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Spatial heterogeneity, incidence-incidence and incidence-lesion density relationship of apple scab (*Venturia inaequalis*) in managed orchards

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Abstract The spatial pattern of apple scab was characterized using 10 years of disease incidence and lesion density data collected in managed orchards located in Quebec, Canada. Distributional analyses indicated that scab incidence was better characterized by the beta-binomial than the binomial distribution in 53 and 65% of the data sets at the leaf and shoot scales, respectively. Median values of the beta-binomial parameter θ, a measure of small-scale aggregation, were near 0 (0.003 and 0.028) at both sampling scales, indicating that disease incidence was close to being randomly distributed (low degree of aggregation). For lesion density, the negative binomial distribution fitted the data better than the Poisson distribution in 86% of the data sets at the leaf scale. The median value of the index of dispersion k was 0.068, indicating that aggregation was present. For all apple scab measurements, the power law models provided a good fit to the data. The estimated slope and intercept parameters were significantly greater than 1 and 0, respectively, suggesting that spatial heterogeneity changed systematically with disease incidence. Results of a covariance analysis showed that spatial heterogeneity of scab incidence at both scales and lesion density was not dependent upon shoot type but that spatial heterogeneity of scab incidence and lesion density at the leaf scale was influenced by the sampling period. A hierarchical analysis showed that scab incidence at the tree scale increased as a saturation-type curve with respect to incidence at the leaf or shoot scales. A similar relationship was observed for incidences at the shoot and leaf scales. An effective sample size model based on the binary power law parameters (Madden and Hughes, Phytopathology 89:770–781, 1999) gave the best fit to the leaf and shoot data, respectively. The incidence-lesion density relationship at both scales was well described by a complementary log-log (CLL) and log transformation model $\left(R^2_{adj} = 0.97 \; and \; R^2_{adj} = 0.94\right)$, however, the models tended to underestimate lesion density. The information of the spatial relations of apple scab within and between hierarchical scales acquired from this study can be used in developing and evaluating practical disease management strategies and to improve apple scab assessments for fungicide or cultivar susceptibility trials.

Keywords Binary power law · Distribution · Incidence-incidence relationship · Quantitative epidemiology

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Introduction

Apple scab, caused by the fungus *Venturia inaequalis* (Cke.) Wint., is the key disease for scheduling fungicide sprays in many apple-growing areas throughout the world (MacHardy 1996). Epidemics of apple scab are caused by two spore types (ascospores and conidia) and comprise both a monocyclic and polycyclic phase. In eastern North America, most current strategies for managing apple scab are based on controlling primary infections in order to avoid epidemic build-up caused by secondary infections and, thus, reduce or eliminate the need for subsequent fungicide applications during the summer months. To achieve this goal, fungicide applications generally begin in the spring at the green tip phenological stage and continue throughout spring in to early summer, resulting in approximately 8–10 fungicide applications (Reardon et al. 2005). If this approach fails to control scab adequately, then fungicides must be applied during the summer and the annual number of fungicide sprays could rise to 14–16, depending on the prevailing weather conditions during the summer months (MacHardy 1996; Reardon et al. 2005). It is therefore important to develop tools to precisely estimate the level of control of primary infections.

Accurate assessment of disease intensity is an essential component of disease management. When the ability to assess disease accurately exists, management actions, such as the timing of fungicide applications, can be tailored to some known action or economic threshold. Disease intensity can be assessed as either incidence (defined as the proportion of plant units diseased), severity (defined as the area of plant tissue diseased), or lesion density (defined as the number of lesions per plant unit). Various parameters have been proposed to study apple scab epidemics, cultivar susceptibility, and fungicide efficacy including disease incidence, the number of lesions or conidia per shoot, the number of conidia per lesion, and the number of lesions or conidia per unit area of leaf tissue (Dewdney et al. 2003; Jeger 1981). In these studies both incidence and severity were considered as good estimators of apple scab epidemics. However, incidence is generally easier and faster to measure than severity or lesion density. For apple scab, lesion density is thought to be better related to yield loss and to provide a better estimation of disease progress (Jeger 1981), but the exact number of lesions is often difficult to count because of coalescence or of the presence of atypical lesions (Holb et al. 2005).

It is possible to define the relationship between disease incidence and lesion density such that measurements of disease incidence can be used to predict lesion density (Jeger 1981; Seem 1984). These types of relationships often rely on measuring disease on a plant unit or at a scale higher than that of direct interest to predict disease at the unit or scale of interest. For example, measuring the incidence of diseased shoots may be used to predict the incidence of disease on individual leaves (Hughes et al. 1997; Turechek and Madden 2003); or, measuring the incidence of diseased leaves may provide sufficient information for predicting lesion density on these same leaves (McRoberts et al. 2003). Jeger (1981) showed a significant relationship between scab incidence (angularly transformed proportion of scabbed leaves) and severity (log of number of lesions per leaf) in unsprayed orchards planted with mixed cultivars and with the cultivar Cox's Orange Pippin (Jeger 1983). Whether these same relationships apply to managed systems or to Canadian scab epidemics where fungicide resistant isolates are widely prevalent is unknown (Carisse and Jobin 2010; Jobin and Carisse 2007).

In order to design efficient sampling strategies and to define incidence/incidence or incidence/lesion density relationships, knowledge of the spatial heterogeneity of disease is needed (Madden and Hughes 1995). Although apple scab has been intensively studied (MacHardy 1996), little is known about its spatial distribution within an orchard. In a recent study, Charest et al. (2002) showed that the primary inoculum is not uniformly distributed within an orchard. Furthermore, the same authors showed that airborne ascospore concentrations are aggregated within an orchard, and Carisse et al. (2006) showed that ascospore deposition within an apple tree was aggregated during the peak of ascospore release. From these results, one may assume that apple scab incidence and lesion density are spatially aggregated in orchards; however, the robustness of this assumption must be evaluated. Recently, Carisse et al. (2009) reported that the number of scabbed leaves per shoot mostly followed a beta-binomial distribution.

A variety of methods are available for quantifying spatial heterogeneity of plant disease (Gosme 2008; Hughes and Madden 1993; Madden et al. 2007). A common method is to fit distribution models to

frequency data (incidence or severity) and determine the level of spatial heterogeneity from estimated parameters (Madden and Hughes 1995). Similarly, indices of aggregation such as the index of dispersion (*D*), for incidence data, and the variance-to-mean ratio (V/M), for lesion count data, can be calculated based on mean and variance relationships (Gosme 2008; Madden and Hughes 1995; Madden et al. 1995; Madden et al. 2007). Likewise, Taylor's power law and the binary power law can be used to describe the relationship between the observed variance of lesion intensity and the theoretical variance of lesion intensity under the assumption that disease is distributed randomly for count and incidence data, respectively (Madden et al. 1995; Taylor 1961; Turechek and Madden 1999). The power law can be used for the identification of factors that affect the spatial heterogeneity of disease incidence such as shoot type or period of sampling (Turechek and Mahaffee 2004).

This study is part of a larger study on apple scab epidemiology under commercial conditions (managed orchards) aiming at developing new management tools. The objectives of this study were: (i) to study the spatial heterogeneity of apple scab incidence and lesion density at different assessment scales; and (ii) to establish the relationships between incidences across scales and between incidence and lesion density at the same scale and across scales with the goal of finding a simple relationship for predicting disease intensity using a measurement of disease incidence.

Materials and methods

Orchard sites and disease assessment

The data were collected in 13 commercial apple orchards located in the southwest and southeast regions of the province of Quebec, Canada. The orchards were selected based on their representativeness of the apple production in the province of Quebec. Because these were commercial orchards, diseases (including apple scab) and insects were managed according to standard grower practices, which varied among orchards and over years. The data were collected at the end of the primary infection period in late spring (mid-June), in the summer (mid-July), and during the pre-harvest period in early fall (early September) in each year from 1998–2007. The characteristics of each orchard are described in Table 1.

Disease assessments were performed on 60–120 trees per orchard depending on the orchard. Sampling was done following a 'W' pattern. Assessments of terminal and cluster shoots were performed on 5 or 10 shoots per tree located at the bottom, at the top and in the

Table 1 Description of the apple orchards in Quebec, Canada, surveyed for apple scab caused by *Venturia inaequalis*

Site		Number of data sets	Varieties	Rootstock	Spacing (m)	
Orchard	County				Within rows	Between rows
1	Rougemont	15	McIntosh, Lobo	M.7	6.0	4.1
2	Franklin	4	McIntosh	MM.106	3.9	1.6
3	Frelighsburg	11	McIntosh, Cortland	Imperial	6.7	4.3
4	Frelighsburg	2	McIntosh, Lobo	M.7	5.8	3.8
5	Frelighsburg	4	McIntosh	M.7	6.1	3.9
6	Dunham	1	McIntosh	MM.106	5.0	3.0
7	Frelighsburg	12	McIntosh, Lobo	M.26	4.5	2.5
8	Rockburn	3	McIntosh	M.7	6.1	3.1
9	Dunham	2	McIntosh	MM.106	4.8	3.0
10	Rougemont	14	McIntosh, Lobo	M.7	6.1	3.8
11	St-Césaire	15	McIntosh	MM.106	4.9	3.2
12	St-Paul	1	McIntosh, Lobo	M.7	6.0	4.0
13	Dunham	11	Lobo	MM.106	4.7	2.0

centre of the tree and all leaves on each shoot were examined for the number of lesions per leaf. The lesion density data was converted to disease incidence data by rating a leaf as diseased if it had one or more lesions, and rating a shoot diseased if it had one or more diseased leaves on it. At the leaf level, this represents a cluster sample where N clusters (trees) of n leaves were sampled for the incidence of scab. At the shoot level this represents a cluster sample where N clusters of m shoots were rated for the incidence of scab.

Scab incidence was determined at each assessment scale. For each data set (orchard site-sampling date), the incidence of scabbed leaves per tree was calculated as, $\hat{p}_f = 1/N \sum_N (x_f/n)$where x_f is the number of scabbed leaves per tree, n is the number of leaves sampled per tree, and N is the number of trees sampled. The incidence of scabbed shoots per tree was calculated as $\hat{p}_t = 1/N \sum_N (x_t/m)$ where x_t is the number of scabbed shoots per tree, and m is the number of shoots sampled per tree. Lesion density was defined as the mean number of lesions per tree and was calculated as $\overline{y}_f = \sum_N y_f/N$, where y_f is the number of scab lesions per tree. Mean number of lesions per shoot was calculated as $\overline{y}_t = \sum_N y_t/N$, where y_t is the number of scab lesions per shoot.

Distribution analysis

Incidence Spatial heterogeneity in scab incidence was evaluated by fitting the binomial and beta-binomial discrete frequency distributions to the observed number of diseased leaves (x_f) and the observed number of diseased shoots (x_t) per tree for each orchard assessment using the SAS macro betabin-v22 (Qi Statistics, www.qistatistics.co.uk). The binomial distribution has a single parameter, π, representing the probability of disease, while the beta-binomial has two parameters, p, which is the expected probability of disease, and θ, a measure of the variation (heterogeneity) in disease incidence among sampling units above what would be expected from a good fit to the binomial distribution. A good fit to the binomial distribution is an indication of a random spatial pattern of disease incidence while a good fit to the beta-binomial is indicative of an aggregated pattern. The parameter θ is an index of aggregation such that the degree of aggregation increases with increasing values of θ, and when $\theta=0$ the beta binomial distribution reduces to the binomial distribution indicating a random of pattern of disease. A log-likelihood ratio test statistic was calculated to test whether the beta-binomial distribution fitted the data better than the binomial distribution.

Lesion density Heterogeneity in lesion density was evaluated by fitting the Poisson and negative binomial discrete frequency distributions to the observed number of lesions per tree (y_f) for each orchard assessment (Madden et al. 2007; Xu and Madden 2002). Chi-square goodness-of-fit tests were calculated for each distribution after lesion density classes (i.e., 0, 1, 2, 3, etc. lesions per tree) were pooled so expected frequencies in each class exceeded five. The Poisson distribution has a single parameter, μ, representing mean lesion density and is appropriate when the lesion density is randomly distributed. The negative binomial has two parameters, μ, representing mean lesion density, and k, a measure of the variation (heterogeneity) in lesion density among sampling units. The negative binomial parameter k is used as an index of dispersion: as k approaches 0, the degree of aggregation increases. For very large values of k, the negative binomial distribution is practically indistinguishable from the Poisson distribution. The distribution analysis was performed using the GENMOD procedure of SAS (SAS Inc., Cary, NC, Version 9.1).

Indices of aggregation

Incidence The index of dispersion, D, was calculated to estimate the degree of aggregation as the ratio of the observed variance of x_f or x_t, $v_{obs,f}$ or $v_{obs,t}$, respectively, to the variance expected under a random distribution of x_f or x_t, $v_{ran,f}$ or $v_{ran,t}$, respectively, where $v_{ran,f} = n\hat{p}_f(1-\hat{p}_f)$ and $v_{ran,t} = m\hat{p}_t(1-\hat{p}_t)$ for leaf and shoot scale assessments, respectively. When the distribution of disease incidence is random $D=1$; when disease incidence is aggregated the observed variance is greater than the expected binomial variance making $D>1$. Under the null hypothesis of a random distribution, $(N-1)\times D$ follows a χ^2 distribution with $(N-1)$ degrees of freedom.

Lesion density The variance-to-mean ratio (V/M) was calculated as an index of aggregation. This index was calculated by dividing the sample variance by the

sample mean. When the ratio is <1, = 1, or >1, the pattern of distribution is considered regular, random, or aggregated, respectively (Madden et al. 2007). If there is a random distribution, $(N-1)\times$ V/M follows a χ^2 distribution with $(N-1)$ degrees of freedom.

Power law relationships

Power law analyses were performed on both the incidence and lesion density data. For incidence data, the binary form of Taylor's power law was fitted separately to the data at the leaf and shoot scales according to the following equation:

$$\ln(v_{obs,\bullet}) = \ln(A_{x,\bullet}) + b_{x,\bullet}\ \ln(v_{ran,\bullet}) \quad (1)$$

where $v_{obs,\bullet}$ and $v_{ran,\bullet}$ are the observed and expected variances (i.e., $v_{ran} = np(1-p)$) of number of scabbed leaves or shoots per tree, respectively, the dot subscript represents either f or t depending on the scale of analysis and $\ln(A_{x,\bullet})$ and $b_{x,\bullet}$ are the intercept and slope parameters, respectively, for assessments made at the leaf or shoot scales and were estimated via linear regression. Significance of estimated parameters was determined with t-tests and goodness-of-fit was determined with the coefficient of determination (R^2). When $A_{x,\bullet}=1$ and $b_{x,\bullet}=1$, diseased leaves (or shoots) have a random spatial pattern that can be described by the binomial distribution. When $b_{x,\bullet}=1$ and $A_{x,\bullet}>1$, diseased leaves (or shoots) have an aggregated distribution but the degree of aggregation does not depend on $p_{\bullet}$. When $b_{x,\bullet}$ and $A_{x,\bullet}$ are both greater than 1 the degree of aggregation or heterogeneity changes systematically with $p_{\bullet}$.

For the lesion density data, Taylor's power law was used to describe the relationship between the mean $(\bar{y}_f)$ and variance (s_f^2) of scab lesions per tree (Taylor 1961):

$$\ln(s_f^2) = \ln(A_y) + b_y \ln(\bar{y}_f) \quad (2)$$

where $\ln(A_y)$ and b_y are the intercept and the slope of the regression line, respectively.

Significance of estimated parameters was determined with a t-test and goodness of fit was determined with the coefficient of determination (R^2).

Covariance analyses A covariance analysis was conducted to quantify the effect of the factors 'sampling period' (spring, summer, fall), and 'shoot type' (cluster, terminal) on the slope ($b_{x,\bullet}$ or b_y) and intercept ($\ln(A_{x,\bullet})$ or $\ln(A_y)$) of the power law (Eqs. 1 and 2), where the dot subscript represents either f or t depending on the scale of analysis. The aim of this analysis was to determine if the factors affected the degree of heterogeneity after correcting for the relationship between the observed variance and $v_{ran,\bullet}$ or $\bar{y}$. The regression and covariance analysis were performed by considering the power law model (Eqs. 1 or 2) as the null model and factors were added individually, first as an intercept term, then as an interaction term with the slope. A factor was considered significant if the new model had a deviance significantly lower than the original power law model (Turechek and Madden 1999; Turechek and Mahaffee 2004). The covariance analysis was performed using the GLM procedure of SAS (SAS Inc., Cary, NC, Version 9.1).

Relationships between measurements of disease intensity

Incidence-incidence relationships (sampling units) The zero term [Pr(0)] and mean (p_f or p_t) of the probability distribution of diseased leaves (or shoots) was used to estimate the proportion of diseased sampling units (trees [p_{su}]). A complete description of the methodology can be found in Turechek and Madden (2003) and Turechek and Mahaffee (2004) and only the forms of the models are presented here for brevity. In addition, we will refer only to the relationship for assessments made at the leaf level for simplicity in presentation; derivation at the shoot-level assessments follows similarly.

If the incidence of scabbed leaves can be described by a binomial distribution then

$$\tilde{p}_{su} = 1 - (1 - \hat{p}_f)^n \quad (3)$$

A tilde (~) is used to denote that the estimate of p_{su} was derived from measurements at a lower spatial

scale. If incidence of diseased leaves can be described by the beta-binomial distribution then

$$\widetilde{p}_{su} = 1 - \prod_{j=0}^{n-1} \frac{1 - \hat{p}_f + j\hat{\theta}_f}{1 + j\hat{\theta}_f}, \tag{4}$$

where $\hat{\theta}_f = \left[a - f(p_f)/n\right] / \left[f(p_f) - a\right]$, and $f(p_f) = \left[p_f(1-p_f)\right]^{1-b}$ and $a = A_{x\cdot f} n^{b-2}$ (Turechek and Madden 1999). Equation 4 is difficult to rearrange to allow prediction of p_f from p_{su}. Following the approach of Rao and Scott (1992), however, Eq. 3 can be modified to correct for the effect of heterogeneity by replacing n in Eq. 3 with an "effective sample size". Two effective sample size models were considered here. The first model is based on the parameters of the power law and assumes D varies systematically with p_f:

$$n_\delta = f(\hat{p}_f)/a \tag{5}$$

where $f(\hat{p}_f)$and a are defined above. The second model uses the effective sample size of Madden and Hughes (1999):

$$n_\nu = n/(0.98135 + 0.8179\,\theta + 0.004958n + 0.30387\,\theta n - 0.3471\,\theta^2 - 0.08475\,\theta^2 n) \tag{6}$$

where θ is the heterogeneity parameter of the beta binomial distribution and was determined using the equation immediately below Eq. 4. To determine which of these effective sample size models best fitted the data, Eq. 3 was rearranged as:

$$\mathrm{CLL}(\widetilde{p}_{su}) = \ln(n_\bullet) + \mathrm{CLL}(\hat{p}_f) \tag{7}$$

where $\mathrm{CLL}(z) = \ln[-\ln(1-z)]$ is the complementary log-log transformation of z (Farrington 1992; Turechek and Madden 2003). This is an equation for a straight line with a slope of 1 and intercept of $\ln(n_\bullet)$, where $n_\bullet$ is replaced by either n, n_δ, or n_ν as defined above. Equation 7 can be viewed as a simple reformulation of Eq. 3 thus requiring no curve fitting to estimate the parameters, or it could be viewed as a statistical model for $\mathrm{CLL}(\widetilde{p}_{su}) : \mathrm{CLL}(\hat{p})$. For the latter, n_δ (Eq. 5) was substituted for $n_\bullet$ into Eq. 7 and, after some rearrangement:

$$\mathrm{CLL}(\widetilde{p}_{su}) = -\ln(a) + (1-b)\cdot\ln[\hat{p}_f(1-\hat{p}_f)] + \mathrm{CLL}(\hat{p}_f). \tag{8}$$

To fit this model, $\mathrm{CLL}\left(\hat{p}_f\right)$ was treated as an offset variable, $\ln\left[\hat{p}_f\left(1-\hat{p}_f\right)\right]$ was treated as an independent variable with slope $(1-b)$ and intercept $-\ln(a)$.

The deviance was calculated for five candidate models: four required no direct curve fitting or parameter estimation as the parameters were estimated beforehand; one model did. The four cases requiring no parameter estimation were: (i) binomial (random) distribution of diseased leaves (Eq. 7 with $n_\bullet = n$); (ii) beta-binomial model [Eq. 4 formulated as a CLL (see Eq. 15 in Turechek and Madden 2003)]; (iii) effective sample size assuming D varies systematically with p according to the binary power law (Eq. 7 with $n_\bullet = n_\delta$); and (iv) effective sample size of Madden and Hughes (1999) with variable heterogeneity (Eq. 7 with $n_\bullet = n_\nu$). The model requiring curve fitting, case (v), was Eq. 8 fitted as described above.

Incidence-incidence relationships (leaves-shoots) The relationship between the incidence of diseased sampling units (p_{su}) and diseased leaves (p_f), and between p_{su} and diseased shoots (p_t) were used as the foundation to model disease incidence between p_f and p_t (Turechek and Madden 2003). Beginning with Eq. 3, $\widetilde{p}_{su}$can be estimated by both $1-(1-\hat{p}_f)^n$ and $1-(1-\hat{p}_t)^m$, for leaves and shoots , respectively, and equating the two gives:

$$\hat{p}_t = 1 - (1-\hat{p}_f)^{n/m}. \tag{9}$$

The effects of heterogeneity can be accounted for by substituting any of the effective sample size models for $n(i.e., n_\delta \text{ or } n_\nu)$ and/or $m(i.e., m_\delta \text{ or } m_\nu)$, depending on the assumptions one is willing to make. As above, Eq. 9 can be rearranged to give:

$$\mathrm{CLL}(\widetilde{p}_t) = \ln(n_\bullet/m_\bullet) + \mathrm{CLL}(\hat{p}_f), \tag{10}$$

with an estimate of the intercept parameter obtained by simply substituting the effective sample sizes from data obtained at the respective scales (i.e., no curve fitting), or through curve fitting. Four possible models were examined here. For case (vi), it was assumed that the incidence of scab was distributed according to the binomial distribution at both scales, so n/m was substituted for $n_\bullet/m_\bullet$ in Eq. 10; for case (vii) it was assumed that heterogeneity could be explained according to Eq. 5 at both the leaf and shoot scales

(i.e., n_δ/m_δ was substituted for $n_\bullet/m_\bullet$ in 10); for case (viii), it was assumed that heterogeneity could be explained according to Eq. 6 at both the leaf and shoot scales (i.e., n_ι/m_ν was substituted for $n_\bullet/m_\bullet$ in Eq. 10). For case (ix), curve fitting was used to estimate the intercept parameter, $\ln(n_\bullet/m_\bullet)$ in Eq. 10 and the slope was assumed to be constant and equal to one.

For all models described, the GENMOD procedure of the statistical package SAS was used to estimate the deviance (SAS Inc., version 9.2, Cary, NC). For cases (i) to (iv) and (vi–viii), where no parameters were estimated, the method described by Aitkin et al. (page 177) (Aitkin 1989) was used to determine the deviances.

Incidence-lesion density relationships The relationship between the incidence of scabbed leaves per tree $\left(\hat{p}_f\right)$ and the number of scabbed lesions per tree $\left(\overline{y}_f\right)$, and between the number of scabbed shoots per tree $\left(\hat{p}_t\right)$ and the number of scabbed lesions per shoot $\left(\overline{y}_t\right)$ were fitted by the following models:

$$\ln(\overline{y}) = \beta_0 + \beta_1 \,\mathrm{CLL}\left(p_f\right) \tag{11a}$$

$$\ln(\overline{y}) = \beta_0 + \beta_1 \,\mathrm{CLL}(p_t) \tag{11b}$$

where β_0 and β_1 are model parameters. Several other models for incidence–severity relationships were evaluated based on previous usage in the literature (McRoberts et al. 2003; Paul et al. 2005; Xu and Madden 2002). However, these models were not subjected to further analysis because their residual plots were non-random, or the shape of the curve for predicted lesion density versus incidence diverged from that exhibited by the raw data. A one-sided *t*-test was used to determine if the slope of Eqs. 11a and 11b was larger than 1, based on the estimated β_1 and its standard error. Model fit was evaluated based on coefficient of determination adjusted for degrees of freedom $\left(R^2_{adj}\right)$, squared correlation between observed and predicted lesion density (R^{*2}) and randomness of the residual plots.

Results

The proportion of scabbed leaves $\left(\hat{p}_f\right)$ ranged from 0 to 0.27, with a median of 0.0145 scabbed leaves per tree, and 77% of the orchards having $\hat{p}_f < 0.05$ (Fig. 1a). The proportion of scabbed shoots $\left(\hat{p}_t\right)$ ranged from 0 to 0.75, with a median of 0.1049 scabbed shoots per tree, and 79% of the orchards having $\hat{p}_t < 0.45$ (Fig. 1b). The mean number of lesions per leaf $\left(\overline{y}_f\right)$ ranged from 0 to 0.92 with a

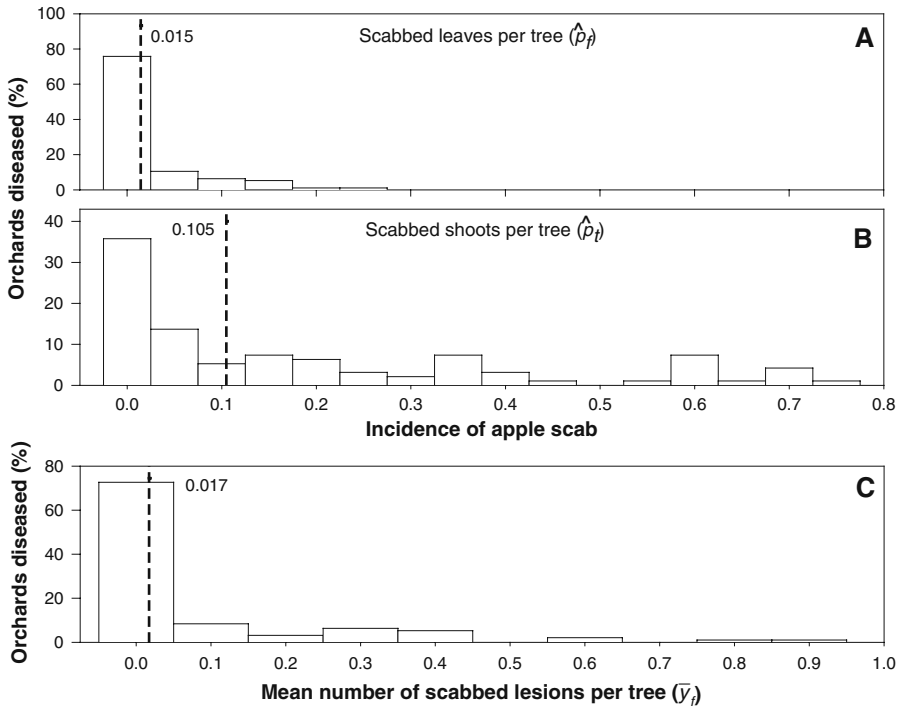

Fig. 1 Distribution of observed proportion of scabbed leaves per tree (**a**), proportion of scabbed shoots per tree (**b**), and number of lesions per leaf (**c**) for the 95 orchards located in Quebec, Canada in 1998–2997. The characteristics of the orchards sampled are presented in Table 1. Median values are labeled on the figure and are shown as a broken line

median of 0.0173 lesions per leaf and 80.0% of the orchards having $\bar{y}_f < 0.30$ (Fig. 1c). Both scab incidence and lesion density increased from sampling in late spring, summer to early fall (Figs. 2a, c, e). The incidence of scabbed leaves was higher on cluster shoots than on terminal shoots (Fig. 2b), while incidence of scabbed shoots and lesion density was similar for both shoot types (Fig. 2d and f).

Distribution analysis

Incidence For assessments made at the leaf scale, the maximum likelihood procedure converged for 70 of the 95 (74%) data sets. The beta-binomial distribution fitted the data better than the binomial distribution in 51 of the 95 (54%) data sets according to a log likelihood ratio test; or 71% of the data sets where the maximum likelihood procedure converged. However, when the incidence of scab was above 0.05 the beta-binomial distribution fitted the data better than the binomial distribution according to the likelihood ratio test in all data sets (Table 2). The heterogeneity parameter of the beta-binomial distribution (θ_f) ranged from 0.00 to 0.08 with a median of 0.008, indicating a low degree of aggregation. For the cases where maximum likelihood estimation was not possible, the moment estimates of $\hat{p}_f$ were between 0.0002 and 0.05. Of the 70 data sets where maximum likelihood estimation of θ_f was possible, θ_f was significantly greater than 0 in 40 (43%) data sets.

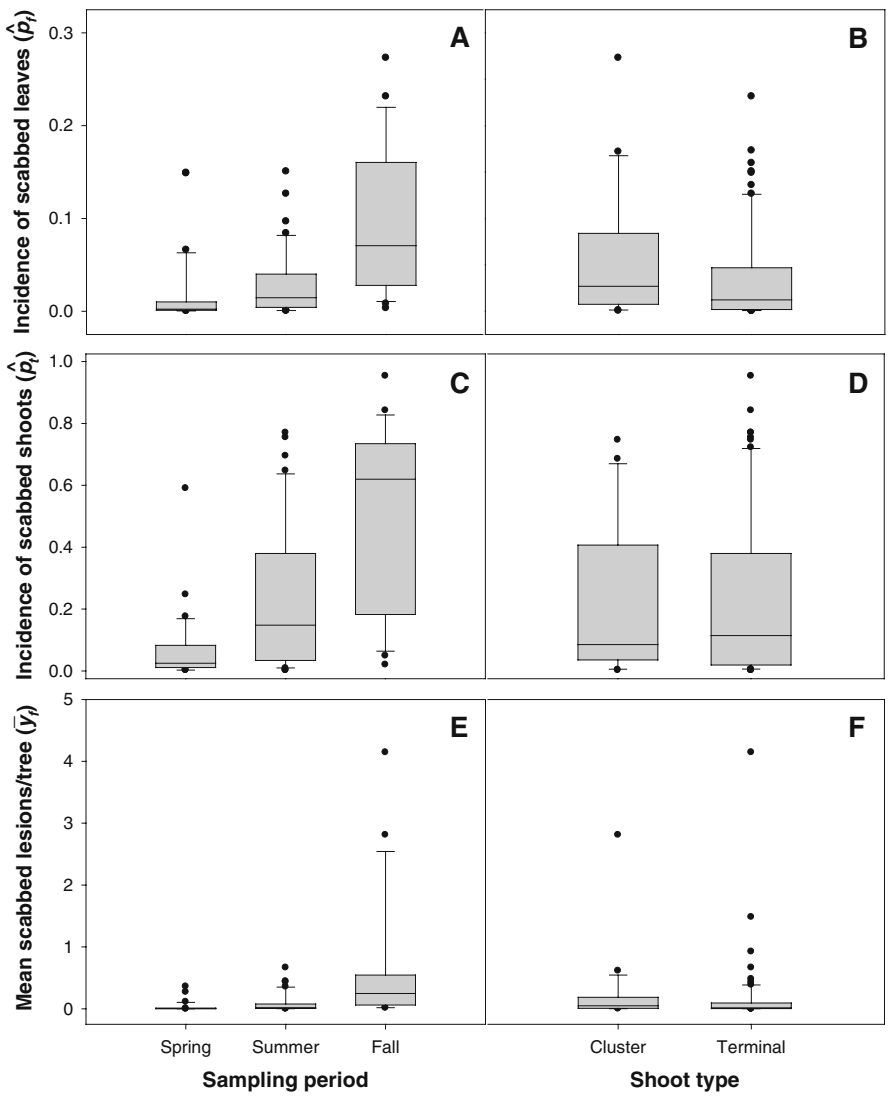

Fig. 2 Boxplots of the proportion of scabbed leaves (**a**–**b**), the proportion of scabbed shoots (**c**–**d**), and the number of lesions per leaf (**e**–**f**) observed in commercial orchards located in Quebec and sampled at different sampling periods (spring, summer and fall) (**a**,**c**,**e**) and on two shoot types (cluster and terminal) (**b**,**d**,**f**). The orchards were sampled from 1998 to 2007

Table 2 Results of spatial analysis characterizing the degree of heterogeneity of apple scab incidence in Quebec, Canada, from 1998 to 2007

Incidence class[a]	Leaf					Shoot				
	Data sets[b]	MLE[c]	LRS[d]	Theta[e]	D[f]	Data sets	MLE	LRS	Theta	D
0.00–0.05	72	65.3	38.9	0.002	1.17	35	31.5	2.86	0.000	0.99
0.05–0.10	10	100	100	0.019	2.82	12	75.0	41.7	0.036	1.24
0.10–0.20	11	100	100	0.023	4.45	13	76.9	61.5	0.070	1.45
0.20–0.40	2	100	100	0.043	4.90	18	100.0	55.6	0.070	1.63
0.40–0.60						5	80.0	20.0	0.019	1.18
0.60–0.80						12	100	83.3	0.100	1.45

[a] Classes end with the listed incidence value and begin with the first value above the listed value in each class

[b] Number of data sets in defined incidence class

[c] Percentage of data sets in which the maximum likelihood estimation (MLE) procedure converged to provide estimates of the beta-binomial parameters p and θ

[d] Percentage of data sets in which the beta-binomial distribution fit the data better than the binomial distribution according to the likelihood ratio test

[e] Median estimated beta-binomial parameter

[f] Median index of dispersion

The value of θ_f increased with increasing leaf scab incidence (Table 2).

For assessments made at the shoot scale, the maximum likelihood procedure converged for 64 of the 95 (68%) data sets. The beta-binomial distribution fitted the data better than the binomial distribution in 35 of the 95 (37%) data sets according to a log likelihood ratio test, or in 55% of the data sets where the maximum likelihood procedure converged. Unlike the results at the leaf scale, the beta-binomial distribution fitted the data better than the binomial distribution according to the likelihood ratio test in only 57% of the data sets when the incidence of scab was greater than 0.05 (Table 2). The heterogeneity parameter of the beta-binomial distribution (θ_t) ranged from 0.00 to 0.41 with a median of 0.028, indicating a low degree of aggregation. For the cases where maximum likelihood estimation was not possible, the moment estimates of $\hat{p}_t$ were between 0.02 and 0.20, except for one value at 0.40. Of the 64 data sets where maximum likelihood estimation of θ_t was possible, θ_t was significantly greater than 0 in 25 (39%) data sets.

Lesion density The negative binomial distribution fitted the data in 86% of the 95 data sets according to a chi-square goodness-of-fit test and the proportion of data sets that fitted the negative binomial distribution well varied from 50 to 100 depending on lesion density class (Table 3). The index of aggregation k ranged from 0.00 to 9.67 with a median of 0.068, indicating some degree of aggregation.

Table 3 Results of spatial analysis characterizing the degree of heterogeneity of apple lesion density (lesions per leaf per tree) in Quebec, Canada, from 1998 to 2007

Density class[a]	Data sets[b]	MLE[c]	χ^2-test[d]	k[e]	V/M[f]
0.00–0.05	60	78.33	66.67	1.545	2.886
0.05–0.10	9	100.00	100.00	1.314	8.385
0.10–0.20	8	100.00	87.50	1.09	17.802
0.20–0.40	9	100.00	77.78	1.402	81.795
0.40–0.60	5	100.00	60.00	1.4	36.159
0.60–0.80	2	100.00	50.00	1.341	52.334
0.80–1.00	2	100.00	100.00	1.771	51.237

[a] Classes end with the listed incidence value and begin with the first value above the listed value in each class

[b] Number of data sets in defined incidence class

[c] Percentage of data sets in which the maximum likelihood estimation (MLE) procedure converged to provide estimates of the negative-binomial parameter k

[d] Percentage of data sets in which the negative-binomial distribution fit the data according to chi-square goodness-of-fit test

[e] Median estimated negative-parameter k

[f] Median index of dispersion V/M

Indices of aggregation and power law relationships

Incidence At the leaf scale, the index of dispersion, *D*, ranged from 0.58 to 7.50, with a mean of 2.08 and median of 1.44. *D* was significantly greater than 1 in 57% of the data sets according to a chi-square test indicating some degree of aggregation in most data sets. At the shoot scale, *D* ranged from 0.86 to 3.65 with a mean of 1.36 and median of 1.19. *D* was significantly greater than 1 in 42% of the data sets according to a chi-square test indicating some degree of aggregation in some data sets. The value of *D* increased with increasing leaf scab incidence (Table 2).

The binary power law provided a good fit to the incidence data at both assessment scales (Fig. 3a and b). The estimated slope and intercept parameters were significantly greater than 1 and 0, respectively, according to *t*-tests, indicating that heterogeneity changed systematically with disease incidence at both assessment scales. The covariance analysis indicated that the factor 'sampling season' had a significant effect only on the intercept parameter at the leaf scale (Table 4). Consequently, the incidence data at the leaf scale were analyzed separately for each sampling season (Fig. 4a). The factor 'shoot type' had no effect on either parameter estimate at both assessment scales.

Lesion density The variance to mean ratio ranged from 0.91 to 500.10 with mean of 15.95 and median of 2.49 (Table 3). The variance to mean ratio was significantly greater than 1 in 72% of the data sets according to a chi-square test indicating that lesion density was aggregated. Taylor's power law provided a good fit to the lesion density data (Fig. 3c). The estimated slope and intercept parameters were significantly greater than 1 and 0, respectively, according to a *t*-test. The covariance analysis indicated that the factor 'season' had a significant effect on the intercept parameter based on a test of the difference of model deviances (Table 4). The factor 'shoot type' had no effect on the parameter estimates of Taylor's power law (Table 4). Consequently, the lesion density data were analyzed separately for each sampling season (Fig. 4b).

Relationships between measurements of disease intensity

Figures 5a and b show the relationships between the incidence of diseased leaves or shoots $\left(\hat{p}_f \text{ or } \hat{p}_t\right)$ and

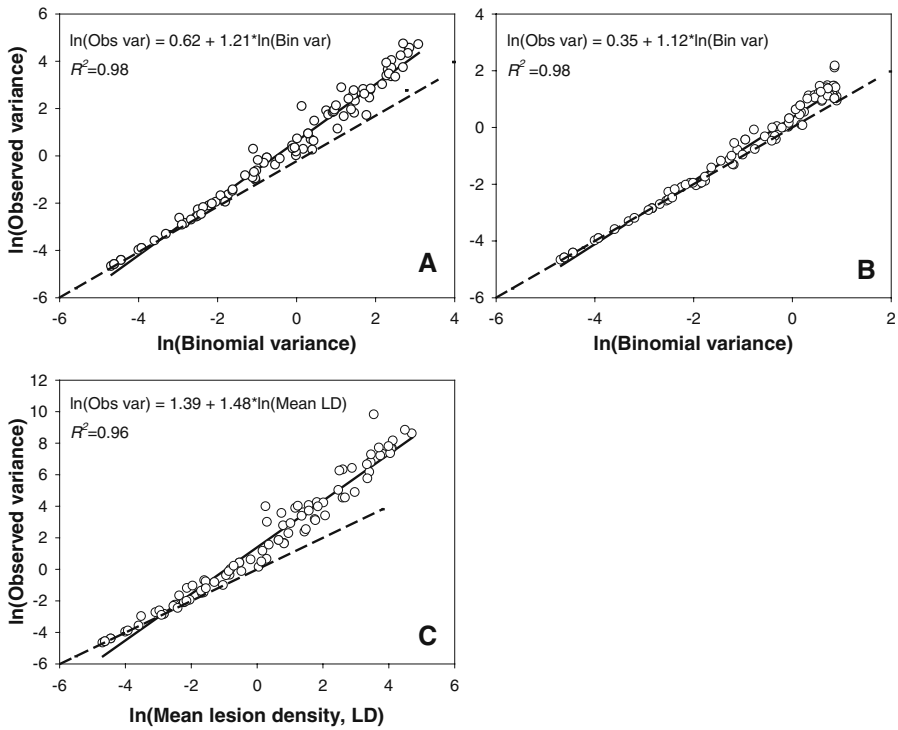

Fig. 3 Relationship between the logarithm of the observed variance ($v_{obs,\bullet}$) and logarithm of the expected variance for a binomial distribution ($v_{ran,\bullet}$) for the incidence of apple scab from assessments of leaves (**a**) and assessments of shoots (**b**) (Eq. 1) and relationship between the logarithm of the observed variance s_f^2 and logarithm of mean lesion density $\bar{y}_f$ (**c**) (Eq. 2) for apple scab assessments on leaves in Quebec, Canada from 1998 to 2007

Table 4 Deviances (Dev.) and sequential *F* tests for the covariance models testing for the effects of the factors season and shoot type on the intercept and slope parameters of the binary power law (Eq. 1) for apple scab orchards located in Quebec in 1998–2007

Measurement	Model	df_1^b	df_2^b	Intercept				Slope			
				Dev.	Diff [c]	F[d]	*P*-value	Dev.	Diff	*F*	*P*-value
Leaf incidence	Power Law	93		13.14				13.14			
	+Season	91(89)	2	11.46	1.68	6.67	0.002	11.12	0.34	1.35	0.264
	+Type	92(91)	1	12.91	0.23	1.61	0.207	12.90	0.02	0.11	0.736
Shoot incidence	Power Law	93		5.66				5.66			
	+Season	91(89)	2	5.39	0.25	2.25	0.111	5.36	0.03	0.23	0.799
	+Type	92(91)	1	5.66	0.00	0.01	0.941	5.60	0.06	0.97	0.328
Lesion density	Power Law	93		49.17				49.17			
	+Season	91(89)	2	42.23	6.95	7.49	0.001	42.04	0.19	0.20	0.822
	+Type	92(91)	1	48.55	0.62	1.18	0.281	48.55	0.00	0.01	0.937

[a] $\ln(A_x)$ and b are the intercept and slope parameters, respectively, of the binary power law (Eq. 1)

[b] df_1 is the degrees of freedom for the model deviance. The numbers in parentheses are the degrees of freedom for the models used to test for a factors effects on the slope parameters; df_2 is the degrees of freedom for the factor in test

[c] The difference between the deviance for the power law model and the power law model with the factor included as either an intercept term or as an interaction with the slope term. If the deviance was reduced by $\chi^2_{df_2}$ the factor was considered to improve the model significantly

[d] The *F* test is a test of a factors significance based on its sequential sum of squares (Type I)

the incidence of diseased sampling units ($\widetilde{p}_{su}$), respectively. The curves shown are the binomial model (case i, solid line), the beta-binomial model (case ii, dotted line), effective sample size model based on the power law parameters (case iii, dash-dot line), and effective sample size model based on Madden and Hughes (1999) (case iv, broken line) and the best fitting model with slope and intercept parameters estimated (case v, large dashed line). No parameter estimation was necessary for cases i, ii, iii, and iv. The binomial model provided the worst fit to the data at both the leaf and shoot scales (Table 5). Applying any correction for heterogeneity to the binomial model improved the fit to the data. The effective sample size model based on Madden and Hughes (1999) gave the best fit to the leaf scale data, while the effective sample size model based on the power law parameters (case iv) provided the best fit to the shoot scale data (Table 5). However, the best-fitting models overall were obtained by fitting both the intercept and slope parameters via regression (as expected).

Figure 5c shows the relationship between the incidences of diseased leaves and shoots. After the best fitting model (case ix), the binomial model provided the best fit (case vi) (Table 5). In this case, correcting for heterogeneity tended to produce underestimates.

Incidence-lesion density relationships The CLL transformation of scab incidence and logarithmic transformation of lesion density were useful for obtaining a linear relationship (on a transformed scale) and explaining a reasonably high percentage of the variation in transformed lesion density with $R^2_{adj} = 0.97$ and $R^2_{adj} = 0.94$ at the leaf and shoot scales, respectively. Similar results were obtained for model fit comparisons using R^{2*}=0.91 and R^{2*}=0.85, at the leaf and shoot scales, respectively (Fig. 6a–b). At the leaf scale, the model tended to underestimate lesion density at scab incidence of more than 0.10 scabbed leaves per tree. Similarly, at the shoot scale the model tended to underestimate lesion density at scab incidence of more than 0.20 scabbed shoots per tree.

Discussion

In this study, the spatial distribution of apple scab was investigated and used to characterize different measurements of disease intensity. Specifically, the relationship

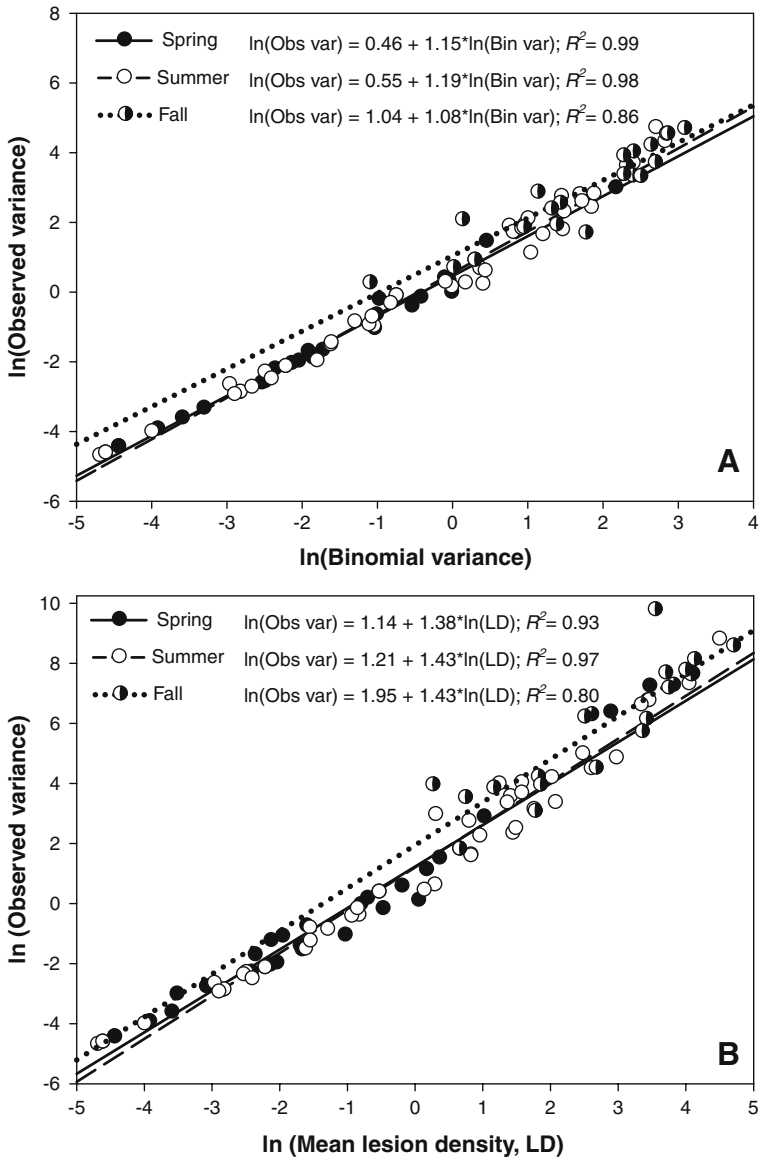

Fig. 4 Relationship between the logarithm of the observed variance ($v_{obs,\bullet}$) and logarithm of the expected variance for a binomial distribution ($v_{ran,\bullet}$) for the incidence of apple scab from assessments of leaves (**a**) (Eq. 1) and relationship between the logarithm of the observed variance s_f^2 and logarithm of mean lesion density $\bar{y}_f$ (**b**) (Eq. 2) for apple scab assessments on leaves sampled at different periods in Quebec, Canada from 1998 to 2007

between lesion density and apple scab incidence, and the relationship between apple scab incidences at multiple hierarchical scales were characterized with the goal of producing models to predict the incidence or lesion density at the leaf or shoot scales from assessments of disease intensity at either the shoot or tree scales, respectively. These relationships were characterized relative to shoot types and the timing of assessment to determine the necessity of tailoring predictions at different stages of epidemic development. Ultimately, the intent is to use this information for decision making regarding the management of summer scab and pre-harvest fungicide sprays.

As opposed to most of the work previously conducted on apple scab measurements (Jeger 1981, 1983), in this study, incidence and heterogeneity of apple scab were characterized using data collected in managed orchards, typically having low levels of disease. Distributional and power law analyses, used to characterize spatial distribution, showed that apple scab incidence at both the leaf and shoot scales were characteristic of a non-random pattern of disease with a low level of aggregation that changed systematically with disease incidence. These results are supported by the findings of Charest et al. (2002) who showed that apple scab inoculum, measured as potential ascospore

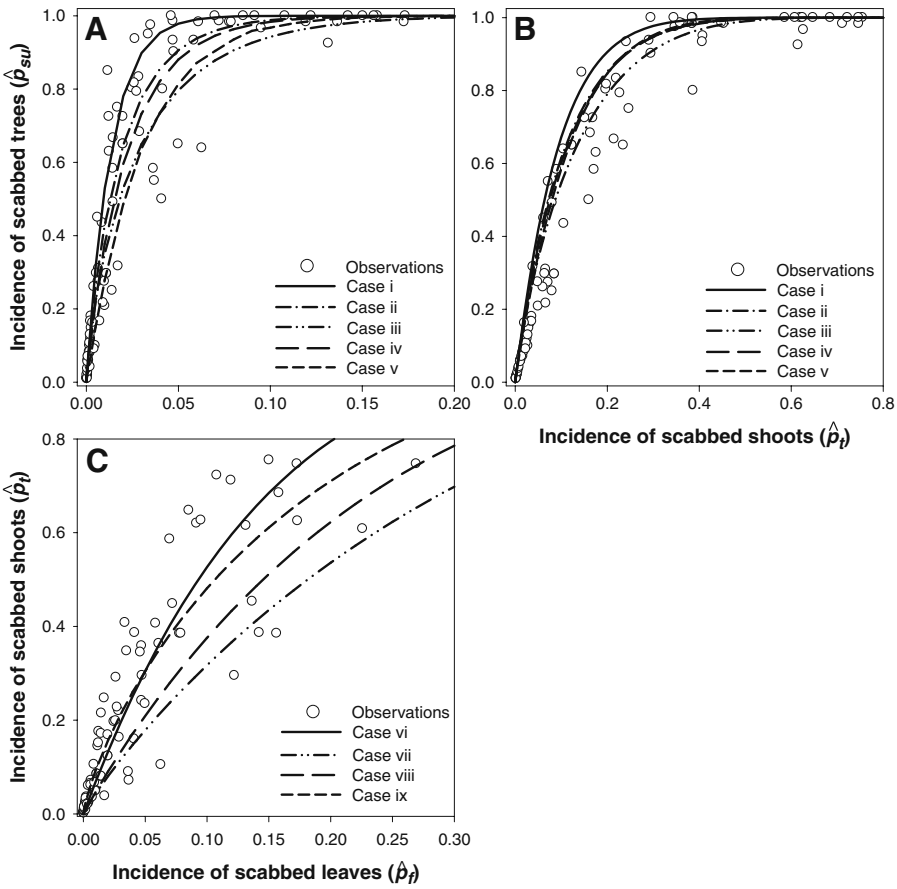

Fig. 5 Relationships between incidence of diseased leaves or shoots $\left(\hat{p}_f \text{ or } \hat{p}_t\right)$ and incidence of diseased sampling units $(\tilde{p}_{su})$, respectively (**a** and **b**) and relationship between the incidence of diseased leaves $\left(\hat{p}_f\right)$ and shoots $\left(\hat{p}_t\right)$ (**c**) for apple scab sampled in Quebec, Canada from 1998 to 2007. Case numbers are referenced in the text and in Table 5

dose (PAD), was aggregated within an orchard, by Carisse et al. (2006) who reported that ascospore deposition within an apple tree was not randomly distributed following peaks of ascospore ejection, as well as by Carisse et al. (2009) who found that beta-binomial parameters p and θ could be calculated in 65% of data sets on number of scabbed leaves per shoot, while the binomial distribution provided the best fit for only 22% of the data sets. In the present study, a covariance analysis indicated that sampling season had a significant effect on the intercept parameter at the leaf scale meaning that the relationship between incidence and aggregation changed significantly over the course of the season. Although the analyses done here do not explicitly identify the cause of this, one could speculate that it is the result of the switch from ascospores to conidia as the predominant source of inoculum as the season progresses and/or the result of climate differences between spring and summer.

The spatial pattern of apple scab lesions (lesion density) was also found to be aggregated. The negative binomial distribution fitted the data well in 86% of the data sets, and Taylor's power law provided a good fit to the lesion density data. As with disease incidence, a covariance analysis indicated that the sampling season had a significant effect on the intercept parameters. These results support the findings of Xu and Robinson (2005) who found the lesion density of apple scab to be aggregated on fruit. More data sets were found to exhibit aggregation when lesion density, rather than disease incidence, was used as the measurement of disease intensity. Although one would expect some agreement, this emphasizes the fact that disease incidence and lesion density measure somewhat different characteristics of the spatial properties of disease.

Various models characterizing the relationship of the incidences of scab between different hierarchical levels were tested. The models requiring direct curve fitting provided the best fit, as expected. Of the models that did not require direct curve-fitting, the effective sample size model proposed by Madden and

Table 5 Deviances and degrees of freedom (*df*) for five cases describing the relationship between incidence of apple scab for assessments taken at the leaf (p_f) or shoot scale (p_t) and at the sampling unit (tree) scale (p_{su}), and for four cases describing the relationship between the incidence of apple scab from assessments of leaves and the incidence of apple scab from assessments of shoots in apple orchards located in Quebec, Canada

Scale	Model case number, variables, and parameters					Model fit	
	Case[a]	Offset[b]	X[c]	Int.[d]	Slope[d]	Dev.[e]	*df*
Leaf/Tree	i	$\ln(n) + \mathrm{CLL}(\hat{p}_f)$[f]	–	–	–	1066.4	95
	ii	$\ln\left[-\ln\prod_{j=0}^{n-1}\left(\frac{1-\hat{p}_f+j\,\theta}{1+j\,\theta}\right)\right]$	–	–	–	578.9	95
	iii	$\ln(n_\delta) + \mathrm{CLL}(\hat{p}_f)$	–	–	–	695.8	95
	iv	$\ln(n_v) + \mathrm{CLL}(\hat{p}_f)$	–	–	–	564.2	95
	v	$\mathrm{CLL}(\hat{p}_f)$	$\ln[\hat{p}_f(1-\hat{p}_f)]$	-ln(a)	1-b	560.8	93
Shoot/Tree	i	$\ln(n) + \mathrm{CLL}(\hat{p}_t)$[f]	–	–	–	993.9	95
	ii	$\ln\left[-\ln\prod_{j=0}^{n-1}\left(\frac{1-\hat{p}_t+j\,\theta}{1+j\,\theta}\right)\right]$	–	–	–	613.2	95
	iii	$\ln(n_\delta) + \mathrm{CLL}(\hat{p}_t)$	–	–	–	397.0	95
	iv	$\ln(n_v) + \mathrm{CLL}(\hat{p}_t)$	–	–	–	572.2	95
	v	$\mathrm{CLL}(\hat{p}_t)$	$\ln[\hat{p}_t(1-\hat{p}_t)]$	-ln(a)	1-b	236.0	93
Leaf/Shoot	vi	$\ln(n/m)+\mathrm{CLL}(p_f)$	–	–	–	2450.3	95
	vii	$\ln(n_\delta/m_\delta) + \mathrm{CLL}(p_f)$	–	–	–	3706.0	95
	viii	$\ln(n_\nu/m_\nu)+\mathrm{CLL}(p_f)$	–	–	–	3168.5	95
	ix	$\mathrm{CLL}(\hat{p}_f)$	$\ln[\hat{p}_f(1-\hat{p}_f)]$	-ln(a)	1-b	2036.5	93

[a] Roman numeral refers to the "case" as described in the text

[b] Refers to terms in the model that are considered known

[c] Independent variable; "–"=no independent variable in the model. All terms are offset, therefore no parameters are estimated

[d] Theoretical value of the intercept and slope estimate; "–"=no intercept or slope estimated

[e] Calculated deviance. For binomial data, values of the deviance equal to or less than the model degrees of freedom indicate a good fit. When comparing two models, if the reduction in deviance is greater than χ^2_v, where $v = df_1 - df_2$ is the absolute difference of the degrees of freedom for competing models, then the model with the smaller deviance is the better fitting model

[f] $\mathrm{CLL}(z) = \ln[-\ln(1-z)]$; complementary log-log function

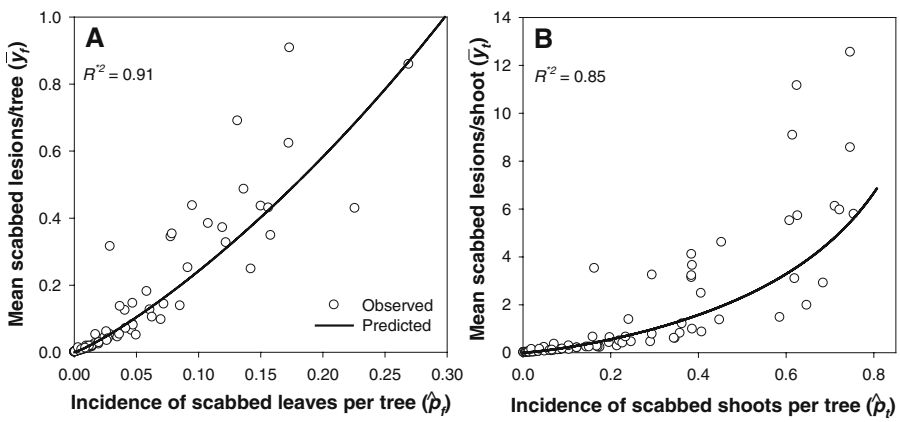

Fig. 6 Relationship between scab incidences $\left(\hat{p}_f \text{ or } \hat{p}_t\right)$ and mean lesion density $\left(y_f \text{ or } y_t\right)$ for untransformed (**a**, **c**) and complementary log-log transformed incidence (**b**, **d**), $\mathrm{CLL}(p_f) = \ln[-\ln(1-p_f)]$ and $\mathrm{CLL}(p_t) = \ln[-\ln(1-p_t)]$ (Eqs. 11a and 11b) at the leaf (**a**, **b**) and shoot scale (**c**, **d**) for data collected in commercial orchards located in Quebec, Canada from 1998 to 2007

Hughes (1999) best described the relationship between incidence of diseased leaves and incidence of diseased trees, while the relationship between incidence of diseased shoots and incidence of diseased trees was best described by the effective sample size model based on the power law parameters. Although both effective sample size models are based on the beta-binomial model, they accounted for the observed heterogeneity better than the beta-binomial model probably because disease incidence did not follow the beta-binomial distribution exactly. From a sampling perspective, the relationship between the incidence of trees and that of leaves (in particular) or shoots does not offer enough precision to be of practical use and, perhaps most important, most scouts would feel uncomfortable making decisions of the disease status of leaves based on the disease status of trees. However, the relationship between the incidences of diseased leaves and shoots, as described by either the curve-fitted model or the binomial model (no curve fitting), offers an alternative (Turechek, and Madden 2001). Although results from this group of models (i.e., models vi through ix) were the most variable, they could be used to predict incidence at the leaf scale from incidence at the shoot scale and would likely be most readily adopted because it is not as far of a stretch to estimate leaf incidence from measurements of shoot incidence as it is to measure leaf incidence from tree incidence.

The relationship between lesion density and scab incidence at the leaf and shoot scales was best described by a linear model of the logarithm of lesion density on the complementary log-log transformation of leaf incidence or shoot incidence. The relationship between incidence and severity or between incidence and lesion density has been studied for several diseases (Dillard and Seem 1990; Groth et al. 1999; Jeger 1981; Seem et al. 1981; Xu and Madden 2002). In most of these studies, limitations in the practical use of incidence-severity (or incidence–lesion density) models were pointed out. These limitations are related to the lack of consistency of the models with respect to location, season, stage of epidemic, host susceptibility, and farming practice. Nevertheless, when a reliable incidence–lesion density model is available, it may be possible to use unskilled personnel to record incidence rather than the time-consuming and more demanding lesion counts. The results of this study are similar to the pioneering work of Jeger (1981) who reported a curvilinear relationship between leaf scab incidence and severity at the shoot level, in unsprayed plots planted with mixed cultivars or with the cultivar Cox's Orange Pippin.

The current practice for apple scab management in eastern North America is to effectively control primary infections to avoid disease build-up and the need for fungicide sprays during the summer. Growers and crop specialists often decide arbitrarily or based on a limited sampling whether or not summer fungicide sprays are necessary. In managed orchards the level of scab is often low thus it is difficult to know without any prior knowledge of the spatial structure of apple scab, which disease measurement (incidence or lesion density) is best, and at what scale measurements of disease should be made. Our research has indicated that sampling plans for summer scab management should account for the low level of aggregation in late spring or early summer. However, this research could serve as groundwork for answering these questions (Madden, and Hughes 1999).

In North America, apple growers must produce fruits free of diseases to respond to the consumer demand and to be stored for prolonged period of time. Their production should be profitable but not too expensive for the consumer and, it should be within acceptable limits of pesticide residues and produced in such way that it will not harm the environment. In this context, decisions regarding summer scab management play a major role. If summer scab is not properly controlled, fruits scab may exceed the limit for profitability, however, several fungicides used to manage summer scab have a bad environmental profile and sprays near harvest increases the probability of fungicide residues on fruits (Carisse et al. 2011). The quality of decision making is thus very important and should not be based only on growers' 'feeling' about the need to apply a fungicide. In this regard, data obtained by sampling are crucial for decision making. In practice, cost and reliability of sampling will influence adoption of sampling practices by growers. In sampling for apple scab, the most time consuming part is to count the lesions. In this study we demonstrated that there is a good relationship between the incidence of scabbed leaves and lesion density, and hence counting lesions as a means to quantify disease could be circumvented. However, the results also show that sampling should be done at the leaf level, in other words it is not sufficient to determine the incidence of

scabbed shoots. Finally, the level of aggregation especially in the spring should be considered when sampling for apple scab incidence (Carisse et al. 2009). The results of this study also indicate that some research challenges remain for sampling very low scab incidence and to relate scab incidence with crop loss.

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References

Aitkin, M., Anderson, D., Francis, B., & Hinde, J. (1989). *Statistical Modelling in GLIM*. Oxford: Clarendon.

Carisse, O., & Jobin, T. (2010). Resistance to dodine in populations of *Venturia inaequalis* in Quebec, Canada. Online. *Plant Health Progress* doi:10.1094/PHP-2010-0614-01-RS.

Carisse, O., Rolland, D., Talbot, B., & Savary. (2006). Heterogeneity of the aerial concentration and deposition of ascospores of *Venturia inaequalis* within a tree canopy during the rain. *European Journal of Plant Pathology, 117*, 13–24.

Carisse, O., Meloche, C., Boivin, G., & Jobin, T. (2009). Action thresholds for summer fungicide sprays and sequential classification of apple scab incidence. *Plant Disease, 93*, 490–498.

Carisse, O., Tremblay, D.-M., Jobin, T., and Walker, A.S. (2011. Disease Decision Support) Systems: Their Impact on Disease Management and Durability of Fungicide Effectiveness "in Fungicides, Book edited by: Odile Carisse, ISBN: 978-953-307-266-1, Publisher: InTech, Publishing

Charest, J., Dewdney, M., Paulitz, T. C., Philion, V., & Carisse, O. (2002). Spatial distribution of *Venturia inaequalis* airborne ascospores in orchards. *Phytopathology, 92*, 769–779.

Dewdney, M., Charest, J., Paulitz, T., & Carisse, O. (2003). Multivariate analysis of apple cultivar susceptibility to *Venturia inaequalis* under greenhouse conditions. *Canadian Journal of Plant Pathology, 25*, 387–400.

Dillard, H. R., & Seem, R. C. (1990). Incidence-severity relationship for common maize rust on sweet corn. *Phytopathology, 80*, 842–846.

Farrington, C. P. (1992). Estimating prevalence by group testing using generalized linear models. *Statistics in Medicine, 11*, 1591–1597.

Gosme, M. (2008). Comment analyser la structure spatiale et modéliser le développement spatio-temporel des épiphyties. *Canadian Journal of Plant Pathology, 30*, 4–23.

Groth, J. V., Ozmon, E. A., & Busch, R. H. (1999). Repeatability and relationship of incidence and severity measures of scab of wheat caused by *Fusarium graminearum* in inoculated nurseries. *Plant Disease, 83*, 1033–1038.

Holb, I. J., Heijne, B., & Jeger, M. J. (2005). Summer epidemics of apple scab: The relationship between measurements and their implications for the development of predictive models and threshold levels under different disease control regimes. *Journal of Phytopathology, 151*, 335–343.

Hughes, G., & Madden, L. V. (1993). Using the beta-binomial distribution to describe aggregated patterns of disease incidence. *Phytopathology, 83*, 759–763.

Hughes, G., McRoberts, N., Madden, L. V., & Gottwald, T. R. (1997). Relationship between disease incidence at two levels in a spatial hierarchy. *Phytopathology, 87*, 542–550.

Jeger, M. J. (1981). Disease measurement in a study of apple scab epidemics. *The Annals of Applied Biology, 99*, 43–51.

Jeger, M. J. (1983). Damage and loss in fruit orchards caused by airborne fungal pathogens. In R. K. S. Wood & G. J. Jellis (Eds.), *Plant Diseases: Infection Damage and Loss* (pp. 225–235). Oxford: Blackwell Scientific Publication.

Jobin, T., & Carisse, O. (2007). Incidence of myclobutanil- and kresoxim-methyl-insensitive isolates of *Venturia inaequalis* in Quebec orchards. *Plant Disease, 91*, 1351–1358.

MacHardy, W. E. (1996). *Apple Scab: Biology, Epidemiology, and Management* (p. 545). St. Paul: The American Phytopathological Society.

Madden, L. V., & Hughes, G. (1995). Plant disease incidence: Distributions, heterogeneity, and temporal analysis. *Annual Review of Phytopathology, 33*, 529–564.

Madden, L. V., & Hughes, G. (1999). An effective sample size for predicting plant disease incidence in a spatial hierarchy. *Phytopathology, 89*, 770–781.

Madden, L. V., Hughes, G., & Ellis, M. A. (1995). Spatial heterogeneity of incidence of grape downy mildew. *Phytopathology, 85*, 269–275.

Madden, L. V., Hughes, G., & van den Bosch, F. (2007). *The study of plant disease epidemics* (p. 421). St. Paul: The American Phytopathological Society.

McRoberts, N., Hughes, G., & Madden, L. V. (2003). The theoretical basis and practical application of relationships between different disease intensity measurements in plants. *The Annals of Applied Biology, 142*, 191–211.

Paul, P. A., El-Allaf, S. M., Lipps, P. E., & Madden, L. V. (2005). Relationships between incidence and severity of Fusarium head blight on winter wheat in Ohio. *Phytopathology, 95*, 1049–1060.

Rao, J. N. K., & Scott, A. J. (1992). A simple method for the analysis of clustered binary data. *Biometrics, 48*, 577–585.

Reardon, J. E., Berkett, L. P., Garcia, M. E., & Gotlieb, A. (2005). Field evaluation of a new sequential sampling technique for determining apple scab risk. *Plant Disease, 89*, 228–236.

Seem, R. C. (1984). Disease incidence and severity relationships. *Annual Review of Phytopathology, 22*, 133–150.

Seem, R. C., Gilpatrick, J. D., & Pearson, R. C. (1981). Fungicide influence on the relationships between incidence and severity of powdery mildew on apple. *Phytopathology, 71*, 947–950.

Taylor, L. R. (1961). Aggregation, variance and mean. *Nature (Lond.), 189*, 732–735.

Turechek, W. W., & Madden, L. V. (1999). Spatial pattern analysis of strawberry leaf blight in perennial production systems. *Phytopathology, 89*, 421–433.

Turechek, W. W., & Madden, L. V. (2001). Effect of scale on plant disease incidence and heterogeneity in a spatial hierarchy. *Ecological Modeling, 144*, 77–95.

Turechek, W. W., & Madden, L. V. (2003). A generalized linear modeling approach for characterizing disease incidence in a spatial hierarchy. *Phytopathology, 93*, 458–466.

Turechek, W. W., & Mahaffee, W. F. (2004). Spatial pattern analysis of hop powdery mildew in the Pacific Northwest: Implications for sampling. *Phytopathology, 94*, 1116–1128.

Xu, X., & Madden, L. V. (2002). Incidence and density relationships of powdery mildew on apple. *Phytopathology, 92*, 1005–1014.

Xu, X., & Robinson, J. (2005). Modelling the effects of wetness duration and fruit maturity on infection of apple fruits of Cox's Orange Pippin and two clones of Gala by Venturia inaequalis. *Plant Pathology, 54*, 347–356.