

The development of a dynamic disease-forecasting model to control *Entomosporium mespili* on *Amelanchier alnifolia*

Q.A. Holtslag, W.R. Remphrey, W.G.D. Fernando, R.G. St-Pierre, and G.H.B. Ash

Abstract: Entomosporium leaf and berry spot, which is caused by the fungal pathogen *Entomosporium mespili*, can cause up to 100% yield loss in *Amelanchier alnifolia* (saskatoon) in years when weather conditions are conducive to disease development. In an effort to optimize the effectiveness and minimize the use of fungicides, a dynamic disease-forecasting model was developed. The model uses a disease pressure index equation, which integrates information regarding the phenological development of saskatoon, the relationship of disease to inoculum potential and production, leaf-wetness duration and temperature, inoculum release, and host susceptibility, to provide an estimate of disease pressure. When the model was evaluated, a strong correlation was found between predicted disease pressure and observed disease symptom development. After the field data were combined, the model could account for 82% of the variation in the increase of mean lesion number per leaf observed during the preharvest period. Control thresholds, together with application and control guidelines for use of the fungicide propiconazole, were added to the model, which was then evaluated in a field trial. A preliminary field test of the model showed that its use resulted in reduced disease development prior to the completion of fruit harvest.

Key words: dynamic disease-forecasting model, disease pressure index, entomosporium leaf and berry spot, saskatoon.

Résumé : L'entomosporiose, causée par le champignon pathogène *Entomosporium mespili*, peut engendrer jusqu'à 100 % de perte de rendement chez l'*Amelanchier alnifolia* (amélanchier à feuilles d'aulne) lors des années où les conditions climatiques sont favorables à la maladie. Dans le but d'optimiser l'efficacité et de diminuer l'usage de fongicides, un modèle dynamique de prévision de la maladie fut développé. Afin de fournir un estimé de la pression de maladie, le modèle utilise une équation de l'indice de pression de maladie qui intègre de l'information sur le développement phénologique de l'amélanchier, la relation entre la maladie et le potentiel et la production d'inoculum, la durée et la température d'humectation des feuilles, la libération de l'inoculum et la sensibilité de l'hôte. Lors de l'évaluation du modèle, une forte corrélation entre la pression de maladie prédite et les symptômes de maladie observés fut remarquée. Après combinaison des données de terrain, le modèle pouvait rendre compte de 82 % de la variation dans l'augmentation du nombre moyen de lésions par feuille observées pendant le délai avant récolte. Les seuils d'intervention, ainsi que les directives d'application et de vérification pour l'usage du fongicide propiconazole, furent ajoutés au modèle qui fut par la suite évalué lors d'essais en champ. Un test préliminaire du modèle sur le terrain a montré que son utilisation entraînait une diminution du développement de la maladie avant la fin de la récolte des fruits.

Mots clés : modèle dynamique de prévision de la maladie, indice de pression de maladie, entomosporiose, amélanchier à feuilles d'aulne.

Introduction

Amelanchier alnifolia Nutt., commonly known as saskatoon, is a perennial, woody, fruit-bearing shrub that is native to the Canadian Prairies. Currently, there are about 1200 ha of saskatoons planted throughout Canada. However, profitability of saskatoon fruit production is limited by entomosporium leaf and berry spot, caused by the fungal pathogen *Entomosporium mespili* (DC.) Sacc. (Lange and Bains 1994). Disease severity can increase rapidly during the growing season because of the pathogen's short incubation period and polycyclic nature (Sinclair et al. 1987; van der Zwet and Stroo 1985). Disease development is depend-

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ent on microclimate (Holtstag et al. 2003a), host susceptibility (Holtstag et al. 2003a; Ronald et al. 2001), and the production and release of inoculum (Holtstag et al. 2003b). The necrotic angular lesions that form on leaves may eventually coalesce, causing the entire leaf to become chlorotic and abscise prematurely (Bains 2000). A reduction in healthy leaf area leads to a decrease in photosynthate available for fruit production (Horie and Kobayashi 1979; St-Pierre 1997), and the inoculum produced on infected leaves can be disseminated to developing fruit and thereafter cause infection. Disease development on saskatoon fruit results in spotting, cracking, and advanced desiccation.

One way of improving management and control of entomosporium leaf and berry spot is to determine conditions conducive to disease development and use this information to develop a model to forecast potential outbreaks. A disease-forecasting model can reduce uncertainty about control decisions by providing a quantitative description of disease pressure (Tait 1987). A model has already been developed to illustrate the relationship between entomosporium leaf and berry spot development and the interaction between leaf-wetness duration and temperature (Holtstag et al. 2003a). However, even if ideal weather conditions for disease development were present, they would be of little value in forecasting a possible disease outbreak if inoculum is not present, or if the host is not susceptible to infection at that point in time. Therefore, to account for these variables, several regression equations could be integrated to describe the interactions between the pathogen and each component of its environment. Once a disease-forecast model has been constructed, it must be validated by comparing observed symptom development from laboratory and (or) field evaluation trials against predicted values that have been generated by regression models (Asher and Williams 1991; Grove 2002; Pfender 2003).

If disease development can be accurately forecasted, the next logical step is to expand the model to incorporate control measure capabilities. Currently, there are three fungicides to control entomosporium leaf and berry spot on saskatoon plants in Canada: triforine (Funginex[®]), propiconazole (Topas[®] 250E), and sulfur (Kumulus[™] DF) (Bains 2000). The best disease control is attained using spray applications of propiconazole at the white-tip, petal-drop, and green-fruit stages (Lange et al. 1998; St-Pierre 1997). Once applied, the fungicide is effective for a period of up to 21 d as long as no rain event occurs within 1 h after application and the plant is not actively growing. Current spray recommendations tend to promote overuse of the fungicide, given that anthesis in saskatoon plants lasts 3.5 ± 0.8 d (St-Pierre and Steeves 1990), with the white-tip and green-fruit stages occurring shortly before and after this period. Moreover, fungicide application programs assume that weather conditions required for disease development are present during this period, which is not always the case.

The objectives of this study were: (1) to create a dynamic disease-forecasting model to estimate the development of entomosporium leaf and berry spot in saskatoon orchards, based on phenological and epidemiological information; (2) to test the accuracy of a disease pressure index equation used in the disease-forecasting model to predict disease development in two saskatoon orchards over several years;

and (3) to expand the model to include control methods and conduct a preliminary test of its effectiveness.

Materials and methods

Model development and testing

The disease-forecast model was constructed using several kinds of data obtained from orchards of saskatoon 'Smoky' in Carman and Winnipeg, Manitoba, as described in Holtstag (2003). Over the course of the study, specific data were obtained to model the relationship of disease development relative to weather variables in these two orchards between 1999 and 2002 (Holtstag 2003). Using Adcon Telemetry (Adcon Telemetry, Klosterneuburg, Austria), weather stations were established in each orchard in 1999, which measured precipitation above the saskatoon plant canopy and leaf wetness and temperature at 1 m in height and 15 cm within the outer edge of the canopy. Because of equipment problems, weather data were not obtained from the Winnipeg site in 2000 and 2002, nor from Carman in 2000.

Phenological data were incorporated into the model to facilitate the timing of events related to plant growth, flowering, fruit harvest, and disease development. Specific details on how phenological data were obtained and analyzed are provided in Holtstag (2003). Data on the relationship of disease to inoculum production was obtained from Holtstag et al. (2003b), and the interaction between leaf-wetness duration and temperature is described in Holtstag et al. (2003a).

Once the model was developed, its accuracy in describing disease development in the field was evaluated at the Winnipeg and Carman orchard sites between 1999 and 2002. In 2002, the model was expanded to incorporate fungicide control measures, and subsequently a preliminary test of the expanded model was performed in the Carman orchard. Specific mechanisms used to develop this model will be elaborated in the description of model development below.

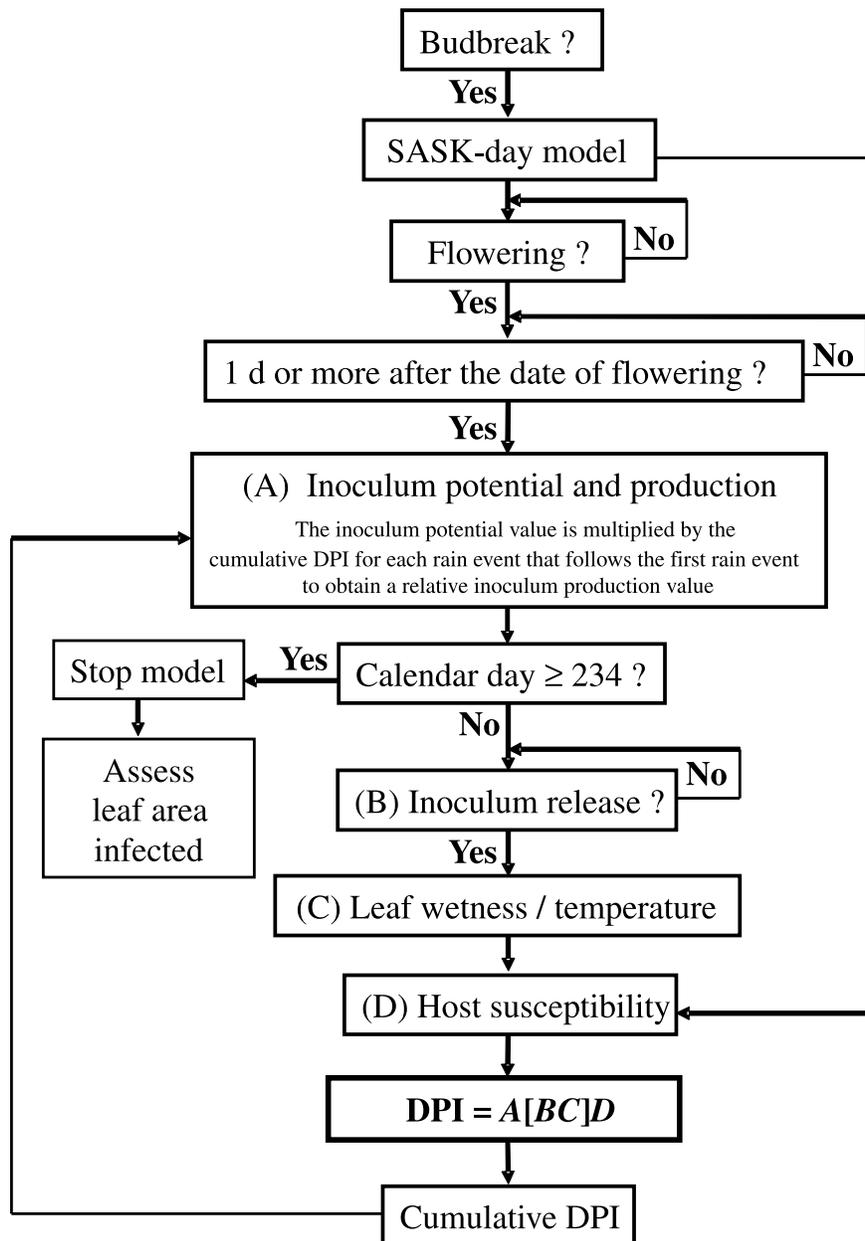
Dynamic disease-forecasting model development

Phenological development of saskatoon

The calendar date of budbreak was used to start the phenological component of the disease-forecasting model (Fig. 1). The minimum, optimum, and maximum temperature parameters of the potato physiological day model developed by Sands et al. (1979) were modified to suit the critical temperatures for saskatoon growth, creating a new model referred to as the SASK-day heat unit model (Holtstag 2003). Phenological development of saskatoon from the date of budbreak and flowering up to fruit harvest was predicted using actual SASK-day heat units measured at the Carman 2002 orchard site and mean historical daily SASK-day heat unit data collected between 1999 and 2001 at the Winnipeg and Carman orchards.

Once the phenological stage of budbreak is reached, the disease-forecasting model awaits the first rain event occurring 1 d or more after the date of flowering, because this coincides with the beginning of the infection period (Holtstag et al. 2003b). The date of flowering can be predicted using the phenological model, or observed visually and entered into the disease-forecasting model when more

Fig. 1. A schematic flow chart illustrating the dynamic disease-forecasting model developed for estimating disease pressure of entomosporium leaf and berry spot on Saskatoon in Manitoba. Inoculum potential and production (module A), leaf-wetness duration and temperature (module C), and host susceptibility (module D) are used to calculate the disease pressure index (DPI) after each inoculum release event (module B), which occur 1 d or more after the date of flowering.



than 50% of the inflorescences in the orchard have reached the flowering stage of development.

Disease pressure index

Disease pressure was assessed using a disease pressure index (DPI) equation (Fig. 1). Ultimately, DPI values are summed to produce a cumulative DPI value that is used as the basis for control measure recommendations on a real-time basis. Fry (1982) described a similar equation that considered the three components of the disease triangle to estimate disease severity; however, the DPI equation used in the current study comprised four modules: (1) an assessment of inoculum potential and production, (2) a signal for

inoculum release, (3) a calculation of disease development, based on the interaction between leaf-wetness duration and temperature, and (4) an estimation of host susceptibility. Equation 1 illustrates how the DPI was calculated after each inoculum release event throughout the growing season:

$$[1] \quad Y = A[BC]D$$

where Y is the DPI value, A is an estimate of the amount of inoculum, B signifies whether the inoculum is being released, C represents a measure of infection success based on leaf-wetness duration and temperature after inoculum release, and D is an assessment of host susceptibility.

Inoculum potential and production. Given that there is a direct relationship between the leaf area that is infected with entomosporium leaf and berry spot and inoculum production (Holtslag et al. 2003b), an estimate of orchard inoculum potential can be made using a standardized protocol for disease assessment at the end of the growing season. This assessment has the potential to ensure that an unbiased estimate of the mean leaf area infected is calculated (Jones and Windels 1991). Such a disease assessment protocol was developed for saskatoon (Holtslag 2003) and forms the basis for assessment of late-season inoculum presence. Inoculum potential and production is the first module in the DPI equation (Fig. 1, module A). The regression equation originally developed to estimate *E. mespili* inoculum production on infected saskatoon leaves (Holtslag et al. 2003b) was multiplied by a constant of 0.1 to reduce its value. This modified regression equation represented a measure of inoculum potential and was used to calculate the DPI for the first precipitation event that occurred after the start of the infection period. However, to account for the possibility of inoculum buildup in the model after the first rain event and a decrease in inoculum after each fungicide application, a feedback loop mechanism was incorporated to estimate fluctuations in inoculum production in relation to disease pressure. This was accomplished by multiplying the inoculum potential value by the cumulative DPI value for all subsequent inoculum release events. For example, if the cumulative DPI value increased, then the feedback loop would reflect the corresponding increase in orchard inoculum potential.

Inoculum release. Based on data in Holtslag et al. (2003b), the start of a precipitation event was considered as the signal for inoculum release. The numerical value for inoculum release was changed from 0 (no release) to 1 (release) at the start of each precipitation event and therefore permitted the calculation of DPI to result in a positive value (Fig. 1, module B). When the leaf-wetness value that followed the precipitation event fell to 0, the inoculum release value in the model also returned to 0 and stopped the calculation of DPI. The DPI calculated after each rain event was summed to produce the cumulative DPI value.

Leaf-wetness duration and temperature required for disease development. The regression equation developed by Holtslag et al. (2003a), which estimates lesion development on saskatoon leaves due to entomosporium leaf and berry spot, based on leaf-wetness duration and mean temperature, was incorporated into the model (Fig. 1, module C). The original equation was multiplied by a constant of 0.25 to reduce its absolute value.

Host susceptibility. To incorporate the observation that young saskatoon leaves tend to be more susceptible to *E. mespili* infection than older leaves (Holtslag et al. 2003a; Ronald et al. 2001), an assumption was made of a negative linear relationship between host susceptibility and physiological age. Thus, equation 2 was created to estimate host susceptibility based on physiological development (see Holtslag (2003) for more detail):

$$[2] \quad Y = -0.0091X + 5$$

where Y is an estimate of host susceptibility and X represents a measure of physiological development as calculated by the sum of SASK-day heat units from the date of budbreak. The equation was calculated from two data points and was based on the assumption that a value of 5 indicates that the plant is highly susceptible to *E. mespili* infection at the time of budbreak, whereas a value of 1 would indicate that the plant is close to cessation of axillary vegetative shoot expansion and therefore much less susceptible to infection. According to the phenological model, the cessation of shoot expansion occurs when the sum of SASK-day heat units reaches 441.4. When shoot expansion ceases, the value for host susceptibility remains at a constant value of 1. The value for host susceptibility is used as the final module in the DPI equation (Fig. 1, module D).

Termination of the calculation of disease pressure index.

The model accumulates DPI values on a real-time basis after each precipitation event; however, once calendar day 243 is reached, the model is stopped (Fig. 1). This point in time is used as an estimate of when the plant has begun to acclimate for winter (Steeves and Steeves 1990). At this time, the model prompts the user to make an assessment of mean percent leaf area infected (PLAI) in the orchard. This value is then used as an estimate of inoculum potential, which is used to start the calculation of the DPI for the next growing season.

Assessing the accuracy of the DPI equation

Data analysis

The accuracy of the DPI equation was evaluated between the date of budbreak and fruit harvest. Calendar day 190 was selected to represent the start of fruit harvest, since this was the median date for the range of fruit harvest periods observed between 1999 and 2002 at the two orchards used in this study. The cumulative DPI values for each site and year, combined years at each site, and the combined sites and years were compared with the observed mean number of lesions per leaf (LESNO) during the preharvest period, using Pearson's correlation and coefficient of determination analysis. Before it was possible to make these comparisons, it was necessary to compensate for the lag period between the increase of DPI after each precipitation event and the actual development of lesions. This period, more commonly known as a latent period, is 5 d (Holtslag et al. 2003b). Each LESNO measurement was therefore moved back in time 5 d and compared with the cumulative DPI value for that date. Ultimately, combined site and year data were used to create a regression equation that estimated the LESNO for a given cumulative DPI value. A visual comparison of the predicted DPI curve relative to the observed LESNO curve over time was also made for each location and year.

Accuracy of the DPI equation

The DPI equation produced cumulative values that were highly correlated with observed LESNO for each site and year, combined years for each site, and combined sites and years (Table 1). The visual comparison of predicted DPI and observed LESNO curves over time showed that the model was generally effective for predicting disease increase prior to fruit harvest at the Winnipeg and Carman or-

chards (Figs. 2 and 3, respectively) between the years 1999 and 2002. However, it should be noted that the DPI curve more accurately predicted LESNO increase at Winnipeg than in Carman for the years it was tested. Moreover, the variation in R^2 values between years was lower at the Winnipeg site (standard deviation, $\pm 7\%$) compared with the Carman site ($\pm 14\%$).

When all the data from the sites and years were combined, 82% of the variability for observed disease development could be explained using the DPI equation. Once it was determined that the DPI equation was reliable for predicting disease development, it was possible to create a regression equation that predicted mean LESNO in a Saskatoon orchard (Fig. 4). The cumulative DPI together with the equation in Fig. 4 allowed us to provide an estimate of the mean LESNO 5 d in advance of actual symptom development. This equation can be used to determine the need for a systemic fungicide like propiconazole, which effectively suppresses mycelial growth and conidiospore germination, provided it is applied in a timely manner after an infection event (Lange et al. 1998).

Incorporation of control mechanisms into the disease-forecasting model

Control thresholds, together with application and control guidelines for propiconazole, were incorporated into the dynamic disease-forecasting model (Fig. 1) to create a more comprehensive model that included disease control capabilities (Fig. 5). In the expanded version of the model, once the cumulative DPI produced a positive integer, an assessment could be made to ascertain that a predetermined control threshold has been exceeded. Once the first control threshold is exceeded, then it is necessary to determine if a spray can be made according to propiconazole application guidelines.

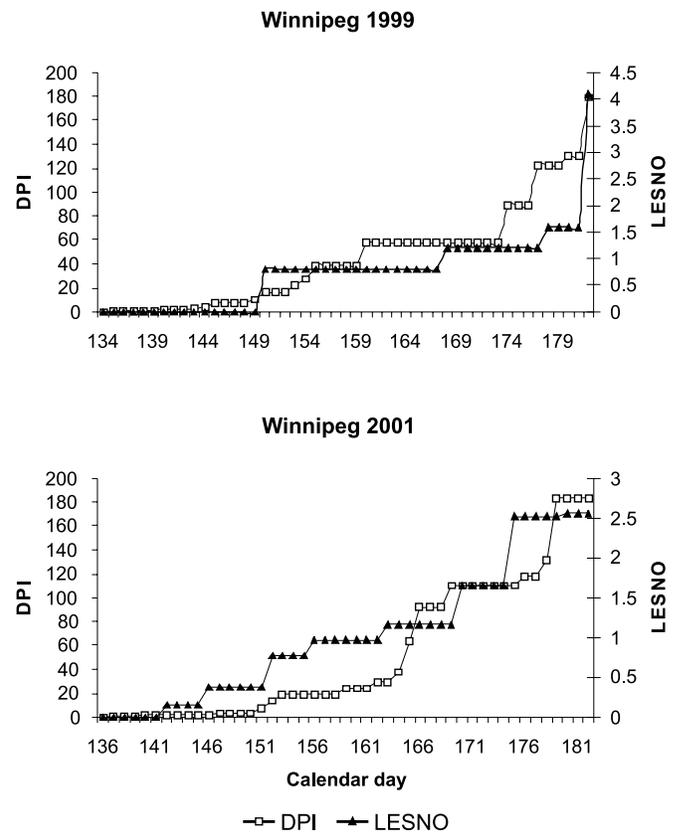
The application guidelines for propiconazole indicate that no spray application is allowed within 38 d of harvest. Therefore, if the phenological modeling system described above estimates that there are 38 d or less until the date of fruit harvest, then no spray recommendation will be made, despite the possibility of high disease pressure (Fig. 5). If there are more than 38 d until the predicted date of fruit harvest, or if harvest has already occurred, then the model will consider whether propiconazole was applied previously. Provided no fungicide was previously applied, the model will suggest an application of propiconazole. However, if propiconazole was previously applied, then the model will determine if the fungicide is still active. While the axillary vegetative shoots are still expanding (SASK-day heat units < 441.4), the model will assume that the fungicide remains active for a period of 14 d. This assumption is based on the premise that the fungicide concentration becomes diluted within a plant that is actively growing. However, if it is the 39th day before fruit harvest, and if the second control threshold is exceeded, the model will recommend another application of propiconazole despite the fact that less than 14 d may have elapsed since the last spray. Once the axillary vegetative shoots have ceased to expand (SASK-day heat units ≥ 441.4), spray applications will be able to control *E. mespili* for a period of 21 d. After this point, if the model determines that the last application of

Table 1. Correlation coefficients (r) and coefficients of determination (R^2) between disease pressure index values for entomosporium leaf and berry spot and the observed mean number of lesions per leaf on Saskatoon during the preharvest period in Manitoba, from year 1999 to 2002.

Site	Year(s)	r	R^2
Winnipeg	1999	0.98	0.97
Winnipeg	2001	0.93	0.87
Winnipeg	1999 and 2001	0.96	0.93
Carman	1999	0.96	0.91
Carman	2001	0.86	0.74
Carman	2002	0.80	0.64
Carman	1999, 2001, and 2002	0.91	0.84
All sites and years combined		0.9	0.82

Note: All coefficients were significant at $P = 0.01$.

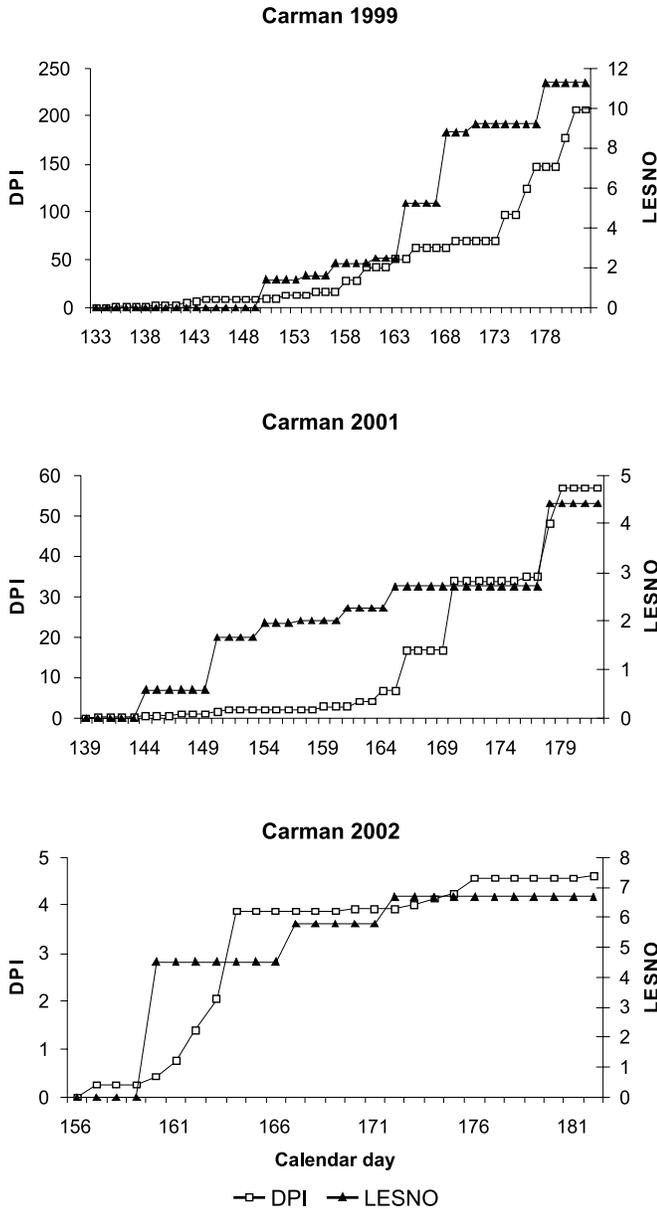
Fig. 2. Predicted increase in disease pressure index (DPI) of entomosporium leaf and berry spot relative to the observed mean lesion number per leaf (LESNO) over time at the Winnipeg Saskatoon orchard, Manitoba, for the years 1999 and 2001.



propiconazole is no longer active, then it will consider whether the second control threshold has been exceeded. If the second control threshold is exceeded, the model will recommend another spray of propiconazole.

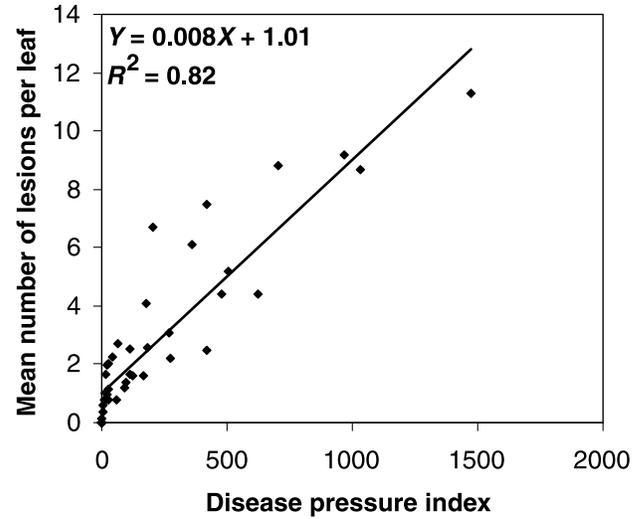
Each time propiconazole is applied, the cumulative DPI value is reset to 0.1 (Fig. 6). The cumulative DPI value cannot be reset to zero after propiconazole is applied because

Fig. 3. Predicted increase in disease pressure index (DPI) of entomosporium leaf and berry spot relative to the observed mean lesion number per leaf (LESNO) over time at the Carman saskatoon orchard, Manitoba, for the years 1999, 2001, and 2002.



of the feedback loop in the model, which connects it with the inoculum potential value. This more accurately reflects the phenomenon whereby not all the pests are eliminated from a field after pesticide application. Moreover, if the cumulative DPI were reset to zero, then the inoculum potential value in the model would also be reduced to zero. Reducing the inoculum potential value in the equation to zero would cause the cumulative DPI value to remain at zero for the remainder of the season. Disease pressure index values continue to be summed regardless of whether propiconazole was applied or not. After each spray application, the number of days is counted in the model to keep track of the fungicide activity period.

Fig. 4. Relationship between mean number of lesions per leaf (LESNO) on saskatoon and disease pressure index (DPI) of entomosporium leaf and berry spot, based on pooled data from the Winnipeg (1999 and 2000) and Carman (1999, 2001, 2002) sites, Manitoba. A regression line was fitted to the data where Y is the predicted LESNO and X is the cumulative DPI value.

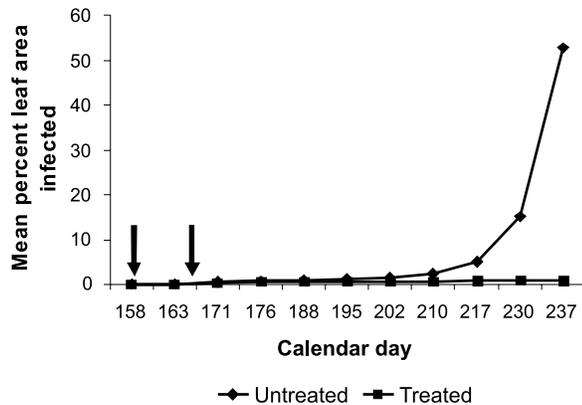


Field testing of the model

During the summer of 2002, a preliminary field test was conducted at the Carman orchard to evaluate whether the model was effective in predicting and controlling entomosporium leaf and berry spot. For this test, the orchard was divided into five sections: three sections received no fungicide applications and two received fungicide treatments as specified by the model. Because of the history of severe disease in the orchard, the modified inoculum production regression equation calculated the inoculum potential value to be 0.22 (maximum level). The control thresholds were conservatively set at 0 and 0.1 for the first and second control threshold, respectively. This was done in an attempt to minimize disease development as much as possible. Propiconazole was applied with a backpack sprayer following the manufacturer’s application guidelines (Engage Agro Corporation, Guelph, Ont.). An assessment of disease severity was made weekly between calendar day 158 and 237 by measuring the mean PLAI on five leaf samples from each of five randomly selected plants in each orchard section. The mean PLAI was determined by importing the pictures into the software program Assess for Windows (American Phytopathological Society Press, Saint Paul, Minn.).

Actual and historical SASK-day heat unit data were used to predict the dates of flowering and fruit harvest from the date of budbreak, which occurred on calendar day 116 in 2002. This system successfully predicted the date of flowering 25 d after the date of budbreak, which was 9 d in advance of the actual flowering date (calendar day 150), and the date of fruit harvest 25 d after budbreak, which was 57 d in advance of the actual fruit harvest date (calendar day 204). The first positive DPI value (0.26), as determined by the model, was calculated on calendar day 157. Therefore, propiconazole was applied for the first time on calen-

Fig 6. Increase in mean percent leaf area infected (PLAI) by entomosporium leaf and berry spot over time for fungicide-treated and untreated sections at the Carman saskatoon orchard, Manitoba, in 2002. The treated section of the orchard received two propiconazole applications (arrows) as predicted by the dynamic disease-forecasting model. Percent leaf area infected after calendar day 204 (start of fruit harvest) was significantly greater in the untreated section of the orchard and exceeded 50% by the end of the season. Conversely, in the treated section, PLAI did not exceed 1% at any point between calendar day 158 and 237.



dar day 158. The spray was permitted because there were 46 d until the predicted fruit harvest date, which was calendar day 204. A second propiconazole application was applied 7 d later on calendar day 165, because the cumulative DPI had risen to 3.73, exceeding the 0.1 control threshold on the 39th day before the predicted date of fruit harvest.

The mean PLAI in the treated sections of the orchard was limited to 0.5% at the start of fruit harvest and was kept below 0.6% until fruit harvest was completed (Fig. 6). Percent leaf area infected in the untreated sections of the orchard averaged 1.3% by the start of fruit harvest and reached 5.0% by the end of fruit harvest. According to paired *t*-test analysis, mean PLAI was significantly higher ($P = 0.05$) in untreated sections compared with the treated sections of the orchard by the time fruit harvest began, and remained so until after harvest was completed. Moreover, additional benefits of disease reduction were realized after fruit harvest when disease levels began to increase exponentially in the untreated sections of the orchard, resulting in a mean PLAI of over 50% by calendar day 237. The mean PLAI in the treated sections of the orchard remained below 1% throughout the entire sampling period of this study.

Discussion

Given actual and historical SASK-day heat unit data, it was possible to predict the dates of flowering and fruit harvest 9 and 57 d, respectively, in advance of actual occurrence in 2002. In another study, Friesen (1986) showed that the growing degree day model was able to predict the date of saskatoon fruit harvest within ± 1 d when predicting began 40 d after the date of flowering. Although the predictive ability of the model in the present study was not specifically tested using regression analysis, the SASK-day model more reliably identified the date of fruit harvest earlier in

the season than Friesen's (1986) model. The heat units required for the cessation of axillary vegetative shoot growth were used to create a negative linear model to estimate how the susceptibility of saskatoon leaves decreased relative to increasing physiological time. A future study is required to verify that host susceptibility actually does decrease in a linear fashion relative to the accumulation of heat units.

The difference in disease-forecast model accuracy at the Carman and Winnipeg orchards may be explained by the difference in plant health at each site. The saskatoon plants in the Carman orchard were notably more stressed than those at the Winnipeg orchard. Plant stress in the Carman orchard was induced by a combination of poor drainage, limited soil nutrients, and a *Cytospora* sp. epidemic, which were not considered in the disease-forecasting model. The relatively low R^2 value at the Carman 2002 site may have been the result of the short preharvest period, which was a result of a very late spring. The late spring in 2002 seemed to accelerate disease severity early in the growing season and did not permit many disease measurements prior to harvest because of accelerated plant growth.

Comparing the R^2 values for the DPI equation against other disease modeling studies provided an assessment of model accuracy. In a controlled environment study, Grove (2002) explained the majority of variation in disease severity on cherry ($R^2 = 0.80$) and peach ($R^2 = 0.83$) foliage, using models based on leaf-wetness duration and temperature. A degree-hour model described 80% of the observed infection variability in *Puccinia graminis* subsp. *graminicola* Z. Urb. on perennial rye grass (*Lolium perenne* L.) seed crops (Pfender 2003). Both studies concluded that the coefficients of determination for each model were acceptable for describing disease development. Therefore, it seems appropriate to conclude that the DPI equation developed in this study is sufficient for predicting the development of entomosporium leaf and berry spot on saskatoon.

In a preliminary test, entomosporium leaf and berry spot at the Carman 2002 site was effectively controlled using the dynamic disease-forecasting model. The model was able to successfully predict disease symptom development 5 d in advance of actual occurrence. Only two applications of propiconazole were suggested by the disease-forecasting model compared with three applications that are currently recommended based on plant development stages (Lange et al. 1998; St-Pierre 1997). Even though the coefficients of determination between DPI and LESNO accounted for only 64% of the variation in disease levels in the untreated sections at the Carman 2002 site, the disease-forecasting model effectively controlled entomosporium leaf and berry spot on the saskatoon plants prior to and after fruit harvest. Future research is required to validate the model in different years and locations. It may also be possible to reduce the number of sprays to below two and still achieve good disease control. However, to ascertain that the number of sprays can be reduced, further research is required to determine suitable economic thresholds.

The fact that early season applications of propiconazole controlled disease symptom development throughout the season, despite the multiple precipitation events that occurred before completion of fruit harvest, indicates that the severity of entomosporium leaf and berry spot is linked to

the availability of early season inoculum. Eleven separate precipitation events occurred between the first and second propiconazole applications, and 12 more occurred between the second application and the completion of fruit harvest. The amount of early season inoculum is also a major limiting factor influencing powdery mildew [*Erysiphe polygoni* DC.] epidemics on sugar beet (Asher and Williams 1991) and apple scab [*Venturia inaequalis* (Cooke) Wint.] (Boone 1971). This emphasizes the importance of including an inoculum potential value in a disease-forecasting model.

A reduction in disease development prior to and during fruit harvest may help to ensure a high yielding and high quality saskatoon crop. Johnson (1988) and Madden et al. (1981) used models to quantitatively describe the effects of disease on yield loss and generally found that as disease severity increased, so did the loss in yield. However, to verify this relationship in saskatoon orchards, a future study is necessary to assess the relationship between infected leaf and fruit area. A prerequisite for such a study is to determine the timing of rain-splashed inoculum dispersal and the period of fruit susceptibility. For example, despite prolific leaf infection early in the season, little disease may develop on saskatoon fruit if no inoculum is released during the fruit maturation period.

A reduction in disease levels after fruit harvest will improve saskatoon plant vigor and, in doing so, will have the potential to increase harvestable yields and limit the development of entomosporium leaf and berry spot in subsequent years. This is primarily because less infected leaf litter will overwinter. The lower disease pressure should also limit the number of fruit and young shoot infections, which can also be a source of initial inoculum in the following season.

In conclusion, the dynamic disease-forecasting model described in this paper has modified current fungicide application practices on saskatoons. Once thoroughly tested and implemented in a production system, this model should provide a mechanism to minimize fungicide use. Fewer fungicide applications will reduce producer costs, decrease environmental damage, and slow the development of fungicide-resistant strains of the pathogen. Similar to other models (Remphrey and Prusinkiewicz 1997), the development of the dynamic disease-forecast model for saskatoons has identified important gaps in our scientific knowledge and has pointed out avenues for further research.

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