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Yield loss in apple caused by *Monilinia fructigena* (Aderh. & Ruhl.) Honey, and spatio-temporal dynamics of disease development

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Abstract

Monilinia fructigena (Aderh. & Ruhl.) Honey causes considerable yield losses in pome fruit culture. During a field study in the Netherlands in 1997 and 1998, the increase in disease incidence in time was assessed and final pre- and post-harvest losses were recorded in the susceptible apple cultivars James Grieve and Cox's Orange Pippin. Each individual tree was considered as a unique quadrat, and the spatial distribution of diseased fruits among fruit trees at every assessment date was characterised by a dispersion index, Lloyd's index of patchiness (LIP). Spatial autocorrelation was applied to detect potential clustering of trees with diseased fruits within rows. In cv. James Grieve, the rate of increase of disease incidence was constant up to harvest time, whereas in cv. Cox's Orange Pippin disease incidence increased markedly 3 weeks before harvest time, which coincided with the harvest of cv. James Grieve in neighbouring rows. Pre-harvest disease incidence was 4.2–4.3% in cv. James Grieve in both years, in cv. Cox's Orange Pippin this was 4.4% in 1997 and 2.7% in 1998. Post-harvest yield losses amounted on average 1.5–2.0% for both cultivars, no significant differences were found between the cultivars (t-test, $P = 0.05$). Both in 1997 and 1998, clustering of diseased fruits among fruit trees was detected; LIP values were significantly higher than 1 ($P = 0.05$ in 1997, $P = 0.01$ in 1998). Clustering of trees with diseased fruits was detected in 1998, when significant ($P = 0.05$) positive correlation coefficients occurred for 2nd, 3rd and 4th lag-order distances in cv. James Grieve, and a significant ($P = 0.05$) positive first-order correlation in cv. Cox's Orange Pippin. Wounding agents, such as insects and birds, may play an important role in the underlying disease dynamics, and crop losses may be minimised by control of these agents.

Introduction

In pome fruit growing areas, *Monilinia fructigena* (Aderh. & Ruhl.) Honey is an important pathogen that causes fruit rot. *M. fructigena* belongs to the group of brown rot fungi, which includes two other species, *M. laxa* (Aderh. & Ruhl.) Honey and the EU-listed quarantine organism *M. fructicola* (Wint.) Honey. The latter two species more commonly affect stone fruits, although *M. laxa* forma *mali* causes blossom wilt and canker of apple trees (Byrde and Willetts, 1977).

Many studies have focussed on the post-harvest stage of fruit rot caused by *M. fructigena* (Berrie, 1989; Snowdon, 1990; Falconi and Mendgen, 1994), in which losses caused by *M. fructigena* are nowadays usually low. Berrie (1989) reported mean post-harvest losses in cultivar Cox's Orange Pippin that ranged from 0.1% to 0.6% in the seasons 1982–1988. Crop losses are an important criterion to declare an organism to be a quarantine pest (OEPP/EPPO, 1993). *M. fructigena* is declared a quarantine pest in the USA, Australia and New Zealand (CMI, 1991). Detailed

studies of pre-harvest yield losses in pome fruit caused by *M. fructigena* are rare in recent literature. In a study over three consecutive seasons, Moore (1950) recorded an average of about 9% of apple fruits becoming infected with brown rot. Differences in susceptibility among apple cultivars under field conditions have been reported from Poland (Cimanowski and Pietrzak, 1991). Under Dutch conditions, apple cvs. James Grieve and Cox's Orange Pippin are among the most susceptible (Anonymous, 1991).

Few studies have been made to characterise temporal and spatial aspects of increase in *Monilinia*-diseased fruits in orchards. As part of a study that dealt with fungicide-resistant strains of *M. fructicola* in New Zealand, Elmer et al. (1998) analysed the spatial distribution of peach fruits affected by brown rot at harvest time. They determined brown rot incidence after a short post-harvest period under controlled conditions. No attempt was made to determine disease incidence during the pre-harvest period *in situ*. In stone fruits, disease incidence can increase very rapidly around harvest time, as mature fruits become more readily infected (Corbin, 1963; Zehr, 1982). *M. fructicola* is able to infect uninjured stone fruits even at the pre-pit-hardening stage (Corbin, 1963). Latent infections of *M. fructicola* can also play a role in the rapid disease development observed around harvest time (Jenkins and Reinganum, 1965; Northover and Cerkauskas, 1994). Latent infections occur in immature fruits particularly in a season of severe blossom infection, and after a period of quiescence that may last several weeks, rots start to develop as the fruits ripen. In contrast, *M. fructigena* relies almost exclusively on pre-existing wounds in the fruit skin for penetration, although uninjured, ripe apples have been successfully infected via lenticels (Horne, 1933). Latent infections of immature fruits as described for *M. fructicola* have never been reported for *M. fructigena*.

The objective of this study was to study space–time variation of disease development of *M. fructigena* in apple. More specifically, it focussed on: (i) quantification of pre- and post-harvest yield losses caused by *M. fructigena* in two susceptible apple cultivars; (ii) the increase in disease incidence in time, based on the hypothesis that disease incidence gradually increases up to harvest time, unlike the rapid increase at harvest time frequently observed in stone fruits affected by *M. fructicola* or *M. laxa*; (iii) determination of spatial pattern of diseased fruits among fruit trees in time; and (iv) determination of the extent of clustering of trees with diseased fruits. Spatial analysis allows for the development of hypotheses to account for observed associations (Campbell and Madden, 1990), thus giving information on the population dynamics of the pathogen. Based on the results, we present some hypotheses in relation to the underlying disease dynamics, and make a comparison with those postulated for *M. laxa* and *M. fructicola* in stone fruits.

Materials and methods

Study area

The study was done in an experimental orchard (IPM) situated in the Rhine basin in the centre of the Netherlands during two consecutive growing seasons in 1997 and 1998. The orchard contained three rows of cv. James Grieve alternating with two rows of cv. Cox's Orange Pippin (Figure 3). The trees were planted in 1984, at a tree spacing of 3×1.25 m. In both years cv. James Grieve bore a moderate fruit crop, with a mean number of 70 fruits per tree in 1997 and 55 in 1998; mean numbers of cv. Cox's Orange Pippin were 58 and 50, respectively. Regular spraying against apple scab (*Venturia inaequalis*) followed normal commercial practice from bud break until the end of June in both years. Insecticide was applied after blossoming against apple sawfly (*Hoplocampa testudinea*) and early codling moth (*Pammene rhediella*) after blossoming to prevent early dropping of fruit.

Evaluations

Disease incidence was assessed by counting the number of fruits per tree which showed sporulation of *M. fructigena*. Fruits showing sporulation of *M. fructigena* in the tree canopy were not removed, whereas those which had dropped to the ground were removed to the grass pathway between the rows to avoid double countings. Observations were made at intervals of 7–9 days until harvest time, starting on 16 July in 1997, and 1 week earlier in 1998. For cv. Cox's Orange Pippin, observations were less frequent at the beginning of the epidemic because of a minimal increase in disease incidence. Harvest of cv. James Grieve took place on 22 August (day 234) in 1997 and 12 August (day 224) in 1998; the equivalent dates for cv. Cox's Orange Pippin were 12 (day 255) and 2 September (day 245), respectively. At harvest time, the number of fruits per box was determined per cultivar for five

randomly chosen boxes. The number of harvested fruits was estimated by multiplying the mean number of fruits per box by the total number of boxes. Percentage disease incidence was defined as the quotient of the (cumulative) number of *Monilinia*-diseased apples at a certain assessment date and the number of harvested fruits, multiplied by 100.

After harvest, the boxes with fruits were stored in an open shed, protected from rain but not wind. Post-harvest disease assessments were made 4 days after harvest in 1997, and 7 days after harvest in 1998. Mean daily temperatures registered in the post-harvest period ranged from 15 to 20 °C in both years. Disease incidence in storage was calculated as the number of fruits showing sporulation of *M. fructigena* relative to the total number of fruits per box. For each cultivar, mean disease incidence of five boxes was determined, and means of both cultivars were compared with a *t*-test.

Spatial statistics

The spatial pattern of diseased fruits among fruit trees was determined for each cultivar. The objects of study were single trees, and each week the (cumulative) number of *Monilinia*-diseased apples per tree was determined. In this study 'clustering of fruits' is defined as the spatial condition in which the number of diseased fruits per tree is more locally condensed than at random (Madden, 1989), i.e. the situation where there are trees with a considerable number of diseased fruits whilst others have none. Furthermore, it was our aim to study to what extent 'clustering of trees with diseased fruits' appeared within the field.

Lloyd's index of patchiness (LIP) was calculated to determine clustering of diseased fruits. LIP was calculated on the basis of the (cumulative) number of diseased fruits per tree. Per cultivar, the rows were considered as one, and the analysis performed. LIP constitutes the ratio of mean crowding (m^*) and mean density (m), where mean crowding is defined as the mean number of other diseased fruits per tree per diseased fruit, and mean density is the mean number of diseased fruits per tree (Lloyd, 1967).

Mean crowding was calculated as

$$m^* = m + \left(\frac{s^2}{m} - 1\right),$$

where m is the estimated mean number of diseased fruits per tree and s^2 the estimated variance.

Unlike mean density (m), m^* is not affected by trees without diseased fruits. The data set with the number of diseased fruits per tree at each assessment date, was considered as a sample of the total population of orchards where cv. James Grieve and cv. Cox's Orange Pippin are grown in an alternating one-row system. To calculate confidence bounds for LIP we applied a bootstrap procedure (Efron and Tibshirani, 1993). Numbers were randomly selected with replacement from the original data set to create a new data set of the same size, for which the LIP value was calculated. This was done 10,000 times, and subsequently mean LIP and a 95% and 99% confidence interval for the population mean were determined. Clustering was considered to be more pronounced when significant at $P = 0.01$ (99% confidence interval) compared with significance at $P = 0.05$ (95% confidence interval). Mean LIP values not significantly different from 1 indicate a random distribution, whereas clustering was indicated by values significantly greater than 1. For cv. Cox's Orange Pippin, LIP was not calculated for the first assessment dates in both years, because there were very few diseased fruits present.

Spatial autocorrelation analysis was used to determine the extent of clustering of trees with diseased fruits within the field (Cliff and Ord, 1981). The number of diseased fruits per tree was compared to the values in the proximal trees. Only within-row comparisons were made. First-order spatial correlations were determined by relating the number of diseased fruits in each tree to that in the adjacent trees. Higher lag-order correlations were based on trees separated by one or more trees. In the analysis, the rows were combined for each cultivar, with an adjustment for unequal row length by input of missing values. Within-row comparisons up to 10 lags were made for every assessment date.

The covariance function between the two values of a pair is defined as

$$C(h) = E[x(s) \cdot x(s+h)] - \mu_x^2,$$

where s and $s + h$ are positions along a row, separated by distance h, $x(.)$ is the number of diseased fruits, and E denotes the mathematical expectation. The covariance is calculated as

$$\hat{C}(h) = \frac{1}{N(h)} \sum_{i=1}^{N(h)} x_i x_{i+h} - m_{-h} m_{+h},$$

where $N(h)$ is the number of pairs (x_i, x_{i+h}), m_{-h} is the mean of the tail values (first attribute value of each pair, x_i), and m_{+h} is the mean of the head values (second

attribute value of each pair, x_{i+h}). The correlation function is defined as the covariance standardised by the respective tail and head standard deviations

$$\rho(h) = \frac{C(h)}{\sigma_{-h}\sigma_{+h}},$$

where σ_{-h} and σ_{+h} are the standard deviations of the tail and head values, respectively (Deutsch and Journel, 1998). For $\rho(h) > 0$, a positive spatial autocorrelation exists, indicating similar numbers of diseased fruits in nearby trees. Negative coefficients indicate dissimilar values. The correlation function is estimated by dividing $\hat{C}(h)$ by estimated $\hat{\sigma}_{-h}$ and $\hat{\sigma}_{+h}$ values, respectively. Calculations were made with the geostatistical program GSLIB (Deutsch and Journel, 1998). The significance of the obtained correlation coefficients ($\rho(h) \neq 0$) was tested ($P = 0.05$) against the hypothesis of no autocorrelation ($\rho(h) = 0$).

Results

The increase in disease incidence during both growing seasons is shown in Figure 1. In cv. James Grieve, the rate of increase was approximately constant throughout the season, although there was a slight increase in the last week before harvest in 1997 (Figure 1). In 1998 disease incidence in cv. James Grieve amounted already to 1.7% on the first assessment date. Initially, in cv. Cox's Orange Pippin a slow, constant increase in disease incidence occurred (0.050% per day in 1997 and 0.015% in 1998). Three weeks before harvest time, which coincided in both years with the harvest of cv. James Grieve, a sudden increase in the rate of increase of disease incidence was observed (Figure 1). In both years, final pre-harvest disease incidence was equal to 4.2–4.3% for cv. James Grieve; for cv. Cox's Orange Pippin this was 4.4% in 1997 and 2.7% in 1998. In the post-harvest phase, disease incidence was 1.3% in cv. James Grieve, and 1.7% in cv. Cox's Orange Pippin in 1997. Means for both cultivars were not significantly different (t-test, $P = 0.05$). In 1998, post-harvest disease incidence was 2.0% in cv. James Grieve, and 1.2% in cv. Cox's Orange Pippin, being an insignificant difference.

Mean values of LIP with time are shown in Figure 2. For cv. James Grieve, diseased fruits were clearly clustered (aggregated) in 1998. At every assessment date, the LIP value was significantly greater than 1 ($P = 0.01$). Some trees had a considerable number of diseased fruits whilst others had none (Figure 3). At the first assessment date in 1998 the maximum number of diseased fruits per tree was 12, whilst half of the James Grieve trees were without any diseased fruit. In 1997, clustering of diseased fruits was less pronounced in cv. James Grieve, though significant ($P = 0.05$). A similar pattern was observed in cv. Cox's Orange Pippin, clustering of fruits tended to be more pronounced in 1998 than in 1997 (Figure 2). Initially, in 1998 high mean values of LIP occurred (2.4–4.4) due to a few trees bearing 2–3 diseased fruits, whilst most of the trees had none. Those LIP values, however, were not significantly different from 1 ($P = 0.05$).

Clustering of trees with diseased fruits was analysed by within-row autocorrelation. In 1997 no significant autocorrelations were found for either cultivar (data not shown), but in 1998 significant autocorrelation was detected in cv. James Grieve as well as cv. Cox's Orange Pippin. Significant, positive correlation coefficients for 2nd, 3rd and 4th lag-order distances were found in cv. James Grieve ($P = 0.05$). The patterns for each of four consecutive assessment dates turned out to be similar (Figure 4). Correlation coefficients ranged from 0.2 to 0.3 for nearby trees (2nd to 4th lag order), and approached zero or became negative at lag orders 8 up to 10. Typically, no significant positive correlation was found for the adjacent quadrat (first-order, $P = 0.05$). In cv. Cox's Orange Pippin, significant first-order autocorrelation occurred in 1998 at the moment when the total number of diseased fruits started to increase considerably (Figure 5, $P = 0.05$). Significant higher lag-order correlations were not found in cv. Cox's Orange Pippin.

Discussion

Our results support the hypothesis that disease incidence in apple caused by *M. fructigena* increases gradually up to harvest time. In cv. James Grieve, we observed no marked increase around harvest time, as often reported for *M. fructicola* (Hutton and Leigh, 1956; Zehr, 1982; Northover and Cerkauskas, 1994). However, in cv. Cox's Orange Pippin a distinct increase in disease incidence occurred in the last 3 weeks before harvest maturity (Figure 1). This increase occurred in both years after cv. James Grieve was harvested. Disease incidence in the late cultivar increased as soon as the early cultivar had been harvested. The latent period is only 3–4 days at temperatures occurring in summertime; at the next assessment date after cv. James Grieve

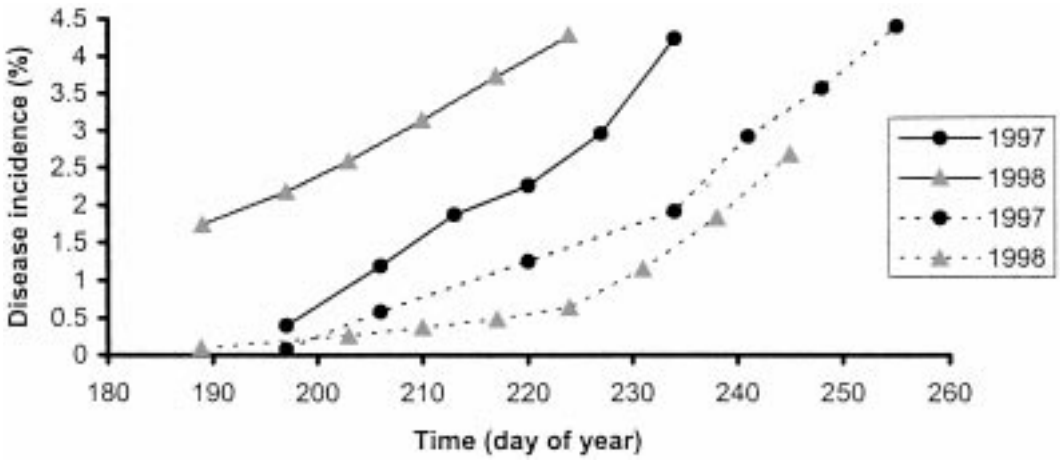

Figure 1. The (cumulative) percentage of apples showing sporulation of *M. fructigena* in the field during two consecutive seasons in cv. James Grieve (drawn line) and cv. Cox's Orange Pippin (dotted line).

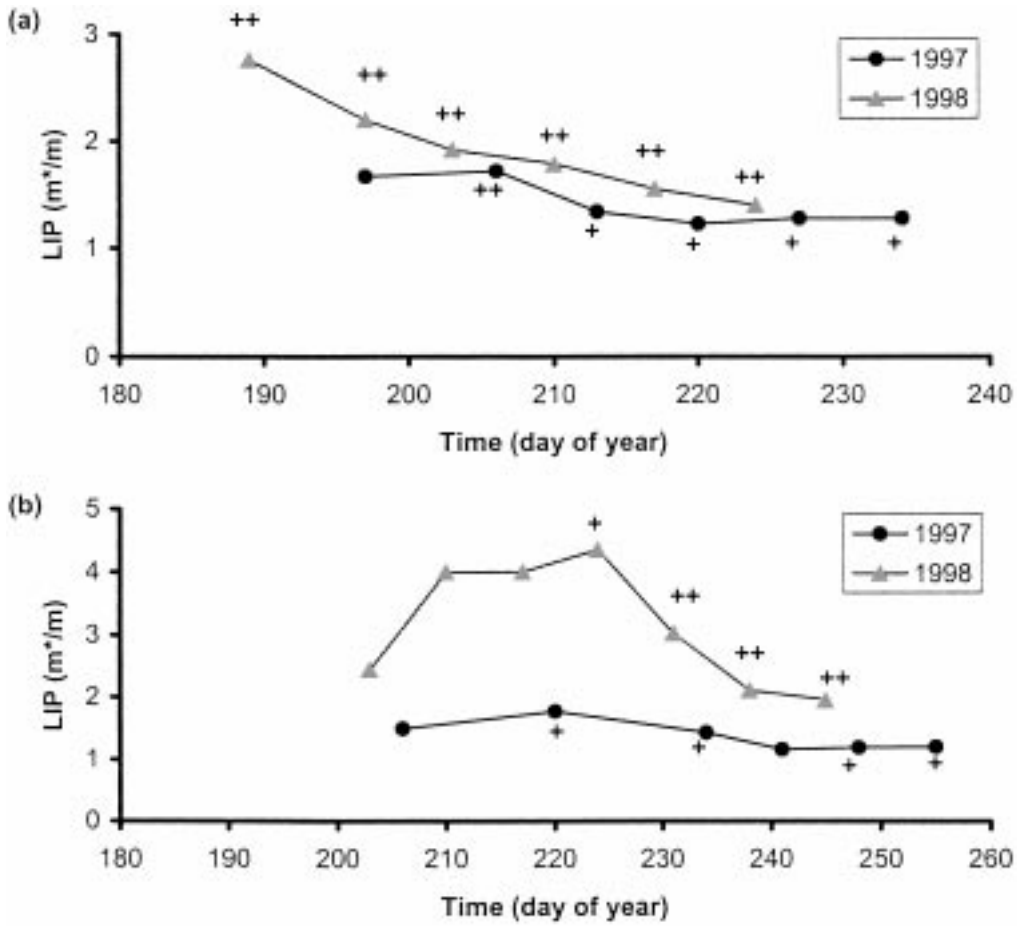

Figure 2. LIP during the season in (a) cv. James Grieve and (b) cv. Cox's Orange Pippin (+ = LIP value significantly different from 1, $P = 0.05$; ++ = idem, $P = 0.01$).

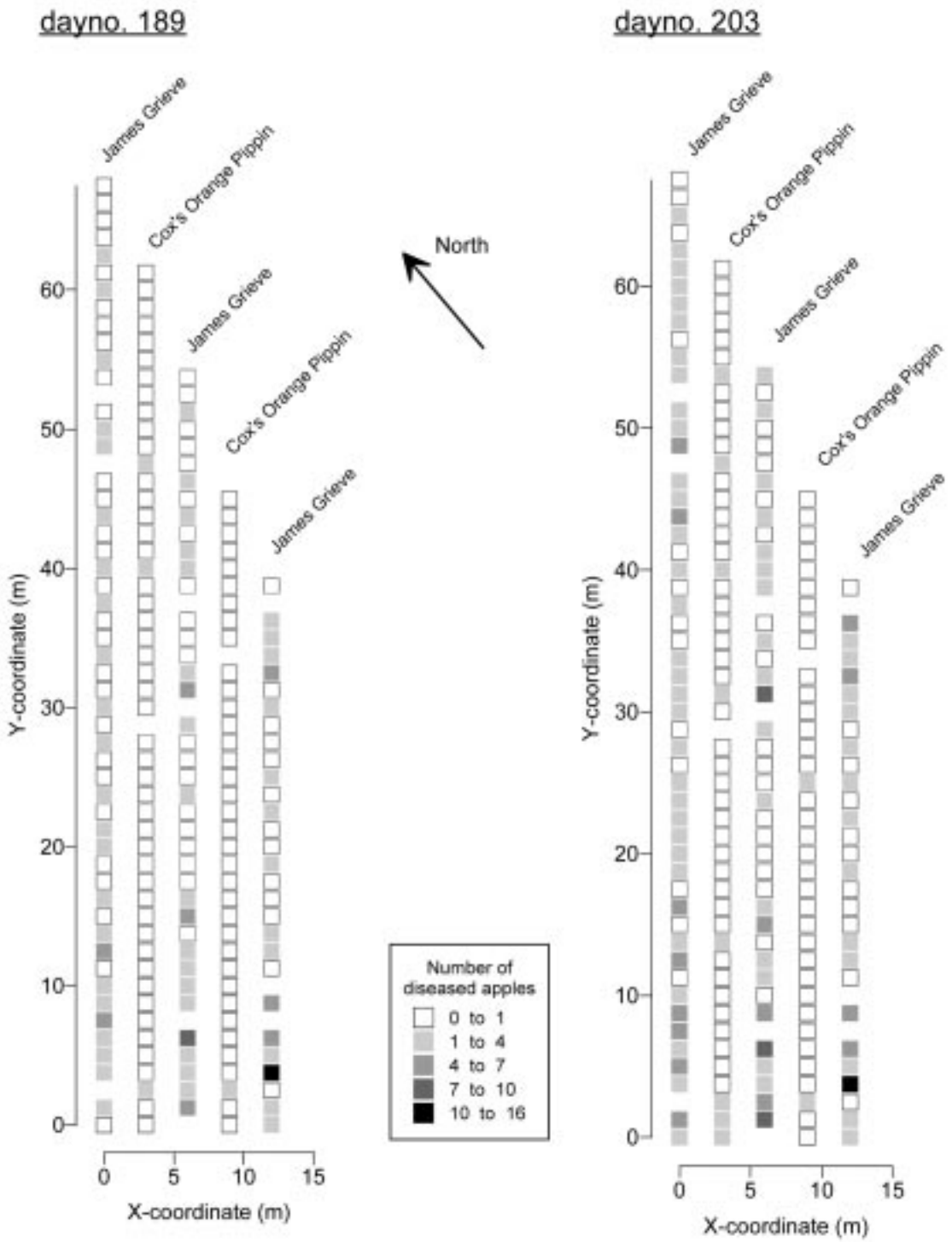

Figure 3. Spatial map of the number of *Monilinia*-diseased fruits per tree at two different assessment dates in 1998. Number of diseased fruits grouped only for presentation not for analysis.

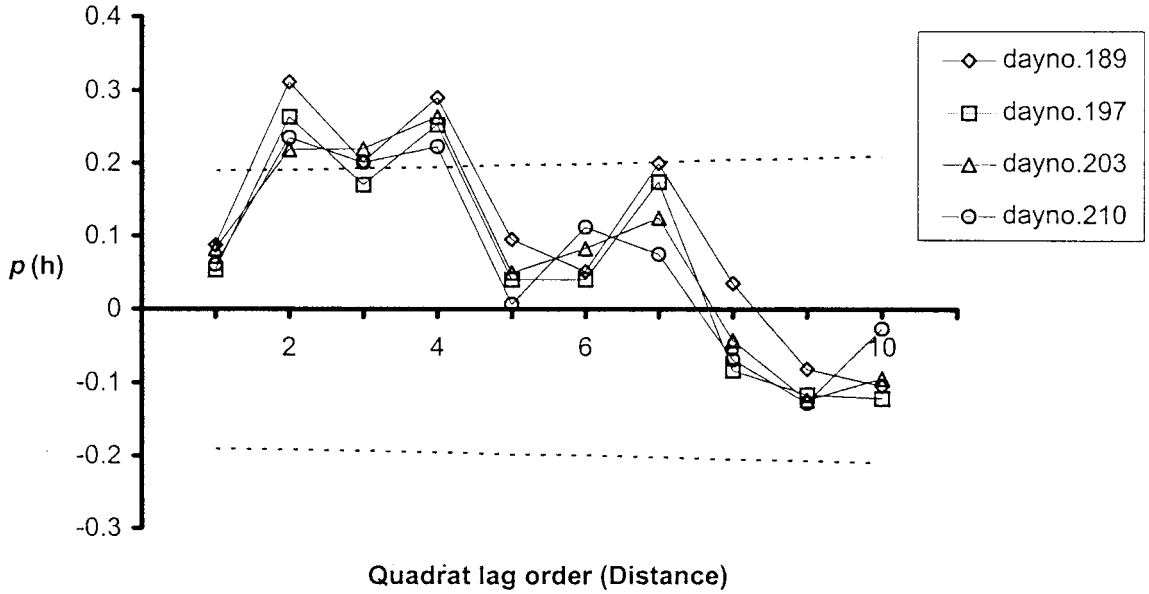

Figure 4. Spatial autocorrelation for 1–10 lags distance at four consecutive assessment dates in cv. James Grieve in 1998. Dotted lines represent critical values of the Pearson product–moment correlation coefficient, $P = 0.05$; $\rho(h)$ values exceeding these lines are significantly different from 0.

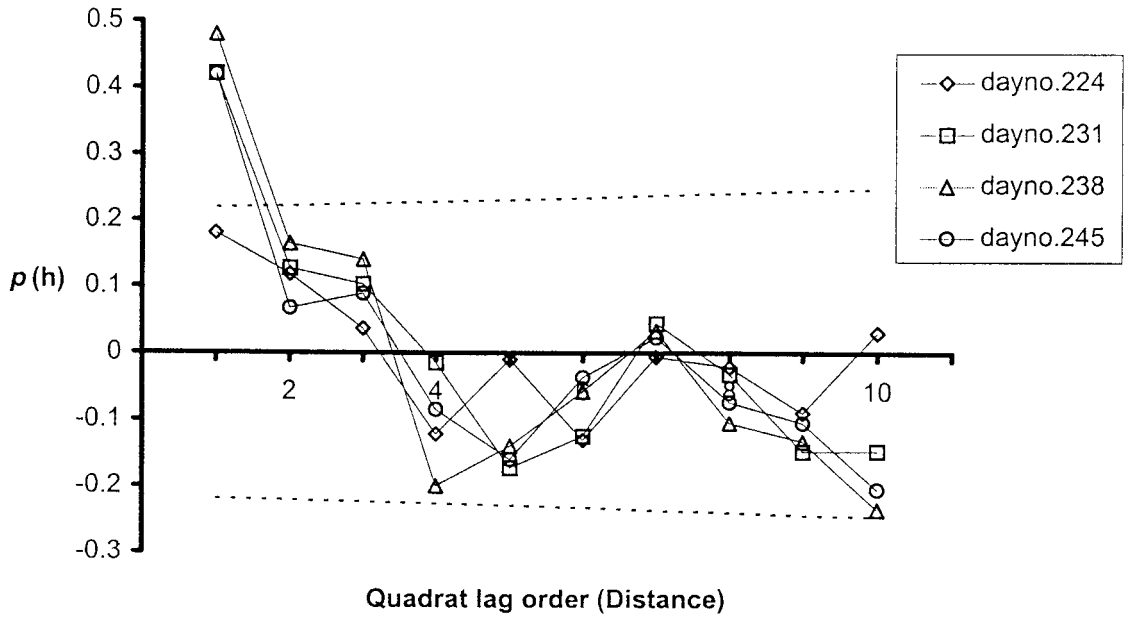

Figure 5. Spatial autocorrelation for 1–10 lags distance at four consecutive assessment dates in cv. Cox's Orange Pippin in 1998. Dotted lines represent critical values of the Pearson product–moment correlation coefficient, $P = 0.05$; $\rho(h)$ values exceeding these lines are significantly different from 0.

was harvested (7 days later), a distinct increase in disease incidence was observed in cv. Cox's Orange Pippin. Most probably, in cv. Cox's Orange Pippin, a build-up of inoculum on the fruits could have taken place during the weeks that cv. James Grieve was ripening, as reported for early and late maturing cultivars in stone fruits (Kable, 1971). The build-up of inoculum in cv. Cox's Orange Pippin may have taken place in the period before harvest of James Grieve, as the aerial spore content dropped sharply after harvest of James Grieve (G.C.M. van Leeuwen, unpublished). The increase in disease incidence in cv. Cox's Orange Pippin cannot be explained by increased susceptibility of ripening fruits. In a laboratory test, ripe Cox's Orange Pippin apples without injuries did not become infected when sprayed with a highly concentrated conidial suspension of *M. fructigena*, and incubated at 16–18 °C at 90–100% RH for 7–10 days (G.C.M. van Leeuwen, unpublished). It is possible that wounding agents (insects, birds), formerly aggregating in cv. James Grieve, shifted to cv. Cox's Orange Pippin after cv. James Grieve was harvested. For birds, it has been shown that they have a preference for early maturing apple cultivars (Mitterling, 1965; Tobin et al., 1989), such as James Grieve.

Final pre-harvest yield losses in both apple cultivars ranged from 2.7% to 4.4% over both years. Over three seasons, Moore (1950) recorded about 9% incidence of brown rot of apples in trees under a complete spray schedule against apple scab, sawfly and codling moth. Probably, the pesticides used in those days were not as effective as current pesticides and alternative control measures. For example, codling moth damage amounted in one year as much as 20% in the complete spray treatment in spite of a lead arsenate spraying in June (Moore, 1950). Pre-harvest losses in stone fruits caused by *M. fructicola* are on average higher compared with those caused by *M. fructigena* in pome fruits. Seasons with high yield losses, amounting up to 30–40%, alternate with those of low to moderate losses of 5–15% (Morschel, 1956; Kable, 1969; Hong et al., 1997). Post-harvest losses observed in both cultivars exceeded those reported by Berrie (1989) during the storage season in cv. Cox's Orange Pippin and cv. Bramley's seedling. However, in the latter case fruits had received a post-harvest fungicide treatment and were stored under controlled atmosphere conditions, before disease incidence was assessed.

In 1998, the spatial distribution of diseased fruits among the trees was clearly clustered, indicated by highly significant LIP values ($P = 0.01$). In time, the mean LIP values decreased as diseased fruits started to appear in trees previously devoid of diseased fruits. The initially high LIP values, especially in cv. Cox's Orange Pippin in 1998 (Figure 2), resulted from the accumulation of diseased fruits within trees where the first diseased fruits were recorded, whilst the majority of trees harboured no diseased fruits at that time. For the brown rot fungi, fruit-to-fruit contact is one of the main mechanisms for spread of disease within a tree, and for which injuries are not necessary (Michailides and Morgan, 1997). Splash dispersal of conidia is one of the other mechanisms for spread of disease within a tree. Pauvert et al. (1969) demonstrated that conidia of *M. fructigena* are easily splash dispersed over short distances; this process facilitates spread of propagules within a tree, though final infection depends on the presence of injuries in this case. LIP values similar to our results, were calculated by Elmer et al. (1998) for *M. fructicola*-diseased fruits in peach and nectarine orchards at harvest time, but the authors did not determine confidence intervals. In our study, mean LIP values gradually decreased towards harvest time for both cultivars, but remained statistically different from 1 (Figure 2).

Clustering of fruits was more apparent in 1998, and so was clustering of trees with diseased fruits. Significant autocorrelations ($P = 0.05$) were found in both cultivars in the second year. For cv. James Grieve, autocorrelation occurred at the 2nd- to 4th-order lag whereas a first-order correlation was absent (Figure 4). The fact that first-order correlations were not significant, rules out the importance of inoculum concentration *per se* in the immediate environment to cause infections. Thus, the observed spatial distribution may depend more on behavioural characteristics of wounding agents than on inoculum concentration in the environment; this is supported by observations in cv. Cox's Orange Pippin, where we observed a distinct increase in the rate of increase of disease incidence after cv. James Grieve was harvested (see above). Remarkably, in 1998 (but not 1997) the intensified increase in disease incidence was accompanied by a significant first-order autocorrelation in cv. Cox's Orange Pippin (Figure 5). Clustering of trees with diseased fruits became apparent, though limited to the direct neighbour trees. Elmer et al. (1998) also found significant correlations in only one out of two seasons in a similar study with *M. fructicola*-diseased fruits.

Based on the results of our study, we have indications that the control of wounding agents during fruit development is an essential part of disease management

of brown rot in pome fruits caused by *M. fructigena*. Although we did not examine closely which wounding agents were responsible for injuries, blackbirds (*Turdus merula*) were often observed pecking at fruits. Bird-scaring methods are an option to limit bird damage (Hasey and Salmon, 1993), but these will also interfere with the activity of predatory birds (e.g. *Parus major*) that feed on noxious Lepidoptera species (Kristin and Patocka, 1997). An interesting approach to limit damage caused by American robins (*Turdus migratorius*) in small fruits, such as blueberries and cherries, was reported by Brugger and Nelms (1991), who suggested that modification of the relative sugar content in fruits might raise an aversion to the food. Some fruit-eating bird pests lack the enzyme sucrase which is necessary to digest the disaccharide sucrose. Direct control of wounding agents in pome fruits would certainly decrease disease incidence caused by *M. fructigena*. Moreover, less easily controllable abiotic factors, such as rain around harvest time, do not cause injuries to pome fruits to the same extent as experienced in stone fruits.

In conclusion, yield loss caused by *M. fructigena* did not exceed 5% in the pre-harvest stage, and losses in the post-harvest stage were comparable with those reported in the literature. In time, disease incidence gradually increased in the orchard, and no sharp increase was observed around harvest maturity, though rainy, damp weather conditions prevailed during harvest in 1998. In this, disease dynamics of *M. fructigena* in apple differ sharply from that of *M. fructicola* in stone fruits. A statistical analysis in space and time, using the LIP index, to which we added bootstrap confidence intervals, proved to be useful to distinguish differences between cultivars and between years. Distinct clustering of diseased fruits among fruit trees occurred in both years, but was more pronounced in 1998. Cluster size did not extend beyond tree boundaries, although first-order autocorrelation was detected in cv. Cox's Orange Pippin in the second year. Clearly, spread of disease within a tree occured more readily with *M. fructigena*, than spread of disease to adjacent trees.

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