

## D-PSA-K: A Model for Estimating the Accumulated Potential Damage on Kiwifruit Canes Caused by Bacterial Canker during the Growing and Overwintering Seasons

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We developed a model, termed D-PSA-K, to estimate the accumulated potential damage on kiwifruit canes caused by bacterial canker during the growing and overwintering seasons. The model consisted of three parts including estimation of the amount of necrotic lesion in a non-frozen environment, the rate of necrosis increase in a freezing environment during the overwintering season, and the amount of necrotic lesion on kiwifruit canes caused by bacterial canker during the overwintering and growing seasons. We evaluated the model's accuracy by comparing the observed maximum disease incidence on kiwifruit canes against the damage estimated using weather and disease data collected at Wando during 1994–1997 and at Seogwipo during 2014–2015. For the Hayward cultivar, D-PSA-K estimated the accumulated damage as approximately nine times the observed maximum disease incidence. For the Hort16A cultivar, the accumulated damage estimated by D-PSA-K was high when the observed disease incidence was high. D-PSA-K could assist kiwifruit growers in selecting optimal sites for kiwifruit cultivation and establishing improved production plans by predicting the loss in kiwifruit production due to bacterial canker, using past weather or future climate change data.

**Keywords :** bacterial canker, disease incidence, disease

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model, Hayward, kiwifruit

Bacterial canker, caused by *Pseudomonas syringae* pv. *actinidiae* on kiwifruit (*Actinidia* spp.), is one of the major diseases that occasionally causes severe damage in orchards and extensive economic losses in Japan (Serizawa et al., 1989; Takikawa et al., 1989), Korea (Ko et al., 2002; Koh, 1995; Koh et al., 1994, 2012), Italy (Balestra et al., 2009; Scortichini, 1994), and many other countries (Balestra et al., 2010, 2011; Bastas and Karakaya, 2012; Everett et al., 2011; Vanneste et al., 2011b). In Korea, bacterial canker on kiwifruit was first observed at Jeju in 1988, and has rapidly spread since (Koh et al., 1994; Lee et al., 2005). This pathogenic bacterium has been isolated from both commercial (*A. deliciosa* and *A. chinensis*) and wild (*A. arguta* and *A. kolomikta*) (McCann et al., 2013; Scortichini et al., 2012) kiwifruits. This bacterium comprises four distinct clades (McCann et al., 2013). Three clades correspond to their geographical origins of isolation, which are Japan, Korea, and New Zealand. The fourth clade is responsible for the recent bacterial canker outbreak in Italy and New Zealand, and is now distributed globally. Pathogenic bacteria in the fourth clade have also been observed in Korea (Koh et al., 2012).

Bacterial canker disease symptoms on kiwifruit include brown–black leaf spots, often surrounded by yellowish halos, blossom necrosis, die-back or blight of twigs, reddening of lenticels, and bleeding cankers on stems, with whitish to red bacterial ooze (Scortichini et al., 2012). The bleeding cankers on kiwifruit stems begin to appear in February or March, and reach their maximum incidence in late April or May (Ko et al., 2002). Symptoms on kiwifruit leaves begin to appear in early April or May (Ko et al., 2002; Koh et al., 1994). *P. syringae* pv. *actinidiae*

is found on both symptomatic and asymptomatic tissues, indicating that it can survive as an epiphyte (Vanneste et al., 2011c). Temperatures within the range of 12°C to 18°C and humid conditions could favor multiplication of the bacterium, allowing it to disperse systemically from infected sites to uninfected ones (Scortichini et al., 2012). Temperatures above about 35°C could suppress multiplication and dispersal of the pathogen. Freezing plant tissue during the cold winter could be a major factor contributing to the development of severe bacterial canker symptoms in early spring, although the pathogenic bacterium does not display ice nucleation activity (Ferrante and Scortichini, 2014; Ko et al., 2000a, 2000b; Serizawa et al., 1989).

To manage bacterial canker on kiwifruit, applying streptomycin and streptomycin sulfate-oxytetracycline from mid-April to early May, copper hydroxide from mid-January to early February, and kasugamycin from mid-March to early April controlled the disease in Korea (Koh et al., 1999). Spraying bactericidal compounds, such as streptomycin and copper formulations, is also a highly reliable means of chemically controlling bacterial canker in other countries (Cameron and Sarojini, 2014; Nakajima et al., 2002). Trunk injection of streptomycin has also been used in Korea and has appeared effective in treating infected vines (Koh et al., 1996). However, streptomycin-resistant isolates of *P. syringae* pv. *actinidiae* were observed in Japan (Han et al., 2003b; Nakajima et al., 1995). Although streptomycin-resistant isolates have not been observed in Korea (Han et al., 2003b), reducing antibiotic (including streptomycin) application is important to preventing antibiotic resistance. Foliar application of copper hydroxide or copper-antibiotic formulated pesticides after mid-April causes severe phytotoxicity (Koh et al., 1996). Consequently, an integrated approach that includes precisely scheduled spray treatments with effective and environmentally friendly bactericides, preventative measures aimed at reducing the bacterial inocula and possible infection pathways, and enhancement of host resistance against the disease seems to be the best solution for coexistence with the disease (Ko et al., 2002; Scortichini et al., 2012). Development of resistant cultivars and biological pesticides including bacteriophages, antimicrobial peptides, and compounds that induce or activate plant defense mechanisms, is in progress (Cameron and Sarojini, 2014; Scortichini et al., 2012).

As there is no effective chemical measure for managing bacterial canker on kiwifruit from May to the harvest season in Korea (Ko et al., 2002; Koh et al., 1996, 1999), growers generally remove canes and trunks with bacterial ooze to prevent dispersion of the pathogen early in the growing season. Cutting off the canes and trunks contrib-

utes to overall yield loss. Although the initial infection on the leaves of healthy plants in the spring can lead to death by the end of the year, the symptoms on canes seldom appear from late spring to the harvest season (Koh, 1995; Koh et al., 1994; Scortichini et al., 2012). These observations indicate that infected plants could produce during the year of infection. Infections in a given year rarely cause yield loss during that year. The death or removal of canes and trunks where bacterial ooze occurred early in the growing season could contribute to yield loss caused by the bacterial canker. The disease incidence (DI) of kiwifruit early in the growing season is strongly correlated with the yield loss during that year. Maximum incidence of bacterial canker could be an indirect way to estimate yield loss caused by bacterial canker.

The maximum incidence of bacterial canker on kiwifruit canes generally occurred in late April or early May in Korea (Ko et al., 2002). The pathogen causing bacterial canker can easily transfer to other plants within and between orchards in all seasons, except for the hot summer, and the detectable symptoms on canes and trunks occur after sufficient colonization and multiplication of infected or migrated pathogens (Ko et al., 2000b; Koh, 1995; Koh et al., 1994; Scortichini et al., 2012). Successful colonization and multiplication causes necrotic lesions under the canes' or trunks' bark, which cannot be detected from the outside view. Symptoms on kiwifruit stems observed early in the growing season do not always appear at sites where successful pathogen infections occurred during the last growing and overwintering seasons. Some symptoms appear at sites where successful infections occurred more than one year previously. The observed maximum incidence in late April or early May is the result of accumulated damage on kiwifruit stems, caused by colonization and multiplication of the pathogen. Maximum incidence is not always the result of infection by the pathogen during the last growing and overwintering seasons, and would vary in direct proportion to the accumulated damage. We conducted the present study to develop a model to calculate the accumulated damage on kiwifruit canes caused by bacterial canker during the last growing and overwintering seasons; and finally estimate the maximum incidence of bacterial canker on kiwifruit canes at early growing season.

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## Materials and Methods

**Effect of temperature on bacterial canker development in kiwifruit canes.** We obtained an isolate (KACC 16843) of *P. syringae* pv. *actinidiae* from the Rural Development Administration Genebank, Jeonju, Korea. The isolate was cultured in tryptic soy broth (Difco;

Becton, Dickinson and Company, Sparks, MD, USA) at 25°C for 48 h, with shaking at 200 rpm. We used this culture suspension, diluted to a density of  $10^8$  cell/ml, to inoculate kiwifruit canes. We cut overwintered canes of *A. deliciosa* cv. Hayward, with diameters of 8–13 mm, into ~18-cm sections in early April. The surfaces of the canes were sterilized with 0.01% NaOCl solution for 5 min and rinsed twice with sterilized distilled water. The surface-sterilized canes were dipped in a 100 ppm cycloheximide solution for 5 s. Excess solution was removed using sterilized and dried filter papers. Approximately 1 cm was removed from each end using sterilized scissors. The resulting middle portion of cane was placed in a cap tube, with a diameter of 18 mm, containing 5 ml of the culture suspension or sterilized distilled water. The cap tubes were stored at 15°C, 20°C, 25°C, 30°C, and 35°C. We measured the length of discoloration under the canes' bark 3 and 6 days after inoculation, observing four or more canes for each treatment. We did not measure the length in any cane that is suspected of being contaminated with other microbes. The measured lengths of discoloration were analyzed to determine the quantitative relationship between air temperatures and daily increment of necrotic lesion lengths by linear regression analysis using the General Linear Model (GLM) Procedure of SAS (SAS Institute Inc., 2011).

**Model for estimating the necrosis rate increase caused by the bacterial canker pathogen with overwinter freezing damage.** Ko et al. (2000a, 2000b) showed that the development of lesions caused by bacterial canker on Hayward is influenced by the presence and duration of freezing temperatures during the previous overwintering season. They also showed that necrotic lesions grow significantly in proportion to freezing duration within the range of 0–36 h. Consequently, the rate of necrosis increase caused by the bacterial canker pathogen at a given freezing temperature over one hour (RNIH) can be calculated as follows:

$$\text{RNIH} = \frac{(\text{NLF} - \text{NLC})}{\text{NLC} \cdot \text{h}}$$

where NLF is the necrotic lesion length of the canes at a given freezing temperature over h hours and NLC is the necrotic lesion length of the canes without the freezing treatment. We reanalyzed the necrotic lesion lengths from Ko et al. (2000b) to quantify the relationship between temperature and RNIH via linear regression analysis, using the GLM procedure in SAS (SAS Institute Inc., 2011). The rate of necrosis increase caused by the bacterial canker in a freezing environment during the overwintering season (RNI) can be calculated as the sum of RNIH

during that season.

**Development and evaluation of a model to estimate the accumulated damage on kiwifruit canes caused by bacterial canker during the last growing and overwintering seasons.** As the length of necrotic lesion caused by the pathogen was dramatically expanded at low freezing temperatures and long freezing durations (Ko et al., 2000a, 2000b), the amount of lesion on kiwifruit canes caused by the pathogen during the last growing and overwintering seasons can be estimated by multiplying the amount of lesions caused by the pathogen within the temperature ranges for lesion development by the lesion increment influenced by freezing temperatures and duration. The amount of lesion on kiwifruit canes caused by the pathogen during the last growing and overwintering seasons can represent the accumulated damage caused by colonization and multiplication of the pathogen during the seasons. Consequently, we developed a model for estimating accumulated damage on kiwifruit canes caused by bacterial canker during the growing and overwintering seasons (D-PSA-K hereafter) as follows:

$$D = \text{NNF} \times (\text{RNI} + 1)$$

where D is the accumulated damage caused by bacterial canker during the overwintering and growing seasons, NNF is the amount of necrotic lesion area on kiwifruit canes in a non-frozen environment during the overwintering and growing seasons, and RNI is the rate of necrosis increase caused by the bacterial canker pathogen in a freezing environment during the overwintering season. Refer to the general crop calendar and a model to estimate blooming date of kiwifruits in Korea (Kwon et al., 2012), assuming that the growing and overwintering seasons in Korea are from May to September and October to April, respectively.

We evaluated the accuracy of D-PSA-K by comparing the observed maximum DI on kiwifruit canes with the damage estimated using weather and disease data collected at Wando during 1994–1997 and at Seogwipo during 2014–2015. Disease and weather data at Wando over 1994–1997 were obtained from the Fruit Research Institute of Jeollanam-do Agricultural Research and Extension Services (JARES), and the Korea Meteorological Administration (KMA), respectively. We examined the incidence of bacterial canker on kiwifruit canes at three kiwifruit orchards in Seogwipo at 7- and 10-day intervals, from 1 April 2015 to 30 June 2015. We observed more than 33 plants in an orchard that occupied at least 33% of the total area of the orchard to measure DI. Hourly weather conditions of the kiwifruit orchards at Seogwipo

during 2014–2015 were monitored by automated weather stations (Watchdog 1450; Spectrum Technologies Inc., Aurora, IL, USA), with air temperature and humidity sensors. The sensors were installed at 120 cm above the ground.

## Results

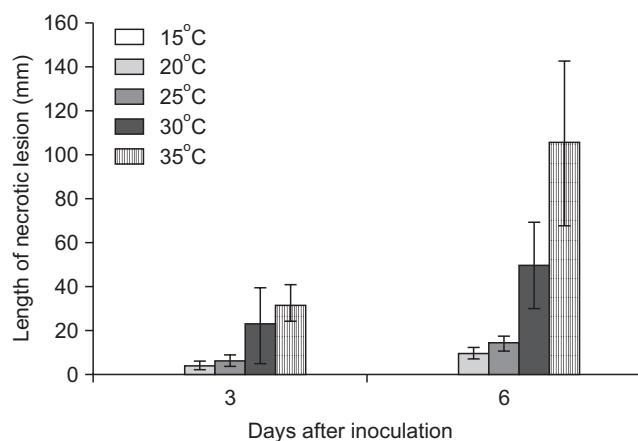
### Effect of temperature on bacterial canker development in kiwifruit canes.

Necrotic lesions on kiwifruit canes inoculated with the pathogen culture suspension were longer than those on kiwifruit canes inoculated with sterilized water at all treatment temperatures, with the exception of 15°C. The length of necrotic lesions caused by the inoculated pathogen increased when the treatment temperature increased within the range of 20–35°C (Fig. 1). Lesions at 30°C and 35°C had lighter color than those at 25°C. Linear regression analysis, using the log-transformed daily increased lengths of necrotic lesions and calculated from the lengths of necrotic lesions 6 days after inoculation, resulted in an exponential equation:

$$\text{DNNF} = \frac{0.1321e^{0.1388T_d} - 1}{10} \quad (15^\circ\text{C} < T_d \leq 35^\circ\text{C})$$

where DNNF is the daily increased length (cm) of necrotic lesions in kiwifruit canes when the temperature ( $T_d$ ) is within the range of 15–35°C (Fig. 2).

**Model for estimating the rate of necrosis increase caused by the bacterial canker with overwinter freezing damage.** Linear regression analysis, based on data reported by Ko et al. (2000b) resulted in the following 2nd order



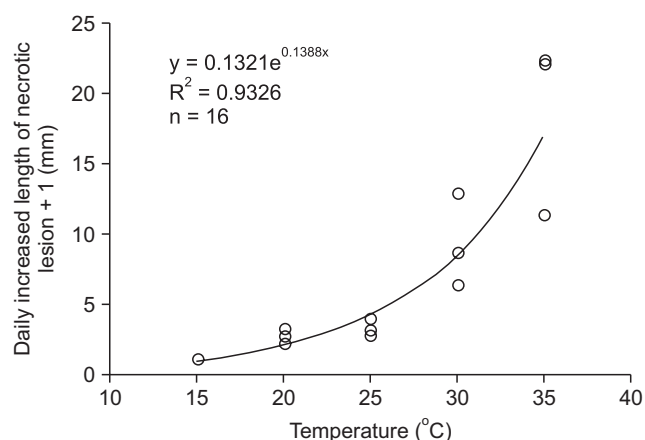
**Fig. 1.** Averages and standard deviations of necrotic lesion lengths in kiwifruit canes (Hayward cultivar) caused by the inoculation with *Pseudomonas syringae* pv. *actinidiae* KACC 16843 culture suspension at different temperatures.

polynomial equation:

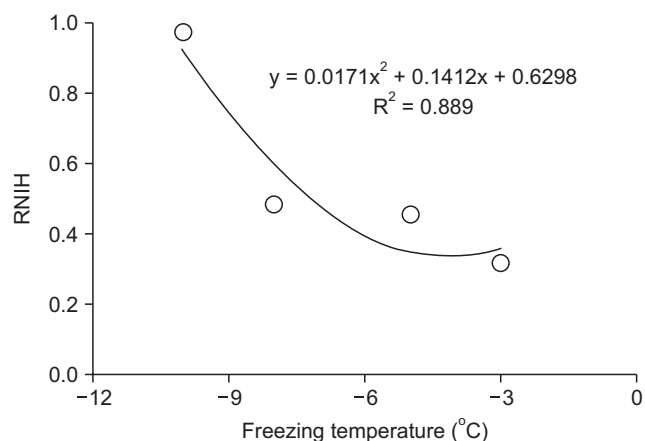
$$\text{RNIH} = 0.0171\text{Th}^2 + 0.1412\text{Th} + 0.6298 \quad (-10^\circ\text{C} \leq \text{Th} \leq -3^\circ\text{C}; R^2 = 0.889)$$

where RNIH is the rate of necrosis increase in kiwifruit cane over an hour compared with a non-frozen control when the temperature ( $\text{Th}$ ) is within the range of  $-10^\circ\text{C}$  to  $-3^\circ\text{C}$  (Fig. 3).

Assuming that the length of a bacterial canker necrotic lesion increases linearly in proportion to the duration of freezing (Ko et al., 2000a, 2000b), and that the overwintering season of kiwifruit in Korea is from October to April, the rate of necrosis increase caused by the bacterial



**Fig. 2.** Relationship between temperatures and daily increase in necrotic lesion length in kiwifruit canes (Hayward cultivar) caused by the inoculation with *Pseudomonas syringae* pv. *actinidiae* KACC 16843 culture suspension.



**Fig. 3.** Relationship between temperatures and the rate of necrosis increase in kiwifruit canes (Hayward cultivar) caused by the bacterial canker pathogen at a given freezing temperature over one hour (RNIH) calculated from the data of Ko et al. (2000b).



**Table 1.** The maximum disease incidences of bacterial canker on kiwifruit canes and damage estimated using D-PSA-K

Location	Disease observation			Damage estimated using D-PSA-K		
	Year	Cultivar	Incidence (%)	NNF (cm)	RNI	D (cm)
Wando	1995	Hayward	4.70	43.12	0	43.12
	1996	Hayward	13.56	33.53	3.13	138.48
	1997	Hayward	68.90	36.44	12.73	500.32
Seogwipo1	2015	Hayward	40.63	29.33	14.33	449.63
Seogwipo2	2015	Hort16A	33.33	32.01	0	32.01
Seogwipo3	2015	Hort16A	51.52	33.05	9.16	335.79

NNF, estimated damage to kiwifruit canes in a non-frozen environment caused by bacterial canker during the previous growing and overwintering season (cm); RNI, rate of necrosis increase caused by the bacterial canker pathogen in a frozen environment during the last overwintering season; D, estimated damage to kiwifruit canes caused by bacterial canker during the previous growing and overwintering season (cm) =  $NNF \times (RNI + 1)$ .

canker pathogen in a freezing environment over winter (RNI) can be calculated as the sum of RNIH from October 1 at 1:00 to April 30 at 24:00.

**Model evaluation.** The amount of necrotic lesions caused by bacterial canker on kiwifruit canes in a non-frozen environment during the overwintering and growing seasons (NNF) was highest at Wando in 1995 and lowest at Seogwipo1 in 2015 (Table 1). The rates of necrosis increase in a freezing environment during the overwintering season (RNIs) were zero at both Wando in 1995 and Seogwipo2 in 2015. The other RNIs were within the range of 3.13–14.33. The D-PSA-K model estimated that there would be no increase in damage caused by the bacterial canker pathogen with freezing damage during the overwintering season at Wando in 1995 and at Seogwipo1 in 2015, and that there would be damage increases of 4.13–15.33 times in the seasons. In both the Hayward and Hort16A cultivars, the estimated damage on kiwifruit canes caused by bacterial canker during the overwintering and growing seasons (D) was high when the observed DI was high.

## Discussion

We developed a model to estimate the accumulated potential damage on kiwifruit canes caused by bacterial canker during the last growing and overwintering seasons (D-PSA-K) and determine the maximum incidence of bacterial canker. The D-PSA-K model can be applied based on hourly and daily mean air temperature data.

To test the effect of temperature on bacterial canker development in kiwifruit canes, we used the *P. syringae* pv. *actinidiae* KACC16843 isolate. The isolate does not belong to the fourth clade responsible for the recent outbreak in Italy and New Zealand. Isolates belonging to the fourth clade have already been observed in Korea. The

mode of pathogenic action of the isolates often collected in Korea is different from the mode of those belonging to the fourth clade (Han et al., 2003a; Koh et al., 2012; Vanneste et al., 2011a). The fourth clade showed significantly lower pathogenicity on Hayward than on the other cultivars, including Hort16A (Balestra et al., 2009). For Hayward, bacterial canker caused by the fourth clade may be rare compared with the disease caused by the Korean clade, including the isolate used in this study. In other cultivars, including Hort16A, the amount of pathogenicity of the fourth clade will need to be tested to develop a more precise model for the cultivars.

We tested the effect of temperature on bacterial canker development in kiwifruit canes using overwintered Hayward canes, but without leaves or roots. If whole plants with leaves and roots had been tested, inoculation using the culture suspension might not have resulted in such long necrotic lesions under the bark as plant growth during the growing season could compensate for the necrosis caused by inoculation. Additional factors, including the diameter and age of canes, could also affect necrotic lesion development on canes. To that end, we did not test the effect of temperature on bacterial canker development on various kiwifruit canes in nature, nor did we compute the equation representing the relationship between temperature and lesion development on kiwifruit canes. The equation developed in this study is only relevant under the assumption that there was no growth and that the diameters of all kiwifruit canes were about 10 mm. Consequently, the resulting equation would describe the relationship between temperature and lesion development on hypothetical kiwifruits, and might not be useful for estimating the lesion progress or the time of symptom appearance on kiwifruit.

The amount of lesion development on hypothetical kiwifruits that do not grow can be considered equal to the potential maximum damage caused by the colonization

and multiplication of infected or migrated pathogens. The resulting equation could be used to estimate the amount of potential damage within the temperature ranges that we tested. In addition, a test using whole plants would not be suitable for developing a model, as it is almost impossible to have many canes in whole plants with no significant differences among them. Whereas the longest distances between inoculated parts and exuded spots of bacterial ooze in new canes were 20–66 cm 28 days after inoculation in April (Serizawa and Ichikawa, 1993a), necrotic lesion development under the bark of overwintered canes was not observed at 15°C 3 and 6 days after inoculation (Fig. 1). Ko et al. (2000a, 2000b) also showed that the length of lesions caused by the pathogen on overwintered canes at 15°C during 18 days is 5–8 mm. Ko et al. (2000a) explained that it would come from the difference in speed of sap flow.

Whereas temperatures within the range of 12°C to 18°C favor disease development caused by the pathogen on new canes (Serizawa and Ichikawa, 1993b), the temperatures did not favor necrotic lesion development on overwintered canes (Fig. 1). It could come from the difference in speed of sap flow and resistance of tissue between new and overwintered canes. Once inside the plant, infections caused by the pathogen can occur on the majority of tissues and rapidly become systemic through the bacterial migration inside the xylem (Spinelli et al., 2011). High speed of sap flow at more than 20°C would enable rapid migration of the pathogen and the pathogen could cause long necrotic lesions at more than 20°C. In addition, the pathogen clades that are mainly isolated and cause bacterial canker in Korea and Japan can grow at 30°C (Choi et al., 2014) whereas the symptoms caused by the pathogen were not discovered on new canes at 25°C (Serizawa and Ichikawa, 1993b). It indicated that the pathogen could invade susceptible tissues and cause the symptoms at 30°C.

The research of Ko et al. (2000a, 2000b), designed to investigate the effect of freezing on bacterial canker development in kiwifruit cane, was also conducted using overwintered Hayward canes without leaves or roots. As there was no growth under the tested freezing conditions, results from a test using whole plants might have been similar to those from our test using kiwifruit canes without leaves and roots. The equation obtained by re-analyzing the data reported by Ko et al. (2000b) could show the relationship between freezing and bacterial canker lesion development on the canes of normal kiwifruits, within the range of –10°C to –3°C.

As D-PSK-K estimates the amount of necrotic lesions on kiwifruit canes caused by bacterial canker during the last growing and overwintering seasons, the best way for evaluating the accuracy of model would be the compari-

son of the estimated and the observed amount of necrotic lesions on kiwifruit canes. However, the necrotic lesion cannot be detected from the outside view and we cannot measure the amount of necrotic lesions without unrecoverable damage on kiwifruit canes. It is actually impossible to observe the amount in kiwifruit orchards. In order to evaluate the accuracy of model, we had to compare the estimated with something that can be measured and could be directly proportional to the amount of necrotic lesions in kiwifruit orchards. We evaluated the accuracy of D-PSA-K by comparing the observed maximum DI on kiwifruit canes with the damage estimated by the model in this study.

In both Hayward and Hort16A, the D-PSA-K model estimated high accumulated damage on kiwifruit canes caused by bacterial canker during the overwintering and growing seasons, when the observed maximum DI was large (Table 1). D-PSA-K could roughly predict the amount of disease occurrence in a kiwifruit orchard, relative to the past disease occurrences in that orchard. For the Hayward cultivar, D-PSA-K estimated the accumulated damage as 7.3–11.1 times the observed maximum DIs, and the relationship equation between the accumulated damage (D) and the observed maximum DI. These results were obtained from linear regression analysis using the data in Table 1, where  $DI = 0.1158D$  and  $R^2 = 0.89$ . The results indicated that the maximum DI for the Hayward cultivar could be predicted with high accuracy using D-PSA-K, and that the accumulated potential damage calculated using D-PSA-K could estimate the actual damage caused by the bacterial canker well. We did not observe such a relationship for Hort16A. These differences might be caused by differences in resistance to low temperatures and pathogen differences between Hayward and Hort16A (Balestra et al., 2009; Ferrante and Scortichini, 2014; Koh et al., 2010). Differences in the resistance of kiwifruit cultivars to the bacterial canker in Korea are not clearly understood. Outbreaks of bacterial canker causing the destruction of an entire orchard have been observed on Hort16A in Korea, but no such outbreaks on Hayward have been reported over the past two decades (Koh et al., 2010). These results indicate that Hort16A might be more susceptible to bacterial canker caused by the pathogen belonging to the Korean clade than is Hayward. In addition, *A. deliciosa*, including Hayward, is more frost tolerant than *A. chinensis*, as represented by Hort16A (Ferrante and Scortichini, 2014). For Hort16A, D-PSA-K also estimated the accumulated damage as 0.96 or 6.52 times the observed maximum DI, indicating that Hort16A might be more susceptible to bacterial canker in Korea than is Hayward. D-PSA-K might be used to estimate trends of real disease occurrences, although the model could only

accurately estimate the maximum DI for Hayward. Some adjustments of D-PSA-K, including consideration of differences between Hayward and other cultivars in terms of disease resistance and frost tolerance, might enable more precise estimation for the other cultivars.

As reported by the Rural Development Administration of Korea in 2009, Hayward was cultivated on 918 ha, whereas the other cultivars were cultivated on 132 ha in 2008 in Korea. Hayward is the most widely cultivated in Korea and, as a result, D-PSA-K would be useful for most kiwifruit orchards there. D-PSA-K would be also useful for summarizing disease occurrence on kiwifruit stems at regional or national scales, assuming that all kiwifruits cultivated in Korea are Hayward.

In conclusion, we developed a model to estimate the accumulated potential damage on kiwifruit canes caused by bacterial canker during the previous overwintering and growing seasons. The D-PSA-K model could estimate maximum DI on the stems of Hayward, as an indirect measure of yield loss caused by the bacterial canker in a given year. Although the model could estimate the maximum DI only for Hayward, D-PSA-K might also be used to estimate disease occurrence in a kiwifruit orchard compared to past occurrences, and to estimate the trends of real disease occurrences in other cultivars. D-PSA-K could help kiwifruit growers select new sites for kiwifruit cultivation and establish an improved production plan by predicting the loss in kiwifruit production due to bacterial canker, using past or predicted weather data.

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