Current Status of Occurrence of Major Diseases on Kiwifruits and Their Control

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The kiwifruit (Chinese gooseberry, *Actinidia deliciosa* (A. Chev) Liang et Ferguson) introduced from New Zealand has been grown commercially since the early 1980s. It is mainly cultivated in southern coastal areas where winters are generally warm. However, vines are frequently damaged by cold during the winter season. Several diseases have been reported on kiwifruit following an increase in cultivated acreage during the past two decades. Bacterial canker and blossom blight and fruits rots are considered as the major diseases on kiwifruit and cause serious economic losses during cultivation and storage.

Bacterial canker caused by *Pseudomonas syringae* pv. *actinidiae* was first reported in Japan in the early 1980s and it was also observed in Jeju Island in the late 1980s. About 34% of kiwifruit vineyards in Jeonnam Province were damaged in 1993 and some of the vineyards were completely destroyed due to the epidemics of the disease. Low temperature in winter was closely related with the disease outbreak. The severe bacterial canker occurred in vineyards with no or poor wind break stands and they were located in the northern slope of mountains. Under these circumstances, cold damage, strong wind and rainfall can easily cause injuries through which the bacterial pathogen infects kiwifruit vines.

Usually bactericides have been used to control bacterial canker of kiwifruit. The occurrence of resistant strains to streptomycin and copper has been reported as a serious problem to control bacterial canker in kiwifruit in Japan. Thus far, there is no report on the occurrence of resistant strains of the pathogen to the bactericides in Korea. Prolonged and/or intensive use of the bactericides may also create a selection pressure for resistant strains in Korea as well. However, Korean strains of *P. syringae* pv. *actinidiae* were quite different with Japanese strains in the ability of phytotoxin production and they were also separated from Japanese strains by RAPD analysis. This reveals that Korean and Japanese strains of *P. syringae* pv. *actinidiae* may have different phylogenic origins.

The period of application of bactericides determined control efficacy of bacterial canker. For example, bacterial canker was very effectively controlled by applying streptomycin+copper hydrxide WP and copper hydroxide WP from late January to middle March in Korea. However, a single or combined use of copper hydroxide with other bactericides caused phytotoxicity on leaves of kiwifruit, and these bactericides should not be applied to newly emerging leaves from middle April. Bacterial canker was also effectively controlled by trunk injection of streptomycin after fruit harvest. Trunk injection can be effective if done just after fruit harvest and dosage of the antibiotics to be used depends on the age of vines, the healthiness, and severity and longevity of canker disease.

Since bacterial canker shows typical symptoms on kiwifruit trees two or three years after latent infection, spraying bactericides or trunk injection of antibiotics has achieved partial success in controlling the disease after symptoms appear. Once it has occurred, the disease seriously damages the kiwifruits enough to destroy vineyards within a very short time. Recently, the development of molecular markers for identification *P. syringae* pv. *actinidiae* made it possible to identify the bacterial canker pathogen from leaves, trunks or saps of kiwifruit trees before any symptoms appear on the trees, even from soil in kiwifruit vineyards.

Disease incidence of bacterial blossom blight caused by *Pseudomonas syringae* pv. *syringae* ranged from 13.0 to 36.2% in naturally infected kiwifruit orchards of major kiwifruit-growing areas in Korea from 1997 to 1999. The disease occurs during flowering in the late May and disease outbreaks associated with rainfall during the flowering season have resulted in a severe reduction in kiwifruit production. A significant correlation occurred between disease incidence and rainfall at flowering. Dead fruit stalks, dead pruned twigs, fallen leaves and soils mainly provided *P. syringae* pv. *syringae* with overwintering places in the kiwifruit orchards, and the inocula also overwintered on buds, trunks, branches, and twigs on the kiwifruit trees. Among the overwintering places, the incula were detected in the highest frequencies from dead fruit stalks. The highest population density of *P. syringae* pv. *syringae* on the overwintering places was detected in May and June when the daily average temperature coincided with the optimum temperature for the bacterial growth.

Bacterial blossom blight has been mainly controlled by spray of several bactericides. Trunk injection of various concentrations of acetic acid also displayed variable control efficiencies. Use of a transparent polyvinyl film to protect kiwifruit trees

from rain or girdling of trunks of kiwifruit trees were also turned out to alleviate bacterial blossom blight more effectively than the spray of bactericides in kiwifruit orchards. Installation of partial rain-proof vinyl cover over kiwifruit trees before about 75 days of full flowering stage of kiwifruit prevented most of the disease occurrence on kiwifruit. However, both girdling of tree trunks and partial rain-proof vinyl cover on the same kiwifruit trees or spray of bactericides on the girdled trees or covered trees did not show significantly synergic effects on the disease control. The results suggest that girdling of tree trunks and partial rain-proof vinyl cover over kiwifruit trees can substitute the routine chemical application as environmentally friendly control methods to manage bacterial blossom blight in kiwifruit orchards.

The overall disease incidence of postharvest fruit rots of kiwifruit in 1999 averaged 32%, but the incidence ranged from 5% to 68% in the orchards surveyed. The percentage of kiwifruit showing internal and external symptoms were 21.9% and 4.9%, respectively, and an additional 5.2% of the kiwifruit showed both internal and external symptoms. *Botryosphaeria dothidea* and *Diaporthe actinidiae* cause ripe rot and stem-end rot, respectively, and were identified as the major postharvest pathogens with average isolation rates of 83.3% and 11.9%. Incidence of the postharvest fruit rots was closely correlated with ripening temperatures favourable to the mycelial growth of the major pathogens. Postharvest fruit rots occurred at all ripening temperatures $\geq$ 11℃ and maximum disease incidence was observed at 29℃. No mycelial growth of *B. dothidea* and *D. actinidiae* occurred on potato dextrose agar plates under 11℃ and the optimum temperature ranges for mycelial growth of the pathogens were 26-35℃ and 26-29℃, respectively. The optimum kiwifruit ripening conditions for minimising damage from the postharvest fruit rots were a 20-day-ripening at 17℃.

Intensive application of fungicides just before or after the rainy season is conducted to control postharvest fruit rots in kiwifruit orchards of Korea. Benomyl WP and thiophanate-methyl WP, registered as preventive fungicides against postharvest fruit rots, are usually applied 5-6 times at 10-day-intervals beginning in early June in the kiwifruit orchards. Tebuconazole WP, iprodione WP, and flusilazole WP were selected as alternative fungicides to prevent emergence of fungicide-resistant strains and reduce the number of fungicide applications. The optimum spray program for controlling postharvest fruit rots was four applications at 10-day-intervals from mid June for tebuconazole WP, iprodione WP, and flusilazole WP, compared with five applications for benomyl WP and thiophanate-methyl WP.