

Leaf-wetness duration and temperature required for infection of saskatoon leaves by *Entomosporium mespili* under controlled conditions

Q.A. Holtslag, W.R. Remphrey, W.G.D. Fernando, R.G. St-Pierre, and S. Panicker

Abstract: The influence of leaf-wetness duration and temperature on infection of *Amelanchier alnifolia* (saskatoon) by *Entomosporium mespili* was quantified in controlled-environment studies. Plants were inoculated with a conidial suspension and then subjected to a period of leaf wetness (0, 6, 12, and 24 h), after which they were moved to a growth room set at a fixed temperature (10, 15, 20, 25, and 30°C) for a period of 14 d. Infection levels rose as the leaf-wetness duration increased for combined temperature treatments up to 24 h. Temperatures between 10 and 20°C promoted optimal leaf infection for leaf-wetness periods longer than 6 h. Above the optimal temperature range, mean disease levels decreased. ANOVA with orthogonal contrasts and stepwise regression were used to create a regression equation that describes the relationship of *E. mespili* infection on saskatoon to both temperature and leaf-wetness duration. Combinations of temperatures between 10 and 25°C and leaf-wetness periods between 6 and 24 h were optimal for lesion development on younger saskatoon leaves.

Key words: *Entomosporium mespili*, leaf-wetness duration, temperature, saskatoon.

Résumé : L'influence de la durée de mouillure des feuilles et de la température sur l'infection de l'*Amelanchier alnifolia* (amélanchier à feuilles d'aulne) par l'*Entomosporium mespili* fut quantifiée lors d'études en environnement contrôlé. Les plantes furent inoculées avec une suspension de conidies et soumises à une période de mouillure des feuilles (0, 6, 12 et 24 h), après quoi elles furent placées dans une chambre de croissance ajustée à une température constante (10, 15, 20, 25 et 30°C) pour une période de 14 jours. Jusqu'à 24 h, les niveaux d'infection ont augmenté avec la durée de mouillure des feuilles pour l'ensemble des traitements de température. Les températures entre 10 et 20°C ont favorisé une infection optimale des feuilles pour une durée de mouillure des feuilles plus longue que 6 h. Au-dessus de la fourchette des températures optimales, les niveaux moyens de maladie ont diminué. Une analyse de variance par contrastes orthogonaux et une analyse de régression par degrés furent utilisées pour établir une équation de régression qui décrit la relation entre l'infection de l'amélanchier par l'*E. mespili* d'une part, et la température et la durée de mouillure des feuilles d'autre part. Les combinaisons des températures entre 10 et 25°C, et des durées de mouillure des feuilles entre 6 et 24 h furent les meilleures pour le développement des lésions sur les plus jeunes feuilles d'amélanchier.

Mots clés : *Entomosporium mespili*, durée de mouillure des feuilles, température, saskatoon.

Introduction

The saskatoon (*Amelanchier alnifolia* Nutt.) is a perennial, woody, fruit-bearing shrub from the apple subfamily Pomoideae, within the Rosaceae. Depending on geographical region, saskatoon plants have also been referred to as

Juneberry or serviceberry (Steeves and Steeves 1991). Fruit are characterized as a pentalocular pome (McGarry et al. 1998; St-Pierre 1997). Aboriginal people and early settlers treasured the saskatoon plant for its sweet, distinctively flavored fruit with subtle almond overtones (St-Pierre 1997).

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The saskatoon is capable of growing in a wide range of soils and climatic conditions and is native to the Canadian prairies, Northwest Territories, Yukon, Alaska, British Columbia, and the northwestern and north central United States. Lange et al. (1998) indicated that saskatoons are an important commercial crop in the provinces of Alberta, Saskatchewan, and Manitoba, Canada. Much of the saskatoon crop is handpicked and sold via the “U-Pick” marketing system, for which an aesthetically pleasing product is important (Lange et al. 1998).

The main challenge to economic production of saskatoon is considered to be entomosporium leaf and berry spot disease, which is caused by the fungal pathogen *Entomosporium mespili* (DC.) Sacc. (Lange and Bains 1994). *Entomosporium mespili* is found worldwide and can cause necrotic spots on leaves, fruit, and succulent stems of more than 60 species in the subfamily Pomoidae of the Rosaceae (Sinclair et al. 1987). Susceptible genera include *Amelanchier*, *Chaenomeles*, *Cotoneaster*, *Crataegus*, *Cydonia*, *Eriobotrya*, *Heteromeles*, *Malus*, *Mespilus*, *Phaphiolepis*, *Photinia*, *Prunus*, *Pyracantha*, *Pyrus*, *Sorbus*, and *Stanvaesia* (Horie and Kobayashi 1980; Sinclair et al. 1987). Lesions caused by the pathogen initially develop as minute dots and, when fully developed, appear as brown to gray, irregular spots, 2–5 mm in diameter. If lesions are numerous, they coalesce to form larger necrotic patches and may cause early season leaf fall. Loss of leaf area and leaves would reduce photosynthate available for fruit production. Furthermore, inoculum produced on infected leaves can be spread to fruit and cause infection. Disease development on saskatoon fruit results in spotting, cracking, and advanced desiccation (Pescic-Van Esbroeck and Bains 1991), so it is essential to prevent epidemic development prior to fruit harvest. Saskatoon fruit with more than 6% of the surface area affected is considered unusable, even for processed products (St-Pierre 1997).

Specific weather conditions are often important in causing disease levels to increase within a crop (Jones 1986). Empirically determined relationships between environmental factors and disease have been used to time fungicide sprays for many diseases (Grove 2002; Thompson and Jenkins 1985). Therefore, management of entomosporium leaf and berry spot disease should depend on knowledge of the environmental conditions that promote pathogen infection and development. In the case of saskatoons, field observations suggest that *E. mespili* development is promoted by warm, moist conditions (Davidson 1990; Horie and Kobayashi 1979; Sinclair et al. 1987; St-Pierre 1997). Lange and Bains (1995) indicated that heavy rainfall is the most important environmental factor contributing to *E. mespili* increase in saskatoon orchards. Baudoin (1986a) showed that the optimum temperature for *E. mespili* infection on *Photinia × fraseri* (DC. ex DUBY) Sacc. is 20°C and that disease levels increase as the duration of leaf wetness increases. However, the specific temperature and leaf-wetness conditions required to promote *E. mespili* development on saskatoon plants are not known. Moreover, a recent study has indicated that disease development depends on leaf age, in that young saskatoon leaves are more susceptible to infection than older leaves (Ronald et al. 2001) and, therefore, the effect of leaf age on disease development in relation to tem-

perature and leaf-wetness duration is also not understood. In this context, the objectives of this study were: (1) to quantify the temperature and leaf-wetness durations required for causing entomosporium leaf and berry spot on saskatoon plants, and develop a regression equation for this relationship and (2) to identify the temperature and leaf-wetness conditions that provide optimal lesion development on young versus old leaves.

Materials and methods

Production of plants

Dormant, 2-year-old saskatoon plants of cultivar ‘Smoky’, derived from tissue culture, were obtained from D’nA Gardens, Red Deer, Alta. Each plant consisted of one main stem that ranged in length from 10 to 25 cm. The plants were kept dormant in cold storage (0°C, 30% RH) for 75 to 105 days after arrival. There were fewer growth rooms available than temperature treatments, so the experiment was conducted over time, resulting in one set of inoculations per temperature and leaf-wetness period. Groups of plants selected for each temperature treatment were taken out of cold storage in 14-day intervals to ensure that each group was at the same stage of phenological maturity for each temperature treatment. Each plant was placed in a pot filled with a mixture of soil, sand, and peat moss (1:1:1, v:v). Granular fertilizer (40 g of 11–53–0) was added to the mix at the time of planting. Subsequent applications of 20–20–20 (N–P–K) were applied during watering at 2-week intervals after planting. The plants were grown in a greenhouse maintained at 20 ± 2°C during the light period (16 h) and at 18 ± 2°C in the dark (8 h). The branches of each plant were sprayed once a week with a 250-ppm gibberellic acid solution to stimulate bud break (Sumner et al. 1999). This planting procedure helped to ensure the plants were leafed-out and ready for inoculation approximately 1 month after the planting date.

Inoculum production

Leaves and fruit that were heavily infected with *E. mespili* were collected from a mature saskatoon orchard located near Winnipeg, Man., in September of 1998 and 1999, and used as a source of inoculum for the first and second repetitions of the study, respectively. This material was air dried and stored in a sealed plastic container at 5°C until needed. Infected leaves and fruit were allowed to acclimate at 20°C for 3 days before preparing the conidial suspension. About 40 leaves and five fruits were placed in a beaker containing 200 mL of distilled water. The suspension was vigorously agitated with a stir rod for 20 min to stimulate release of the conidia from the acervuli. Plant debris was filtered from the suspension and the number of conidia per millilitre was determined on an hemacytometer. The suspension was diluted to approximately 10⁵ conidia/mL. Before the plants were inoculated, two drops of Tween 20 were added to the suspension to ensure good leaf surface contact. The conidial suspension was agitated continuously, until it was applied to the plant, to ensure that the conidia were evenly distributed in the suspension.

Treatment and inoculations

In the first repetition of the study, two leaf-wetness periods were tested (12 and 24 h), each at one of four temperatures (15, 20, 25, and 30°C). Given the limitation in growth room resources, 24 h was selected as the maximum period of leaf wetness in accordance with other crop disease studies (Grove 2002; Monroe et al. 1997). Moreover, leaf-wetness duration in Manitoba, which is one of the wetter areas of the Prairies, typically remains well below 24 h, based on our field monitoring (data not shown). After analyzing the results, two leaf-wetness periods (0 and 6 h), and one temperature treatment (10°C) were included in the second repetition of the study. This was done to better assess the minimum temperature and hours of leaf wetness required for infection. Each trial had control plants that did not receive inoculation or a period of leaf wetness. Temperature treatments were randomized within each study.

Plants were inoculated with an air atomizer operated at 20 psi (1 psi = 6.895 kPa) until runoff. Inoculated plants were randomly placed in a mist chamber assembled from polyethylene plastic sheets and positioned within a growth room set at 18°C. Continuous mist was provided from a mist humidifier located to one side of the humidity chamber and above the plant canopy. After removal from the mist chamber, each pot was placed in an acetate sleeve to prevent interplant contact, and in a dish to trap excess water runoff after watering. The plants were then moved to another growth room preset at the desired temperature treatment and a constant relative humidity of 30%. The growth room provided a constant temperature and a 16-h light to 8-h darkness photoperiod over the 14 days of the temperature-treatment period. Light for the 16-h day period was supplied from both fluorescent and incandescent bulbs. Each plant was kept adequately watered during the entire study by applying water to the soil. Plants were moved randomly within the growth room every second day to minimize the impact of variable conditions within the growth room itself.

Disease evaluation

Plants were examined for leaf lesions every second day after the first day of each 14-day temperature-treatment period. After the 14-day temperature-treatment period, five leaves from the top and five from the bottom half of each plant were randomly sampled to compare susceptibility of the young leaves found near the apex of the plant, with physiologically older leaves found near the base of the plant. Disease severity at the end of each trial was determined by calculating the percent-infected leaves per plant (PINFL):

$$[1] \quad \text{PINFL} = (X/Y) \times 100$$

where X is the number of infected leaves on a plant and Y is the total number of leaves on each plant. The mean number of lesions per leaf (NLPL) was calculated for each plant with the following equation:

$$[2] \quad \text{NLPL} = Z/10$$

where Z is the total number of lesions counted on the sample leaves and 10 is the number of sample leaves. Using Statistix (Analytical Software, Tallahassee, Fla.), a paired t test was performed to see if each temperature and leaf-

wetness interaction produced a significant difference in the NLPL on the top leaves compared with the bottom leaves on each of the plants. It was determined that there was a significant difference in the susceptibility of young and old leaves if $P < 0.05$.

Regression-model analysis

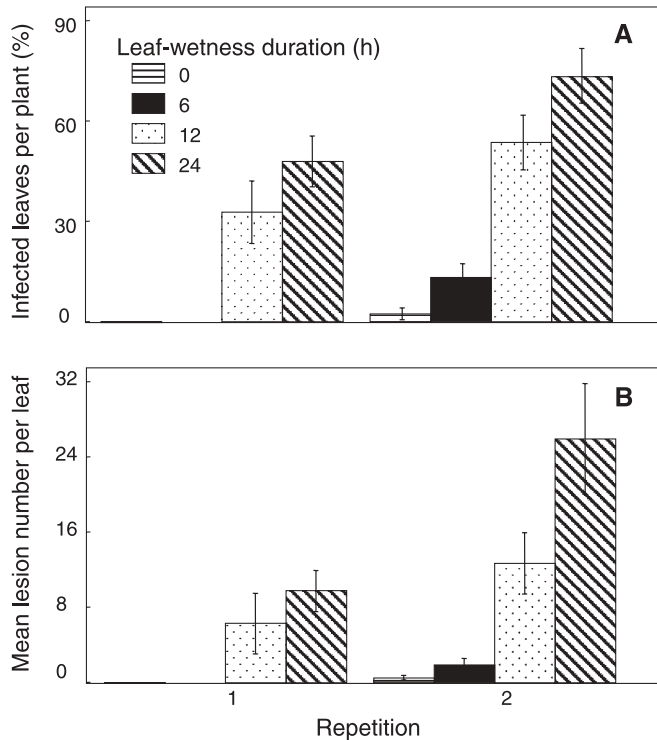
An F test was performed to determine if combining the data from both experiments was warranted (Bulger et al. 1987). The relationship between PINFL and NLPL was assessed using Pearson's correlation coefficient analysis. In the treatment with absence of leaf wetness, few disease symptoms were observed in the second repetition of the study, resulting in disease-severity data that were not normally distributed. Therefore, data were log transformed, $[\log_{10}(Y + 2)]$, prior to analysis of variance. The relationship between temperature and leaf-wetness duration for infection by *E. mespili* was described following analysis of variance and multiple-regression procedures. Linear, quadratic, and cubic effects of temperature and leaf-wetness duration and their interactions on PINFL were tested. Based on Statistical Analysis System software (SAS Institute Inc., Cary, N.C.) stepwise regression, parameters not significantly different from zero ($P < 0.05$) were omitted from the equation, using backward elimination, unless higher-degree terms of the corresponding variable were associated with significant parameters. This stepwise procedure was conducted until a simple model with the best distribution of parameter estimates was derived. Using the simplified regression model, the coefficient of determination (R^2) and the pattern and distribution of residuals were examined for each repetition. A coefficient of determination (R^2) was also calculated for both repetitions combined.

Results

When the temperature treatments for each leaf-wetness period were combined, both the PINFL and NLPL increased with an increase in the leaf-wetness period (Fig. 1). In general, disease-severity trends relative to increasing leaf-wetness periods were similar in both repetitions of the study, despite the fact that the first repetition did not have as many treatments as the second, and PINFL and NLPL values were both higher in the second repetition. In the second repetition, a small number of lesions formed without a period of leaf wetness when the plants were exposed to temperatures of 20 or 30°C after inoculation.

An F test indicated that the results of the two studies were not significantly different from each other ($P < 0.05$). Therefore, the data from the two repetitions were pooled. Symptoms were not observed on uninoculated plants. Increasing the leaf-wetness period after the plants were inoculated increased the PINFL and NLPL for all temperature treatments (Fig. 2). Considerably more infected leaves and lesions per leaf developed at temperatures between 10 and 20°C and when leaf-wetness periods were greater or equal to 12 h. There was a significant correlation ($r = 0.78$) between combined data of PINFL and NLPL, so only PINFL was used for regression analysis. Percent-infected leaves per plant provided an effective measure of disease development over the entire plant. The relationship between tem-

Fig. 1. *Entomosporium mespili* development on saskatoon plants relative to leaf-wetness duration (0, 6, 12, and 24 h) for combined temperature treatments from two repetitions of a growth-room study. (A) Mean (\pm standard error) percent infected leaves per plant (PINFL). (B) Total mean (\pm standard error) lesion number per leaf (NLPL). No disease development was observed in the first repetition, when the leaf-wetness duration was 0 h. There was no treatment for 6 h of leaf wetness in the first repetition of this study, therefore, the space left for this column is blank.



perature and leaf-wetness duration in regards to PINFL is as follows:

$$[3] \log_{10}(Y + 2) = -b_0 + b_1T - b_2T^2 + b_3T^3 + b_4W - b_5W^2 - b_6TW + b_7T^2W - b_8T^3W$$

where *Y* is the predicted PINFL, *T* is temperature, and *W* is leaf-wetness duration. The significant parameter estimates for [3] are listed in Table 1. The *R*² values were 0.94 and 0.85 during the first and second repetitions, respectively, and a random pattern of residuals was observed across the range of predicted means. When the repetitions were combined, the *R*² value was 0.79.

Younger leaves closest to the top of the plant were generally more susceptible than older leaves found near the bottom of the plant for each temperature and leaf-wetness treatment (Table 2). At 15°C, NLPL differences were significant at 6, 12, and 24 h of leaf wetness. As the leaf-wetness period increased, so did the temperature range at which there was a significant difference in the NLPL on the top versus bottom leaves.

Discussion

This study demonstrated that the duration of leaf wetness is an important factor for entomosporium leaf and berry

Fig. 2. Effect of temperature and leaf-wetness duration on *Entomosporium mespili* development on saskatoon plants. (A) Mean (\pm standard error) percent infected leaves per plant (PINFL). (B) Total mean (\pm standard error) lesion number per leaf (NLPL).

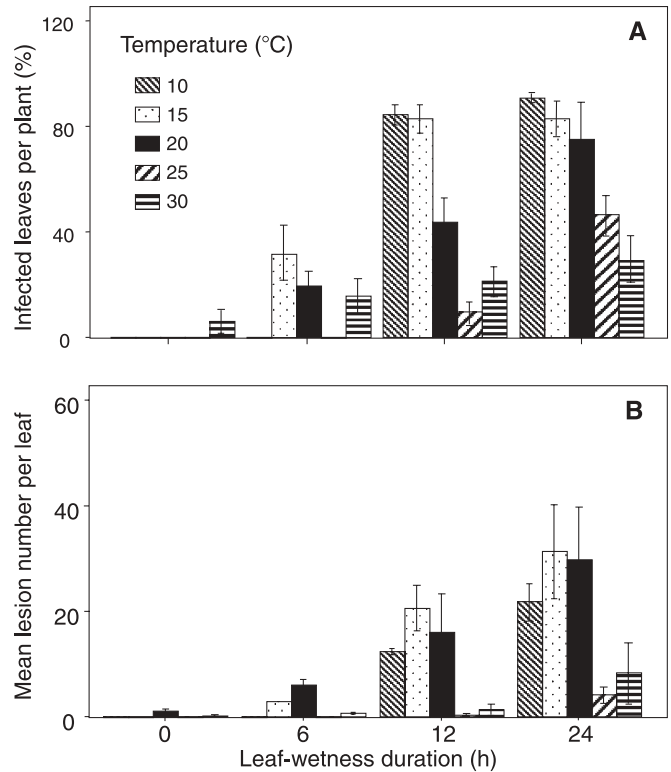


Table 1. Significant parameter estimates and their standard errors used in empirical model [3] to forecast the effects of temperature and leaf-wetness duration on infection of saskatoon leaves by *Entomosporium mespili*.

Parameter	Estimate	Standard error
<i>b</i> ₀	-23.3187	5.6821
<i>b</i> ₁	4.0839	0.9793
<i>b</i> ₂	-0.2190	0.0520
<i>b</i> ₃	0.0037	0.0009
<i>b</i> ₄	1.3094	0.4158
<i>b</i> ₅	-0.0082	0.0019
<i>b</i> ₆	-0.1625	0.0712
<i>b</i> ₇	0.0089	0.0038
<i>b</i> ₈	-0.0002	0.0001

spot disease development on saskatoons. There was a large increase in disease development with duration of leaf wetness of 24 h. However, as our study did not look at leaf-wetness periods beyond 24 h, it was not possible to determine if 24 h was the optimal leaf-wetness period for infection of saskatoon. Similarly, studies of other pathogens (Grove 2002; Monroe et al. 1997) and *E. mespili* on *Photinia xfraseri* (Baudoin 1986a) also revealed a pattern of increased disease with increasing leaf wetness. In these studies, a period of 24 h was the longest duration examined.

Table 2. Effect of temperature and leaf-wetness duration on the mean number of lesions per leaf caused by *Entomosporium mespili* on saskatoon.

Temperature (°C)	Leaf-wetness duration (h)							
	0		6		12		24	
	Top	Bottom	Top	Bottom	Top	Bottom	Top	Bottom
10	0	0	0	0	20.6*	4.1	33.9*	9.6
15	0	0	4.7*	0.9	23.5*	17.5	46.3*	16.2
20	0	0	2.1	0.3	18.4*	2.3	33.9*	12.0
25	0	0	0	0	0.3	0.2	6.1*	2.1
30	0.4	0.2	0.8	0.5	2.0	0.6	11.8	4.8

Note: Samples consisted of five leaves from the top portion (young leaves) and five leaves from the bottom portion (older leaves) of each plant.

*Data followed by an asterisk indicate a significant difference between the mean number of lesions per leaf on the top versus bottom leaves for individual treatment combinations.

Along with leaf wetness, temperature also had an important impact on infection. Temperature influences both pathogen development and plant growth (Harrison 1992). In the growth-room study, saskatoon stem basal diameter grew minimally at 10 and 30°C, and optimally at 20°C (data not shown). For pathogen growth and development, temperatures above 20°C suppressed *E. mespili* leaf infection and lesion development on saskatoons, while maximum infection of saskatoon by *E. mespili* occurred between 10 and 20°C after 24 h of leaf wetness post inoculation. Baudoin (1986a) showed that the optimum temperature for *E. mespili* development on *Photinia xfraseri* was 20°C; however, infection was only slightly less at 15 and 25°C. In contrast, the present study showed that infection was reduced on saskatoon plants when the temperature was 25°C or higher. Baudoin (1986a), Horie and Kobayashi (1979), and Rosenberger (1981) indicated that 20 to 25°C is the optimal temperature range for *E. mespili* conidia to infect different host plants. Besides the effect of temperature on disease development, some preliminary data from the growth-room experiments suggests that the latent period decreases with increasing temperature up to 20°C and then stays approximately constant (data not shown). Such a response could potentially have the effect of reducing the duration of each infection cycle, and ultimately reduce the time required for an epidemic to develop (Fry 1982). Further work is needed with larger sample sizes to confirm the relationship of temperature and leaf-wetness duration with the latent period required for disease development.

Leaf wetness is not only important for disease development, but its duration will also influence the minimum temperature required for disease to develop. Although our study showed that leaf wetness is important for disease development, a few lesions developed without leaf wetness at temperatures of 20 and 30°C in the second repetition of the study. This could have resulted from the stomata being open more frequently when the temperature was 20 or 30°C. Although conidia of *E. mespili* typically germinate and then form an appressorium that directly penetrates the leaf cuticle, van der Zwet and Stroo (1985) showed that *E. maculatum* Lév., which is a synonym for the anomorphic state of *E. mespili*, can enter and infect pear leaves through their stomata. This may explain why disease symptoms increased when the temperature was raised to 30°C for an extended period.

The present study suggests that the relationship between temperature and leaf-wetness duration can be used to predict the likelihood of disease-symptom development. Similar relationships between temperature and leaf wetness have been well documented for a variety of plants and diseases (Evans et al. 1992; Mathieu and Kusalappa 1993; Monroe et al. 1997). Although the regression equation developed here applies to the controlled environmental conditions of the present study, it lays the foundation for the development of a model for fungicide spray scheduling under field conditions. When used in combination with a fungicide application program in the field, this regression equation has the potential to help reduce early-season leaf infection. A reduction in the amount of diseased tissue on plants has the potential to limit inoculum abundance for saskatoon fruit infection. Currently we are investigating conidia release relative to fruit development and maturation. Nevertheless, it is still conceivable that fruit may become infected directly from inoculum that is released from overwintering structures that can be found on young branch tissue, dead infected leaves, and possibly on infected pedicels and fruit that remain on or around the plant (Horie and Kobayashi 1980; Stathis and Plakidas 1959).

Tissue susceptibility to a particular pathogen can vary with organ age. In our experiment, young newly expanded saskatoon leaves near the top of the plant tended to develop more lesions per leaf on average than older, more physiologically mature leaves on the lower half of the plant. This distinction was particularly evident when temperature and leaf-wetness durations were optimal. Ronald et al. (2001) found that young saskatoon leaves were more susceptible to infection than older leaves. Baudoin (1986b) also showed that *E. mespili* infection is more aggressive on young leaves of *Photinia xfraseri* than on older leaves. Jacobs et al. (1996) showed that young leaves of *Photinia* species could be infected by *E. mespili* 10- to 30-fold more often than mature leaves.

Understanding how the chemical and (or) physical condition of a leaf changes as it matures provides insight into how older leaves become more resistant to symptom development. For example, Wetzstein and Sparks (1983) noted that higher levels of resistance in older leaves of *Carya illinoensis* Wang. were associated with low trichome density, high phenolic content in the mesophyll, and thick cuticles. As a plant leaf begins to age, waxes are built up on

their surface. Waxes on leaf and fruit surfaces form a water-repellent layer and thereby prevent the formation of a film of water in which pathogens might be deposited and germinate or multiply (Agrios 1997). *Entomosporium mespili* primarily infects plants like *Photinia* through direct cuticle penetration and, therefore, a thicker cuticle would provide superior physical protection from infection.

In conclusion, we created a regression equation that defines disease development based on the interaction between temperature and leaf-wetness duration. We also showed that susceptibility of young and old leaves depend on the temperature and leaf-wetness duration. A disease-forecasting model, which is currently being developed, will incorporate the empirical model developed in this study with host susceptibility, inoculum levels present in the field, and time of conidia release to create a mechanistic model describing *E. mespili* development on saskatoon plants.

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