Analyses of temporal dynamics of brown rot development on fruit in organic apple production

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Summary: In a two-year study, yield loss and temporal dynamics of brown rot development caused by Monilinia fructigena (Aderh. & Ruhl.) Honey were quantified and analysed in two organic apple orchards (Debrecen–Pallag and Debrecen–Józsa). The first infected fruits were observed at the beginning of August in both years and both locations, except for one occasion when the first infected fruit was found at the end of July. Temporal disease development was continuous up to harvest time in both years and locations. In the two years, pre-harvest yield loss on the trees amounted between 8.9% and 9.3% at Debrecen-Pallag and between 9.7% and 10.8% at Debrecen–Józsa by fruit harvest. Incidence of infected fruits on the orchard floor ranged from 32.4% to 43.2% and from 53.3% to 61.9%, at Debrecen–Pallag and Debrecen–Józsa, respectively, by fruit harvest. Analyses of temporal disease progress showed that the best-fitted mathematical function was the power function in both orchards and years. Both parameters of the power function clearly demonstrated that incidence of brown rot on fruit increased faster on the orchard floor than on the tree. Moreover, the disease increase was faster at Debrecen–Józsa in most cases than at Debrecen–Pallag. Our results indicated that the strategy of disease management, the ripeness of the fruit and the presence of a wounding agent played an important role in the yield loss and in the temporal development of fruit disease incidence caused by M. fructigena in organic apple orchards. Biological and practical implications of the results are discussed.

Key words: temporal dynamics of brown rot, Monilinia fructigena, power function, curve fitting, epidemiology, organic apple production

Introduction

Brown rot fungi of fruit crops consist of three old species: *Monilinia fructigena* (Aderh. & Ruhl.) Honey, *Monilinia laxa* (Aderh. & Ruhl.) and *Monilinia fructicola* (Wint.) Honey; the latter species is a quarantine organism in Europe. Although, all the three species can infect apple, *M. fructigena* is the most common on pome fruits and the other two species attack mainly stone fruits. Most studies presented that fruit infection by *Monilinia* spp. can happen during fruit swelling and fruit ripening but can also cause post-harvest losses (*Moore*, 1950; *Berrie*, 1989; *Falconi & Mendegen*, 1994; *Van Leeuwen* et al., 2000; *Xu & Robinson*, 2000). As the most common *Monilinia* species on apple fruit is *M. fructigena*, this study was focused on this fungus.

Losses caused by *M. fructigena* are usually low in most conventional and integrated apple orchards. One report in the early 1950's presented that an average of 9% of the apple fruits become infected during the growing season in conventional apple orchards (*Moore*, 1950). Later workers demonstrated that the level of fruit infection under field conditions is dependent upon cultivar susceptibility (*Cimanowski & Pietrzak*, 1991) and the presence of wounds in the fruit skin caused by wounding agents, such as codling moth (*Cydia pomonella* Linneus), common earwig (*Forficula auricularia* Linneus), or blackbirds (*Tordus merula* Linneus) (*Croxall* et al., 1951; *Lack*, 1989; Van

Leeuwen et al., 2000; Xu & Robinson, 2000). Recently, one study demonstrated that pre-harvest disease incidence was low (ranged from 2.7% to 4.4%) on susceptible apple cultivars 'James Grieve' and 'Cox's Orange Pippin' in an integrated apple orchard (Van Leeuwen et al., 2000). However, yield loss caused by M. fructigena has never been reported from organic apple orchards. Recently, temporal aspects of increase in M. fructigena diseased fruits in apple orchards were investigated in two studies. One was made in an integrated orchard in the Netherlands (Van Leeuwen et al., 2000) and the other in integrated pear and apple orchards in the United Kingdom (Xu et al., 2001). Both studies demonstrated that disease incidence of brown rot increased gradually up to harvest. However, no attempt has been made to determine the temporal development of brown rot epidemics in organic apple orchards.

The objective of this study was to quantify the preharvest yield losses and analyse the temporal dynamics of disease development caused by *M. fructigena* in organic apple orchards.

Material and method

Orchard site and plant material

The study was made in two experimental orchards. Both orchards were situated in the eastern part of Hungary, one at

Debrecen-Pallag and the other at Debrecen-Józsa. The two orchards were treated by approved fungicides (copper and wettable sulphur) and insecticides (e.g. Bt products) according to the Hungarian organic production guidelines (Anonymous, 1997). Trees were grafted on M26 rootstock, planted at a spacing of 4×1.5 m and pruned to a spindle shape in both orchards. The 0.5 ha orchard at Debrecen-Pallag was established in 1997 and the 12.5 ha orchard at Debrecen-Józsa was set up in 1998. In both orchards several cultivars were grown. The selected cultivar for disease assessments was cv. 'Elstar' in both orchards.

Disease assessment

Disease incidence on fruits caused by *M. fructigena* was assessed from 15 May until 30 September in 2002 and 2003 in both orchards. Assessments were made on a biweekly basis in both years. Disease incidence was assessed in four replicates. Fourteen trees were selected for each replicate and the brown rot incidence was assessed on all fruits of a selected tree. At each assessment date healthy and diseased fruits were counted on the trees as well as on the orchard floor separately. Disease incidence on fruit caused by *M. fructigena* was calculated as the number of diseased fruits divided by the selected number of fruits, separately for fruits on the trees and on the orchard floor, for each assessment date.

Statistical analysis

Collected data were averaged for each assessment date and for each orchard. First, all data were subjected to analyses of variance (ANOVA) in order to test the effect of year, location and position of a fruit (on the tree or on the orchard floor) on yield loss at harvest time. Before the analyses, data were transformed to angular to correct normality. Significant F-tests (P<0.05) were performed for pairwise comparison of the disease development means of the two years and two locations. Then, for each orchard, the nonzero values of mean disease incidence on fruit were plotted against time after the first assessment date, separately for fruits on the trees and on the orchard floor.

Then, disease progress curves were fitted to the data points. Linear, exponential, power and logistic functions were tested for goodness-of-fit to the disease progress data. We aimed to select one function which gave the best fit, overall, to the data of disease progress on fruits caused by *M. fructigena*.

Results

Disease progress in general

Brown rot incidence on fruit was zero until the beginning of August in both years and both locations, except for Debrecen–Józsa in 2002 when the first infected fruit was found on 24 July (Figures 1–4). In both years, disease incidence was higher at Debrecen–Józsa than at Debrecen–Pallag at each assessment date. Moreover, the level of brown rot incidence was generally higher on the orchard floor than on the tree in both locations and both

years. Disease progress continuously increased from the beginning of August up to harvest in both orchards. In the two years, the incidence of infected fruits on the tree was between 8.9% and 9.3% at Debrecen–Pallag and between 9.7% and 10.8% at Debrecen–Józsa (Figures 1 and 3), by fruit harvest. Incidences of infected fruits on the orchard floor ranged from 32.4% to 43.2% and from 53.3% to 61.9%, at Debrecen–Pallag and Debrecen–Józsa, respectively, by fruit harvest (Figures 2 and 4). Analyses of variance showed that the effect of year was not significant on yield loss at harvest time but that of the position of a fruit (the presence of an infected fruit on the tree or on the orchard floor) and location were significant at P=0.001 and P=0.046, respectively.

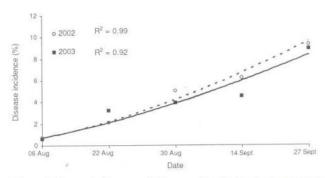


Figure I Disease incidence on fruits caused by M. fructigena on the tree, in an organic apple orchard on cv. 'Elstar' (Debrecen-Pallag, Hungary, 2002 /drawn line/ and 2003 /dotted line/).

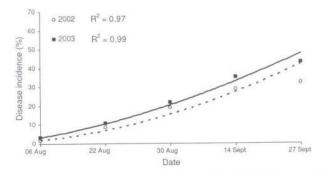


Figure 2 Disease incidence on fruits caused by M. fructigena on the orchard floor, in an organic apple orchard on cv. 'Elstar' (Debrecen–Pallag, Hungary, 2002 /drawn line/ and 2003 /dotted line/).

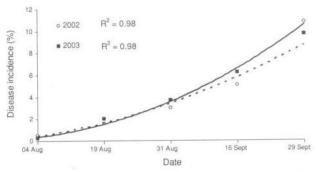


Figure 3 Disease incidence on fruits caused by M. fructigena on the tree, in an organic apple orchard on cv. 'Elstar' (Debrecen–Józsa, Hungary, 2002 /drawn line/ and 2003 /dotted line/).

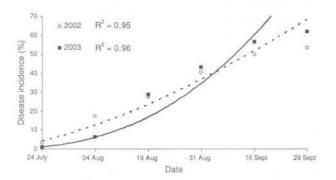


Figure 4 Disease incidence on fruits caused by M. fructigena on the orchard floor, in an organic apple orchard on cv. 'Elstar' (Debrecen–Józsa, Hungary, 2002 /drawn line/ and 2003 /dotted line/).

Analyses of disease progress curves

Analyses of disease progress curves showed that the best-fitted mathematical function was the power function in both orchards and both years (Table 1). When fruits were assessed on the orchard floor at Debrecen-Józsa in 2003, the linear function gave a better fit $(R^2 = 0.97)$ than the power function ($R^2 = 0.96$). However, fulfilling our aim to select one function which gave the best fit, overall, to the data of disease progress, the power function was selected. The coefficient of determination was above 0.95 for all mathematical functions, except for when fruits were assessed on the tree at Debrecen-Pallag in 2003 ($R^2 = 0.92$) (Table 1). Both parameters of the power function clearly demonstrated that incidence of brown rot on fruit increased faster on the orchard floor than on the tree. Moreover, the disease increase was faster at Debrecen-Józsa in most cases than at Debrecen-Pallag.

Discussion

This study demonstrated that a significant yield loss caused by *M. fructigena* was detected in two organic apple

orchards in 2002 and 2003 at Debrecen-Pallag and Debrecen-Józsa in Hungary. Disease development started at the beginning of August and it increased continuously up to harvest. This result is in agreement with the hypothesis of earlier studies (Moore, 1950; Van Leeuwen et al., 2000). In this study the first infection occurred 9-11 weeks before harvest on cv. 'Elstar', however, in the study of Van Leeuwen et al. (2000) it happened 4-5 weeks and 7-8 weeks before harvest maturity for cv. 'James Grieve' and cv. 'Cox's Orange Pippin', respectively. In our study, orchards were only treated with sulphur and copper fungicides according to organic guidelines, while the orchard in the study of Van Leeuwen et al. (2000) was treated regularly against apple scab with contact and systemic fungicides approved in integrated apple production. Sulphur and copper fungicides are less effective against brown rot than modern contact and systemic fungicides used against apple scab. Most probably this was one of the reasons why the first infected fruits occurred earlier in our organic orchards than in an integrated orchard in the Netherlands, even if the weather conditions for brown rot infection are more favourable in the Netherlands than in Hungary. Moreover, based on the results of our study and others (Croxall et al., 1951; Lack, 1989; Van Leeuwen et al., 2000; and Xu & Robinson, 2000), we have indications that wounding agents also play an important role in the increase of Monilinia infected fruits. Codling moth (Cydia pomonella Linneus), common earwig (Forficula auricularia Linneus) and birds (such as blackbirds, Tordus merula Linneus) are considered to be the most common wounding agents in apple orchards (Croxall et al., 1951; Lack, 1989; Van Leeuwen et al., 2000; and Xu & Robinson, 2000). In the study of Moore (1950) the codling moth damage was as much as 20% and the brown rot damage was an average of 9% by harvest time. In the study of Van Leeuwen et al. (2000) the peak of the brown rot damage on fruit was 4.4% and the codling moth damaged less than 5% of the fruits in the two experimental years (I.J. Holb & G.C.M. Van Leeuwen, unpublished). In our organic orchards, brown rot incidence by harvest time reached

Table I The best-fitted mathematical function and its characteristics in the analyses of temporal dynamics of brown rot fungi on fruit in two Hungarian organic apple orchards in 2002 and 2003.

Location	Year	Positiona	Function ^b	R ^{2 c}	parameter 'a'	SE^d	parameter 'b'	SE^e
Debrecen-Pallag	2002	on the tree	power (axb)	0.99	0.71	0.04	1.61	0.11
	2002	on the orchard floor	power (axb)	0.97	1.79	0.15	1.96	0.22
	2003	on the tree	power (axb)	0.92	0.73	0.09	1.51	0.13
	2003	on the orchard floor	power (axb)	0.99	3.32	0.11	1.65	0.08
Debrecen-Józsa	2002	on the tree	power (axb)	0.98	0.46	0.02	1.82	0.09
	2002	on the orchard floor	power (axb)	0.95	4.44	0.22	1,52	0.16
	2003	on the tree	power (axb)	0.98	0.35	0.01	2.11	0.09
	2003	on the orchard floor	power (axb)	0.96	1.14	0.17	2.46	0.28

a Disease incidence on fruit caused by M. fructigena was assessed separately for fruits on the trees and on the orchard floor.

b The name and the general equation of the best-fitted mathematical function tested for goodness-of-fit to the disease progress data.

^c Coefficient of determination.

d Standard error for parameter 'a'.

e Standard error for parameter 'b'.

30–60% on the orchard floor depending on locations and years. The incidence of codling moth was extremely high in these organic orchards, it ranged from 40 to 70% in the two experimental years (*I.J. Holb*, unpublished). Moreover, in most cases, 80–90% of the *Monilinia* infected fruits were also damaged by codling moth (*I.J. Holb*, unpublished). Therefore, probably, the most important factor in the severe *Monilinia* infection was the wounding of the fruit skin caused by codling moth.

The analyses of the temporal dynamics of brown rot disease development showed that the disease progress curves could be characterised by the power function in all cases. Recently, the temporal analysis of brown rot development was made in the study of Van Leeuwen et al., (2000) in the Netherlands and of Xu et al. (2001) in the United Kingdom. However, in their studies the disease incidence was low (below 5% and 11% in the Netherlands and in the United Kingdom, respectively), therefore, mathematical functions were not fitted and the temporal dynamics of disease progress curves was not analysed. Instead, the Lloyd's index of patchiness (LIP) was calculated (Van Leeuwen et al., 2000) in order to show whether the diseased fruits were clustered (aggregated) or not. The study clearly showed that most diseased fruits were clearly clustered on susceptible apple cultivar 'James Grieve'. In the study of Xu et al. (2001), significant aggregation of diseased fruits among trees was also detected for assessment dates when the overall incidence of disease was greater than 5%. Although we did not examine closely the clustering of diseased fruits, clusters of two to four diseased fruits were often observed on the trees in both orchards at harvest time. These cluster fruits were severely damaged by codling moth or other wounding agents and these fruits were important bases for mummification of the fruits (1.J. Holb, unpublished). However, we also observed that most diseased fruits dropped on the orchard floor, especially if they were damaged by codling moth. Therefore, the proportion of damaged fruits was higher and the increase of disease development was faster on the orchard floor than on the tree (Table 1).

In sum, it can be stated that the strategy of disease management, the ripeness of a fruit and the presence of a wounding agent, primarily *C. pomonella*, play an important role in the yield loss and in the temporal development of fruit disease incidence caused by *M. fructigena* in organic apple orchards.

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