions. A pathogenicity test also showed that lesions on 'Wuheshi' appeared at 70 h after inoculation at 25°C and conidial mass at 90 h; lesions formed after 10 d at 17°C but no conidial mass; no lesions developed at 15°C (Zhang and Hu 2004). The secondary infection cycle can be repeated many times during the growing season as long as young succulent twigs are available and warm, wet, and humid conditions prevail. Therefore, severe disease occurs after long periods of heavy overcast, rainy weather.

Pathogen infection and the development of diseased lesions cease once the color of twigs turns from green to light brown with rapid growth. However, conidia produced from lesions may continue to infect leaves (leaf petioles and veins) and fruits during the growing season during rainy weather. Conidia can also infect leaves of older persimmon trees and fruits (20- to 30-year-old) adjacent to persimmon orchards, but their twigs can not be infected during the entire growing season (Li *et al*. 2009). It is possible that the fungal conidia are disseminated by wind over longer distances under dry environmental conditions.

CONTROL MEASURES

Effective control of persimmon anthracnose disease usually involves the use of one or a combination of several management strategies, including cultivar resistance, cultural control and chemical control.

Resistant persimmon cultivars are the most significant aspect of disease management in a persimmon crop, but resistance to anthracnose disease has hardly been exploited in China due to the longer time-frame required for breeding and selecting for resistance. In addition, replacing susceptible hosts is costly, and most growers tend to tend to select cultivars based on commercial criteria other than disease resistance. For example, in the Chunan area of Zhejiang Province, 'Zhejiangshi' (*D. glaucifolia*), a local cultivar immune to anthracnose disease, was used as rootstock (Zhang and Xu 2005) but the susceptible 'Wuheshi' has to be used as the scion because its qualities must be taken into account. In other areas, some plants of *D. lotus* were also used as rootstock.

Cultural measures usually aim at reducing the initial inoculum sources (Ploetz 2007). Nurseries should be established in a place away from the diseased regions. Resistant rootstocks and disease–free scions should be selected and only disease-free nursery stock should be used. Meanwhile, planting site selection is an important step in establishing a production area, and can be an important tactic for avoiding disease. In the field, removal of inoculum sources such as infected twigs and fallen leaves in the winter or in early spring when still dormant can help limit the amount of fungal inocula present for infection of newly twigs the following spring. Other measures such as spacing and pruning can reduce the suitability of environmental conditions for disease development by assisting more rapid drying of the tree canopy, as well as allowing better penetration of fungicide sprays. Spring fertilizer applications should not include

high amounts of readily available nitrogen, which encourage vigorous twig growth, especially on fruitless persimmon trees.

Chemical control methods are widely used on perennial persimmon. Generally, the anthracnose of persimmon can be controlled by a range of chemicals such as carbendazim, chlorthalonil, benomyl, thiophanate-methyl, manozeb and thiram (Jia *et al*. 1997; Yu *et al*. 2001; Li 2004). For successful control of disease, timing and placement are of critical significance. These fungicides are applied once or twice in winter and in early March or before foliation. During the growing season, fungicides also are applied to the young, newly expanding twigs and fruits against infection before or immediately after every rain. Application of fungicides to the right target is often difficult during this period of the rainy season, and repeated applications are often necessary to protect the rapid expansion of the surface of susceptible host tissues from infection. These measures are rate-limiting measures (Ploetz 2007).

RESISTANCE TO ANTHRACNOSE

Different persimmon cultivars are cultivated widely in different areas or provinces of China for adapting to diversified climate conditions. The geographic isolation and coevolution of the long-term interaction between host and anthracnose fungus may lead to a diversity of persimmon resistance. The anthracnose fungus may be restricted to certain cultivars or organs and tissues that it attacks. Some observations showed that persimmon cultivars vary greatly in their susceptibility to anthracnose disease of twigs, leaves (petioles and veins), and fruit (**Table 1**) (Jia *et al*. 1997; Xu *et al*. 1999; Luo 2002; Xue *et al*. 2003; Li 2004; Zhang and Xu 2005). No new studies have emerged in China between 2006 and 2008.

Differences in susceptibility of persimmon cultivars in the Chunam area of Zhejiang province have been noted in the field and laboratory (Zang and Xu 2005). In a cultivar of *D. glaucifolia*, 'Zhejing persimmon' is completely resistant; in *D. kaki* cultivars, 'Wuheshi', whose twigs, leaves (petioles and veins), and fruit can be infected, is very susceptible, 'Dongshi', whose fruit infected, is susceptible, and 'Wild persimmon' (*D. kaki* var. *sylvestris*), whose twigs are infected, is only lightly susceptible (Zhang and Xu 2005). It is considered that 'Wuheshi' has been cultivated for a long period of time as a locally important cultivar; finally, the pathogen overcomes host resistance, resulting in the serious occurrence of disease. In the same way, most widely grown cultivars in other areas, such as astringent persimmon cvs. 'Jingmianshi', 'Niuxinshi', 'Bayuehuang', 'Denglongshi', 'Sifangshi', 'Taoyuanshi', 'Dataohuang' and 'Gongchengshuishi' and so on, are very susceptible (Xu *et al*. 1999; Luo 2002; Xue *et al*. 2003). In recent years, some sweet persimmon cultivars were introduced from Japan and cultivated extensively, but anthracnose disease occurred frequently on them (Jia *et al*. 1997). However, although some cultivars, such as 'Maekawa-Jirou', 'Youhou' and 'Zenjimaru', were susceptible, and 'Nishimura-wase' and 'Okitsu 20' appeared to be resistant (Li 2004).

Almost most resistance of persimmon cultivars to anthracnose disease is unknown in China. Despite many reports about the occurrence of anthracnose disease of persimmon, most of them hardly involved host resistance or no records were available. Thereby, identification and use of resistance to anthracnose disease and genetic resistance obtained via breeding will be the most important aspects in management strategies of persimmon anthracnose disease in the future.

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