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Temporal development and relationship amongst brown rot blossom blight, fruit blight and fruit rot in integrated and organic sour cherry orchards

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The aim of this 4-year study was to characterize temporal development of brown rot blossom blight and fruit blight (caused by *Monilinia* spp.) and their sporulating areas in sour cherry orchards; and to determine the relationships amongst incidence and sporulating area of blossom blight, fruit blight and fruit rot. The study was performed in integrated and organic orchard blocks on two cultivars (Újfehértói fürtös and Érdi bőtermő). On both cultivars, disease progress on flowers and fruits was 2–10 times slower in the integrated than in the organic management system. The peak incidence values were 9 and 31 days after petal fall for blossom blight and fruit blight, respectively. After these dates, no new blight symptoms on flowers and/or fruits appeared and the disease was levelling off. Final blossom blight incidence ranged from 1 to 5% and from 12 to 34%, and fruit rot incidence from 2 to 6% and from 11 to 26% in the integrated and the organic orchards, respectively. The sum of fruit blight incidence ranged from 9 to 22% for the organic system, but was below 5% for the integrated system, while the final sporulating area was 5–16 mm^2 and <3 mm^2, respectively. Among the five highest Pearson's correlation coefficients, relationships between blossom blight and early fruit blight stage (r = 0·845, P = 0·0087 integrated; r = 0·901, P = 0·0015 organic), and between sporulating area and fruit rot (r = 0791, P = 0·0199 integrated; r = 0·874, P = 0·0039 organic) were the most significant relationships from an epidemic standpoint as they indicated a connection between different brown rot symptom types.

Keywords: epidemiology, *Monilinia laxa*, *Monilinia* spp., *Prunus vulgaris*, sporulating area

Introduction

Brown rot, caused by *Monilinia* spp., is a devastating disease of sour cherry (*Prunus vulgaris*) all over the world (Batra, 1991; Ogawa *et al.*, 1995). Among *Monilinia* spp., *M. laxa* is the most prevalent organism causing severe blossom and twig blight of sour cherry in Europe. In spring, the fungus can spread quickly from the blighted blossom to the shoot stem and then to the leaves (Byrde & Willetts, 1977; Stensvand *et al.*, 2001; Holb & Schnabel, 2005; Gell *et al.*, 2007; Holb *et al.*, 2008; Everhart *et al.*, 2011). Under favourable conditions, flowers, leaves and shoots can die rapidly and the fungus can kill larger twigs under high disease pressure. In the fruit swelling stage, *Monilinia* spp. can also infect young fruits and cause green fruit rot (Byrde & Willetts, 1977; Biggs & Northover, 1988a,b; Holb, 2003) and at ripening stage the typical brown rot in fruits. In both fruit rot types, infected fruit tissues become brownish and mycelia in these infected tissues begin to sporulate and produce mass conidial inoculum on the surface of the infected fruit tissues (Tamm & Flückinger, 1993; Tian & Bertolini, 1999; Stensvand *et al.*, 2001; Fourie & Holz, 2003; Xu *et al.*, 2007; Holb, 2008; Gibert *et al.*, 2009). Fruit infections may lead to a disease epidemic by harvest and to mummified fruits (Byrde & Willetts, 1977; Batra, 1991).

Under favourable conditions, not only flowers and twigs but also fruit can produce blighted symptoms similar to blossom and twig blight (I. J. Holb, unpublished data). Fruit blight occurs on green fruits with sizes <10 mm under Hungarian environmental conditions. Blighted fruits do not rot as with green fruit rot, but die suddenly with their penducle attached to the shoot and become firm, like blighted flowers or leaves (Fig. 1). Fruit blight can occur in two ways: as a result of blossom and/or twig blight proximal to the fruit (Fig. 1a), or at certain green fruit stages in the absence of blossom or twig blight (Fig. 1b). When the fungus was isolated from blighted fruits and then green fruits were artificially inoculated with these isolates, the reisolation from the artificially inoculated green fruits matched well with the description of *M. laxa* (Byrde & Willetts, 1977). In rainy periods, blighted fruits may also produce sporodochia and conidia on the dead tissues in late spring and during the summer, as occurs on blighted flowers and leaves (Stensvand *et al.*, 2001). However, the basic features of temporal development and/or sporulation patterns of fruit blight symptoms are not known. These may differ under well-managed and poorly managed orchard conditions and on cultivars differing in their susceptibility to brown rot.

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Fruit blight occurs between the periods of blossom blight and harvest fruit rot, and it often appears on the same shoots that have already shown blossom and/or shoot blight previously, but it also occurs separately from these symptoms (I. J. Holb, unpublished data). In addition, as the fungus can sporulate in infected tissues of blighted flowers and fruits, so conidia from these dead tissues may provide inoculum for subsequent fruit rot symptoms by harvest. If these hypotheses hold true, then there may be an infection link between blossom blight and fruit blight, and, in addition, sporulating tissues of blighted fruits may serve as a potential inoculum source for fruit rot by harvest. Temporal assessments of fruit blight coupled with assessments of blossom blight and fruit rot symptoms, as well as investigation of the relationship amongst these symptom types, may help to understand the epidemiological role of fruit blight in the brown rot life cycle.

The aim of this 4-year study was first to characterize the temporal development of brown rot blossom blight, three fruit blight stages (fruit size <1·5 mm, 1·6–5 mm and 5·1–10 mm) and their sporulating capacity in sour cherry orchards; and secondly, to determine the relationship amongst incidence and sporulating area of blossom blight, fruit blight and fruit rot. The study was performed in a well-managed orchard (integrated) and a poorly managed (organic) one on two sour cherry cultivars differing in their susceptibility to brown rot.

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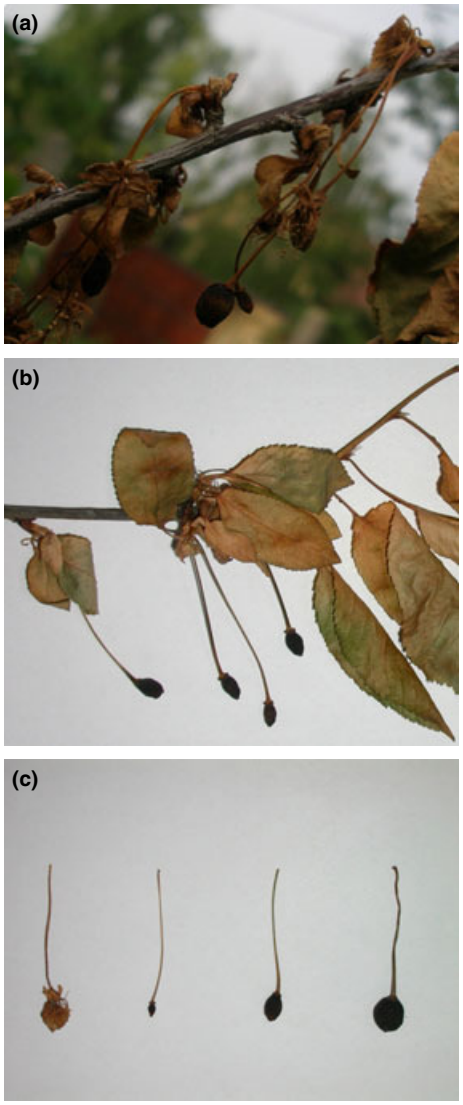

Figure 1 (a) Fruit blight symptoms, with symptoms of blossom blight and twig blight, on sour cherry cv. Érdi bőtermő. (b) Fruit blight symptoms occurring alone on sour cherry cv. Érdi bőtermő. (c) Blossom blight (left) and fruit blight at fruit three fruit size stages (<1·5, 1·6–5 and 5·1–10 mm) on sour cherry cv. Érdi bőtermő.

Materials and methods

Orchard site, general orchard management

A 4-year study (2005, 2006, 2008 and 2009 [2007 was excluded because of severe late spring frost]) was carried out in two sour cherry orchards, one with integrated management and one organic. The integrated orchard was located at 47°31′60″ N and 21°37′60″ E, in Eperjeske, Eastern Hungary. The organic orchard was also in Eperjeske, 0·5 km south of the integrated orchard.

The 6-ha integrated orchard consisted of 20 rows, with distances of 5 m between rows and 3 m between trees within a row. The orchard was planted in 1998 with three self-fertile sour cherry cultivars: Újfehértói fürtös (Balaton), Érdi bőtermő and Debreceni bőtermő. Trees were grown according to the Hungarian IFP (Integrated Fruit Production) guidelines derived from international IFP standards (Cross & Dickler, 1994). The integrated orchard relied on annual application of synthetic fertilizers for nutrient supply.

The 5·8-ha organic orchard consisted of 19 rows with distances of 6 m between rows and 4 m between trees within a row. The orchard was planted in 1997 with three self-fertile sour cherry cultivars: Újfehértói fürtös, Érdi bőtermő and Érdi jubileum. Trees had been grown according to organic production guidelines (Anonymous, 2000). Stable manure and compost were applied every other year.

Both orchards were divided into four blocks each during the experimental periods (2005, 2006, 2008 and 2009) in order to create replications for the two management system. Trees in both orchards were grafted on *Prunus mahaleb* rootstock. The orchard soil type was brown forest soil with alternating layers of clay. Trees were approximately 3·5–4·5 m tall during the 4-year assessment period. Intrarow spacing between branches in the crown of adjacent trees was approximately 0 1–0 5 m and between adjacent rows was approximately 2·0–2·5 m. Bare soil, 0 7 m wide, was maintained in the rows, and grass was grown in the row middles. The orchards were not irrigated. A winter pruning before budbreak was carried out each year. Grass in the row middles was cut with an orchard flail mower four times each year (early June, early July, early August and early September) in the integrated blocks and three times a year (early June, early July and early September) in the organic blocks. Fungicide application schedules in the integrated and organic blocks are listed in Table 1.

Experimental design

The experimental design was a split split-plot with the 4 years as blocks, the two management systems as main plots (replicated

four times) and two sour cherry cultivars as subplots. The management systems were integrated and organic blocks.

Each main plot was split into subplots corresponding to early and late-season cvs Érdi bőtermő and Újfehértói fürtös, respectively. Both cultivars are susceptible to blossom blight and fruit rot, but Érdi bőtermő is considered to be more susceptible to blossom blight than Újfehértói fürtös (Soltész M, 1997; Holb & Schnabel, 2005). Fruit maturity dates for Érdi bőtermő and Újfehértói fürtös range from 13 to 22 June and from 5 to 12 July, respectively (Soltész & M, 1997; Holb & Schnabel, 2005).

Environmental monitoring

Rainfall (mm/day) and mean daily temperature (°C) were recorded from 20 April until 15 July during each of the 4 years using a Metos Compact agrometeorological station (Pessl Instruments GmbH).

Brown rot assessment

Three brown rot symptom types were considered for disease assessment: (i) blossom blight and/or blossom blight incidence coupled with leaf blight, (ii) fruit blight incidence at different fruit size ranges (<1 5 mm, 1·6–5 mm and 5·1–10 mm; Fig. 1c), and (iii) fruit rot incidence. Assessments were performed on the following dates: (i) 2 days after petal fall, (ii) 9 days after petal fall for fruit size of <1·5 mm, (iii) 18 days after petal fall for fruit size of 1·6–5 mm, (iv) 31 days after petal fall for fruit size of 5·1–10 mm, and (v) at harvest (42 and 50 days after petal fall for cvs Újfehértói fürtös and Érdi bőtermő, respectively). Ten trees per cultivar were selected randomly for observation of each brown rot symptom type at each assessment date.

For blossom blight and fruit blight, 100 randomly selected and tagged shoots from each quadrant of a tree were examined for disease symptoms as described previously for blossom blight assessment (Osorio *et al.*, 1994; Holb & Schnabel, 2005). A flower was considered to be diseased when the petals, calyx and at least 1/3 of the pedicel were necrotic (Tamm *et al.*, 1995). A shoot with fruits was considered to be diseased if a blighted fruit was present. Brown rot incidence was calculated as the percentage of flower or fruits blighted.

For fruit rot, 200 randomly selected fruits from each quadrant of a tree were examined for disease symptoms. A fruit was considered to be diseased if at least one visible brown rot lesion was present on a fruit. Brown rot incidence was calculated as the percentage of diseased fruits.

Sporulating area of blighted flowers and fruits

At harvest, all flowers and fruits subjected to blight symptom assessment were also assessed for sporulating area. The largest and smallest diameters of the sporulating area on each blighted flower and fruit were measured with a Vernier caliper, and the mean of these two measurements was used to calculate sporulating area based on the equation for the area of a disc. Sporulating area was expressed as mm^2 sporulating area per blighted flower and/or fruit.

Data analysis

Generally, all disease symptom types were assessed at each assessment date; however, fruit rot was not found at assessment dates (i)–(iv) and no additional blossom and fruit blight symptoms were detected at assessment date (v). Values from the quadrants were averaged to obtain the percentage disease incidence per tree and to produce disease measures including: (i) blossom blight incidence (BB), (ii) fruit blight incidence at fruit size <1·5 mm (FB1), (iii) fruit blight incidence at fruit size of 1·6–5 mm (FB2), (iv) fruit blight incidence at fruit size of 5·1–10 mm (FB3), (v) fruit rot incidence (FR), and (vi) sporulating area of blighted flowers and fruits (SA). In addition, measures FB1, FB2 and FB3 for fruit blight incidence were summarized in a single disease measure (ΣFB). Brown rot disease measures (except for fruit rot at harvest) were plotted over time to obtain progress curves for each year. Final incidence of the seven disease measures was subjected to split-plot analysis of variance (SAS v. 8·1; SAS Institute Inc.). Prior to the analyses, values for brown rot incidence were transformed using the arcsine-square root transformation to stabilize variances.

In order to quantify relationship among blossom blight, fruit blight, fruit rot and sporulating area, Pearson's correlation coefficients were calculated among the seven brown rot measures in all combinations (Table 4). Correlation analyses were done separately for the two management systems using GenStat 5 v. 4·1 (Lawes Agricultural Trust, IACR, Rothamsted, UK). Selected per-variables were then plotted against each other and linear regression functions were fitted in order to investigate the hypothesis that earlier brown rot symptom types can serve as inoculum for later brown rot symptom types. A *t*-test was used to determine whether the regression slopes were significantly different between the two management systems ($\alpha = 0{\cdot}05$).

Results

Environmental monitoring

Daily mean temperature was in the ranges 6·2–24·3, 9·1–26·8, 8·8–22·5 and 7·1–23·1°C in 2005, 2006, 2008 and 2009, respectively, from 20 April to 15 July. Rainfall amounts during the same periods were 175·4, 247·0, 150·7 and 138·4 mm in 2005, 2006, 2008 and 2009, respectively.

Disease progress

Data collected in 2009 were used to illustrate patterns of disease progress typical of the four system–cultivar combinations (Fig. 2). On both cultivars, disease progress on flowers and fruits was 2–10 times slower in the integrated than in the organic management system. Patterns of temporal disease progress within each production system were similar on both cultivars; however, the incidence of brown rot symptom types was generally higher on the early season cv. Érdi bőtermő.

In both production systems, blossom blight started at petal fall (data not shown) and rapidly levelled off 9 days after petal fall on both cultivars (Fig. 2). Fruit blight incidence at fruit sizes of < 1·5 mm (FB1), 1·6–5 mm (FB2) and 5·1–10 mm (FB3) levelled off 9, 18 and 31 days after petal fall, respectively, for both cultivars and systems, while the sum of fruit blight incidence (ΣFB) increased until 31 days after petal fall and then remained the same until harvest. Sporulating area of blighted flowers and fruits (SA) was first observed 9 and

Table 1 Spraying schedules from dormant bud stage to harvest in integrated and organic sour cherry orchards (Eperjeske, Hungary, 2005–2009)

	Dates of phenological stages and fungicide (%) applications											
	Bud swelling	Early tight cluster	Closed blossom	Full bloom	Petal fall	Shuck split	1st Cover	2nd Cover	3rd Cover	4th Cover	5th Cover	6th Cover
Integrated, 2005												
Date	21 March	10 April	24 April	30 April		10 May	21 May	6 June	16 June	22 June		
a.i., Dosage	CoH, 0·1	Ca, 0·2	Ip, 0·1	Pe, 0·05	-	Te, 0·075	Pr, 0·05	Pr, 0·05	Te, 0·075	CoH, 0·1	–	–
Organic, 2005												
Date	22 March	8 April	23 April	29 April	4 May	12 May	20 May	30 May	7 June	17 June	22 June	29 June
a.i., Dosage	Ca-Po, 0·15	CoH, 0·1	Es, 0·75	Es, 0·4	Es, 0·6	Es, 0·6	Es, 0·6	Es, 0·4	Es, 0·6	Es, 0·6	Es, 0·4	Es, 0·4
Integrated, 2006												
Date	22 March	9 April	22 April	26 April		16 May	27 May	12 June	19 June	28 June		
a.i., Dosage	CoH, 0·1	–	Ip, 0·1	Bo+Pi, 0·075	–	Pr, 0·05	Pr, 0·05	CoH, 0·1	Pr, 0·05	Te, 0·075	–	–
Organic, 2006												
Date	23 March	10 April	23 April	26 April	3 May	17 May	25 May	4 June	13 June	17 June	23 June	30 June
a.i., Dosage	Ca-Po, 0·15	CoH, 0·1	Es, 0·75	Es, 0·4	Es, 0·6	Es, 0·6	Es, 0·4	Es, 0·6	Es, 0·4	Es, 0·6	Es, 0·4	Es, 0·4
Integrated, 2008												
Date	20 March	7 April	17 April	24 April		16 May	22 May	10 June	22 June			
a.i., Dosage	CoH, 0·1	Ca, 0·2	Te, 0·075	Fe, 0·1	–	Te, 0·075	Pr, 0·05	CoH, 0·1	Pr, 0·05	–	–	–
Organic, 2008												
Date	22March	9April	18 April	25 April	29 April	13 May	18 May	26 May	4 June	12 June	21 June	29 June
a.i., Dosage	Ca-Po, 0·15	CoH, 0·1	Es, 0·75	Es, 0·4	Es, 0·6	Es, 0·6	Es, 0·4	Es, 0·4	Es, 0·6	Es, 0·4	Es, 0·6	Es, 0·5
Integrated, 2009												
Date	18 March	12 April	22 April	26 April	5 May		22 May	5 June	18 June	26 June		
a.i., Dosage	CoH, 0·1	Ca, 0·2	Te, 0·075	Bo+Pi, 0·075	Ma, 0·2	–	Pr, 0·05	CoH, 0·1	Te, 0·075	CoH, 0·1	–	–
Organic, 2009												
Date	20 March	10April	21April	25April	5 May	14 May	22 May	31 May	5 June	14 June	24 June	30 June
a.i., Dosage	Ca-Po, 0·15	CoH, 0·1	Es, 0·75	Es, 0·4	Es, 0·4	Es, 0·4	Es, 0·4	Es, 0·4	Es, 0·3+ CoH, 0·1	Es, 0·4	Es, 0·4	Es, 0·3+ CoH, 0·1

No schedule is presented for 2007 as this year was omitted from the experiment because of severe late spring frost.

a.a.i., active ingerdients; CoH, copper hydroxide: Funguran-OH 50 WP, 77%, Spiess-Urania Chemicals GmbH; Es, elementary sulphur: Kumulus S: 80%, BASF Hungaria Ltd; Te, tebuconazole: Foliculr solo 25WG, 250 g L^{-1}, Bayer Hungaria Ltd; Ma, mancozeb: Dithane DG NeoTec, 75%, Dow Agrosciences Hungary Ltd; Pr, prochloraz: Mirage 45 EC, 450 g L^{-1}, Makhteshim Agan Hungary Ltd; Ca, captan: Merpan 50 WP, 470 g kg^{-1}, Makhteshim Agan Hungary Ltd; Ip, iprodione: Rovral 50 WP, 50%, BASF Hungaria Ltd; Pe, penconazole: Topas 100 EC, 10%, Syngenta Ltd; Bo + Pi, boscalid + piraclostrobin: Signum WG, 27 + 7%, BASF Hungaria Ltd; Fe, fenhexamid: Teldor 500 SC, 500 g L^{-1}, Bayer Hungaria Ltd; Ca-Po, calcium polysulphide: Tiosol, 29%, Tiolos Ltd.

18 days after petal fall in the organic and integrated orchard blocks, respectively, and slowly developed up to 42 and 50 days after petal fall on cvs Újfehértói fürtös and Érdi bőtermő, respectively. A considerable increase in sporulating area occurred between 18 days after petal fall and harvest in both production systems and on both cultivars.

Final disease incidence

Analyses of variance for final disease incidences of BB, FB1, FB2, FB3, ΣFB, FR and SA indicated significant ($P < 0{\cdot}05$) differences amongst years, management systems and cultivars (Table 2). Analyses of variance for final disease incidence of FB2 indicated significant ($P < 0{\cdot}05$) differences among years and management systems, but not between cultivars. There were no significant interactions among treatment factors.

According to the results of analyses of variance, all brown rot measures were shown separately for years, management systems and cultivars (Table 3). Final disease incidence and sporulating area were 2–15 times higher in all years in the organic than in the integrated management system and were significantly different ($P < 0{\cdot}05$) for all brown rot measures (Table 3). The differences amongst brown rot measures and between the

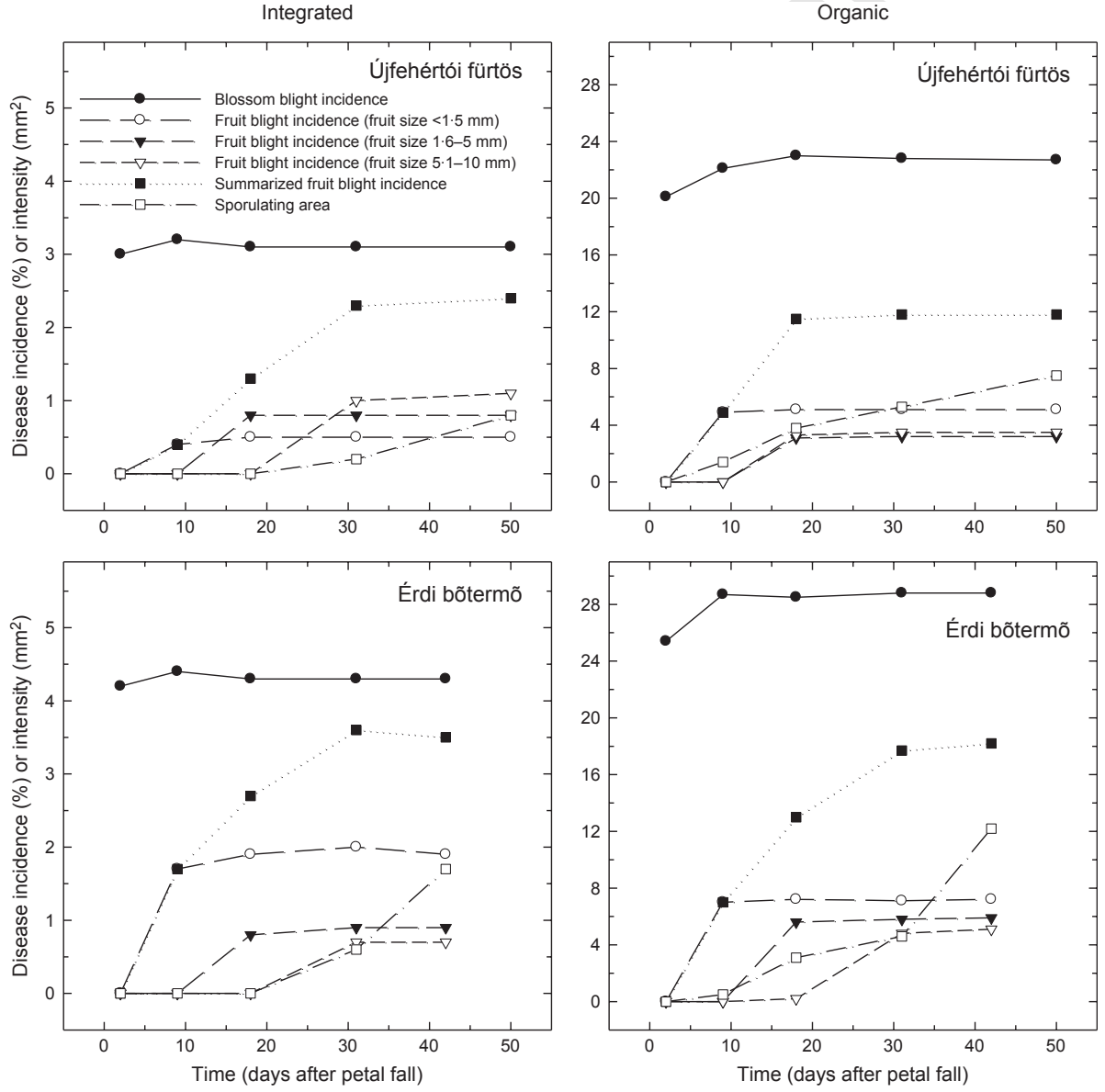

Figure 2 Temporal development of brown rot blossom blight (BB), fruit blight incidence at fruit size <1·5 mm (FB1), fruit blight incidence at fruit size 1·6–5 mm (FB2), fruit blight incidence at fruit size 5·1–10 mm (FB3), summarized fruit blight incidence (ΣFB), and sporulating area (SA) in integrated and organic sour cherry orchards on cultivars Újfehértói fürtös and Érdi bőtermő (Eperjeske, Hungary, 2009).

two systems increased in the order of fruit rot, blossom blight, fruit blight and sporulating area.

In the organic production system, final blossom blight incidence (range 11·7–34·4%) was the greatest of the blight measures in both cultivars and all years (Table 3). In this production system, similar levels of final disease incidence were reached for fruit rot (10·8–25·5%) and for sum of fruit blight (8·5–22·3%). Values of final fruit blight incidences increased in the order of FB2, FB3 and FB1 fruit blight measures. The final sporulating area ranged from 5·2 to 16·4 mm^2.

In the integrated production system, final fruit rot incidence (range 2·3–6·2%) was the greatest of the blight measures, followed by final blossom blight incidence (0·5–5·3%), on both cultivars and in all years (Table 3). In this production system, values of final disease incidences for fruit blight (FB1, FB2, FB3 and ΣFB) and sporulating area were below 5% and 3 mm^2, respectively, on both cultivars and in all years.

Final disease incidences of all brown rot symptom types were larger on the early -season cv. Érdi bőtermő in all years and both production systems than on the late-season cv. Újfehértói fürtös, except for FB2 in 2005 (Table 3). However, cultivar differences in brown rot symptoms were significant ($P < 0·05$) only in the organic production system, except for the FB2 measure (Table 3).

Relationships between brown rot symptom types

Pearson's correlation coefficients showed that brown rot blossom blight (BB) incidence correlated significantly with incidence of fruit blight on various fruit sizes (FB1, FB2, FB3, and ΣFB; Table 4). Incidences of fruit blight on different fruit sizes were significantly correlated with each other. Sporulating area of blighted flowers and fruits (SA) significantly correlated with incidences of fruit rot (FR), blossom blight (BB) and the sum of fruit blight (ΣFB). In every case, correlation coefficients were larger in organic than in integrated blocks.

Among the five highest correlation coefficients, relationships between BB and FB1 and between SA and FR were the strongest from an epidemic standpoint as they indicated a connection between different brown rot symptom types (Table 4). Thus, the relationships between these two pairs of variables were investigated further by linear regression analysis (Fig. 3). This revealed a highly significant ($P < 0·001$) relationship between BB and FB1 ($r = 0·878$ and 0·989, for integrated and organic plots, respectively), but the slopes were not significantly different between the two management systems ($P = 0·216$ according to a t-test). The linear regression analyses also indicated a significant relationship between SA and FR ($r = 0·810$, $P = 0·004$ and $r = 0·933$, $P < 0·001$ for integrated and organic plots, respectively), with the slopes being significantly different between the two management systems ($P = 0·006$ according to a t-test).

Discussion

This study is the first to describe the symptoms and temporal development of brown rot fruit blight in integrated and organic sour cherry production systems. Incidence of fruit blight exceeded 20% in the organic system, but remained below 5% in the integrated production system by harvest. This study also indicated that fruit blight incidences were linked to blossom blight in spring and fruit rot by harvest; in addition, the relationships between blossom blight vs. fruit blight, as well as between sporulating area of blighted flowers and fruits vs. fruit rot, were stronger in the organic than in the integrated production system.

During the entire assessment periods, fruit rot and blight incidences were 2–10 times higher in the organic orchard than in the integrated one, which might be associated with larger inoculum sources in the organic orchard. In organic orchards there are commonly two to three applications of copper (0·05–0·2%) or calcium polysulphide (0·15–0·2%) in early spring followed by

Table 2 Analysis of variance of the effects of year (2005, 2006, 2008 and 2009), management system (integrated vs organic) and cultivar (Újfehértói fürtös and Érdi bőtermő) on final disease incidence of blossom blight (BB), fruit blight incidence at fruit size <1·5 mm (FB1), fruit blight incidence at fruit size 1·6–5 mm (FB2), fruit blight incidence at fruit size 5·1–10 mm (FB3), summarized fruit blight incidence (ΣFB), sporulating area (SA), and fruit rot incidence (FR) at harvest in sour cherry orchards (Eperjeske, Hungary)

Source of variation	d.f.[a]	BB		FB1		FB2		FB3		ΣFB		SA		FR	
		MS[b]	$P > F$[c]	MS	$P > F$	MS	$P > F$	MS	$P > F$	MS	$P > F$	MS	$P > F$	MS	$P > F$
Year (Y)	3	74·26	0·019	145·33	0·016	250·66	0·048	222·11	0·029	39·27	0·051	20·08	0·013	36·39	0·049
Management (M)	1	1368·15	0·031	1916·34	0·045	2960·29	0·046	4105·21	0·033	710·96	0·037	200·09	0·001	548·43	0·031
Main plot error	3	45·34		96·03		161·55		138·13		27·58		15·48		18·02	
Cultivar (C)	1	100·83	0·014	84·24	0·027	97·77	0·088	181·22	0·035	47·67	0·032	5·61	0·041	59·88	0·031
M × C	1	24·13	0·063	18·47	0·107	21·07	0·345	67·69	0·083	27·59	0·086	1·48	0·134	32·39	0·079
Subplot error	2	1·49		2·37		14·13		6·38		2·58		0·245		1·93	

[a]d.f. = degrees of freedom.
[b]MS = mean squares.
[c]Probability values associated with F-tests.

Table 3 Final disease incidence of brown rot blossom blight (BB), fruit blight incidence at fruit size <1·5 mm (FB1), fruit blight incidence at fruit size 1·6–5 mm (FB2), fruit blight incidence at fruit size 5·1–10 mm (FB3), summarized fruit blight incidence (ΣFB), sporulating area (SA) and fruit rot (FR) at harvest in integrated and organic sour cherry orchards on cvs Újfehértói fürtös (Uf) and Érdi bőtermő (Eb) (Eperjeske, Hungary, 2005–2009); 2007 was omitted from the experiment because of severe late spring frost

	Disease measures															
Cultivar/year	BB		FB1		FB2		FB3		ΣFB		SA [a]		FR			
Integrated																
Uf 2005	1·5	b	0·2	a	0·6	bcd	0·2	a	1	ab	0·5	ab	4·1	b		
Uf 2006	3·3	c	0·6	ab	0·4	abc	0·5	abc	1·5	bc	0·7	ab	4·8	bc		
Uf 2008	1·5	b	0·5	ab	0·8	cde	1·1	d	2·4	d	1·1	b	4·3	b		
Uf 2009	0·5	a	0·1	a	0·1	a	0·2	a	0·4	a	0·2	a	2·3	a		
Eb 2005	4·8	d	0·8	ab	0·6	bcd	0·8	bcd	2·2	cd	0·3	a	3·9	b		
Eb 2006	5·3	d	1·2	b	0·9	de	0·7	bcd	2·8	d	1·1	b	6·2	d		
Eb 2008	5·2	d	2·2	c	1·1	e	0·9	cd	4·2	e	2·1	c	5·5	cd		
Eb 2009	2·9	c	0·4	ab	0·3	ab	0·4	ab	1·1	ab	0·4	ab	4·1	b		
$LSD_{0·05}$ [b]	0·9		0·8		0·4		0·4		0·8		0·7		1·1			
Organic																
Uf 2005	22·5	cd	6·7	b	4·2	d	6·0	bc	16·9	c	8·7	bc	15·8	b		
Uf 2006	26·7	e	8·3	cd	2·6	bc	6·5	c	17·4	c	11·5	c	16·8	b		
Uf 2008	17·3	ab	4·9	a	3·1	c	2·2	a	10·2	a	7·5	ab	10·1	a		
Uf 2009	11·7	a	4·6	a	0·9	a	3·0	a	8·5	a	5·2	a	10·8	a		
Eb 2005	34·4	f	10·2	e	2·9	bc	9·1	d	22·2	d	16·4	d	25·5	c		
Eb 2006	32·8	f	9·2	de	4·4	d	8·7	d	22·3	d	15·2	d	24·7	c		
Eb 2008	23·8	de	7·1	bc	5·8	e	4·9	b	17·8	c	12·2	c	15·4	b		
Eb 2009	19·8	bc	6·1	ab	1·9	ab	5·5	bc	13·5	b	8·4	b	15·1	b		
$LSD_{0·05}$	3·8		1·6		1·0		1·3		2·6		2·9		2·9			
Overall (cultivar and year)																
Integrated	3·1	a	0·8	a	0·6	a	0·6	a	2·0	a	0·8	a	4·4	a		
Organic	23·6	b	7·1	b	3·2	b	5·7	b	16·1	b	10·6	b	16·8	b		
$LSD_{0·05}$	5·9		1·6		1·2		1·9		4·0		3·1		4·4			
Overall (year)																
Integrated, Uf	1·7	a	0·3	a	0·5	a	0·5	a	1·3	a	0·6	a	3·9	a		
Integrated, Eb	4·6	a	1·2	a	0·7	a	0·7	a	2·6	a	1·0	a	4·9	a		
Organic, Uf	19·6	b	6·1	b	2·7	b	4·4	b	13·2	b	8·2	b	13·4	b		
Organic, Eb	27·7	c	8·1	c	3·8	b	7·1	c	19·0	c	13·1	c	20·2	c		
$LSD_{0·05}$	7·5		2·0		1·7		2·4		4·9		3·5		5·3			

[a]SA = porulating intensity is given in mm^2.

[b]$LSD_{0·05}$ = least significant differences at $P = 0·05$ level. Values followed by the same letter are not significantly different according to this test.

two to three fungicide applications of elemental sulphur (0·4–0·7%) during flowering, then weekly applications of elemental sulphur until harvest (11–14 applications). However, neither blossom and fruit infections nor mass production of conidia can be suppressed effectively by the fungicides used in organic orchards, such as copper- and sulphur-based compounds (Tamm *et al.*, 2004; Holb & Schnabel, 2005, 2008a,b; Everhart *et al.*, 2011), and therefore, brown rot development is continuous in spring and summer in organic orchards. Consequently, large amounts of inoculum were present by harvest in organic orchards. This rarely occurs in integrated cherry orchards because of the frequent use of contact and systemic fungicides against brown rot which can effectively reduce brown rot blossom and fruit infections (Batra, 1991; Tamm, 1994; Ogawa *et al.*, 1995; Holb, 2004).

Neither epidemiological features nor control options have been described for brown rot fruit blight in previous studies. This study showed that fruit blight symptoms were related to different fruit sizes of a given phenological stage (Figs 1 and 2; Table 3). Significant correlation and linear regression analyses revealed that brown rot blossom blight may have an epidemiological connection to early fruit blight symptoms (Table 4; Fig. 3). This result was also supported by the observation that 33–54% and 42–68% of infected shoots included both symptom types of blossom blight and early fruit blight in integrated and organic production systems, respectively, in all years (I. J. Holb, unpublished data). These results suggest that inoculum sources for fruit blight infection may be present on the same shoot where both symptom types occur.

Significant correlation between sporulating area and fruit rot, as well as linear regression analyses, revealed that inoculum sources of blighted flowers and fruits may increase fruit rot by harvest. Therefore, removal of blighted shoots including both symptoms of blossom and fruit blight may effectively reduce fruit rot. This control

option has higher priority when fruits are injured before harvest. Fruits can be injured by several means, including insect damage, e.g. *Rhagoletis* spp., and/or fruit crack caused by rainy periods before harvest (Opara *et al.*, 1997; Sekse, 1998; Holb & Scherm, 2008). The brown rot susceptibility of an injured fruit is greatly increased and the sporulation capacity may also increase on cracked fruits as a result of precipitation events (Wilcox, 1989; Tamm & Flückinger, 1993; Xu *et al.*, 2007). Fruit rot in Hungary is mostly related to cracking caused by heavy and frequent rains 5–10 days before harvest (I. J. Holb unpublished data), which is controlled by chemical treatments and quick harvest operations. However, in integrated orchards, sprays before harvest are usually avoided because of the issue of chemical residues in fruit tissue (Holb, 2004). In organic orchards, chemical residues are not an issue, but elemental sulphur has low efficacy against fruit rot (Holb & Schnabel, 2008a,b) and the spraying material contaminates fruit surfaces, which need to be washed before marketing. Therefore, removal of blighted shoots during the season coupled with plastic rain shields above the trees (Borve & Stensvand, 2003) is one the most sustainable control options to avoid fruit rot in both integrated and organic sour cherry orchards.

In their work, Byrde & Willetts (1977) and Batra (1991) listed *M. laxa* as able to overwinter on mummified fruits, blighted flowers, and leaves or twigs (canker) on stone fruit species. However, in the case of sour cherry, there are some key differences from other stone fruit species. For instance, unlike plums, peaches and apricots, where mummified fruits typically remain attached to the tree, mummified fruits of sour cherry do not remain on the tree, but fall to the ground by the end of autumn. This difference may be related to the longer fruit stalks of sour cherry, resulting in easier fruit drop than in other stone fruits. Thus, no mummified fruits appear on sour cherry trees by the beginning of the following season and, as a consequence, do not provide a sporulation source (I. J. Holb, unpublished data). It is likely that even if mummified fruits did remain on sour cherry trees, they would produce conidia of both *M. fructigena* and *M. laxa* fungi, of which only those of *M. laxa* infect cherry blossoms (Holb, 2003). When the mummified fruits fall to the ground, they will be decomposed by soil microorganisms, and in addition, no reports of sexual forms of the fungus (such as apothecia) are known from central Europe (Holb & Schnabel, 2005). Of the other potential inoculum sources, blighted flowers and leaves disappear during the winter under central European weather conditions; only blighted twigs remain, with a tough covering of gummy materials (quite characteristic of cvs Érdi bőtermő and Újfehértói fürtös), through which it is difficult for the fungus to sporulate in the following spring (Holb, 2003). In addition, the sporulation capacity of twigs appears to end at the end of May, which is too early for fruit rot infection of sour cherry, as fruits start to be susceptible to brown rot at the end of June or early July in Hungary. Thus, inoculum sources other than blossom and/or fruit blight are rarely present in June in sour cherry orchards.

In integrated orchards, fruit blight caused by *M. laxa* seemed to be effectively controlled as overall incidences of the symptoms remain below 5% (Table 3). Therefore, no additional chemical control means are needed against fruit blight in integrated orchards. However, the level of fruit blight symptoms in organic cherry orchards (9–22%) can result in significant yield losses. In these orchards, the sum of fruit rot and total fruit blight incidences together exceeded blossom blight incidence alone (Table 3). Blossom blight incidence always contains invisible unset fruits; therefore, the real impact

Table 4 Pearson's correlation coefficients and associated significance levels (in italic) among measures of brown rot blossom blight (BB), fruit blight incidence at fruit size <1·5 mm (FB1), fruit blight incidence at fruit size 1·6–5 mm (FB2), fruit blight incidence at fruit size 5·1–10 mm (FB3), summarized fruit blight incidence (ΣFB), sporulating area (SA), and fruit rot (FR) at harvest in integrated (INT) and organic (ORG) sour cherry orchards on cultivars Újfehértói fürtös and Érdi bőtermő (Eperjeske, Hungary, 2005–2009)

	BB		FB1		FB2		FB3		ΣFB		SA	
	INT	ORG	INT	ORG	INT	ORG	INT	ORG	INT	ORG	INT	ORG
FR	0·681	0·745	0·657	0·734	0·683	0·740	0·694	0·766	0·712	0·755	0·791	0·874
	0·0538	*0·0316*	*0·0633*	*0·0354*	*0·0533*	*0·0337*	*0·0479*	*0·0273*	*0·0411*	*0·0296*	*0·0199*	*0·0039*
SI	0·736	0·786	0·567	0·687	0·584	0·652	0·604	0·676	0·718	0·775		
	0·0346	*0·0222*	*0·0877*	*0·0511*	*0·0842*	*0·0642*	*0·0789*	*0·0556*	*0·0404*	*0·0248*		
ΣFB	0·722	0·773	0·754	0·865	0·772	0·822	0·783	0·863				
	0·0395	*0·0251*	*0·0299*	*0·0046*	*0·0254*	*0·0127*	*0·0231*	*0·0049*				
FB3	0·702	0·742	**0·812**	**0·888**	**0·845**	**0·926**						
	0·0444	*0·0332*	***0·0142***	***0·0030***	***0·0087***	***0·0006***						
FB2	0·768	0·817	**0·876**	**0·923**								
	0·0270	*0·0135*	***0·0037***	***0·0007***								
FB1	**0·845**	**0·901**										
	0·0087	***0·0015***										

Year 2007 was omitted from the experiment because of severe late spring frost. The five largest correlation coefficients each for integrated and organic blocks are shown in bold.

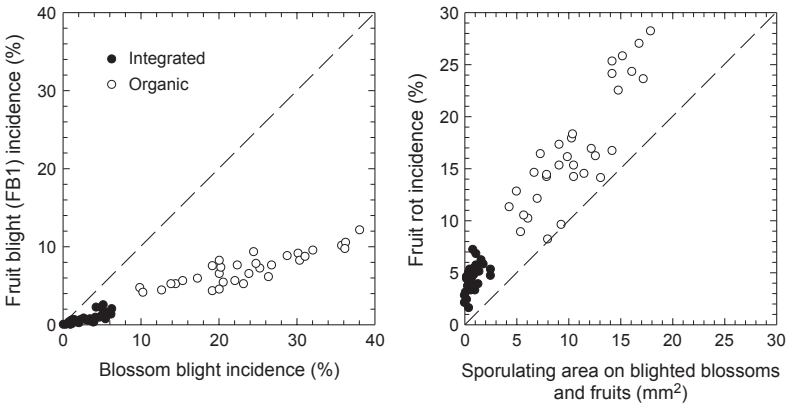

Figure 3 Relationships between brown rot fruit blight (FB1) incidence and brown rot blossom blight incidence, as well as between fruit rot incidence and sporulating area on blighted flowers and fruits in integrated (●) and organic blocks (○) of sour cherry orchards at Eperjeske, from 2005 to 2009. Each point represents data from a single site, a single year and a single cultivar. The dashed line indicates a 1:1 relationship.

of blossom blight damage on yield loss is somewhat indirect and less than the final blossom blight incidence (Holb & Schnabel, 2005). However, fruit blight incidence relates to fruits which are already set, so it has a direct influence on yield loss. Thus, specific control against fruit blight may be required in organic orchards. As fungicides approved in this system are not effective enough (Tamm *et al.*, 2004; Holb & Schnabel, 2005, 2008a,b; Everhart *et al.*, 2011), other control means are needed for reducing fruit blight symptoms and fruit blight sporulation.

According to the results of this study, peaks in blight symptoms are linked to certain phenological stages of the tree. The peak for blossom blight incidence was 9 days after petal fall, whilst that of final fruit blight incidence was 31 days after petal fall (Fig. 2). After these dates, no new blossom blight and/or fruit blight symptoms appeared and the disease levelled off. As a consequence, in epidemiological studies, these peak points can be used as final assessment dates for cumulative disease symptom or incidence detection.

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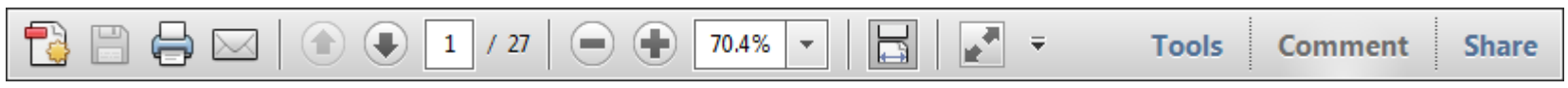

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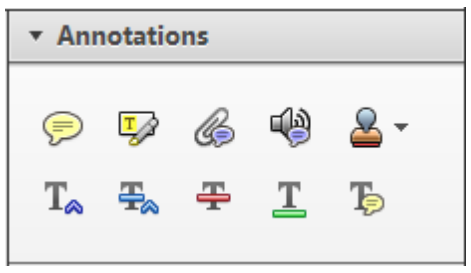

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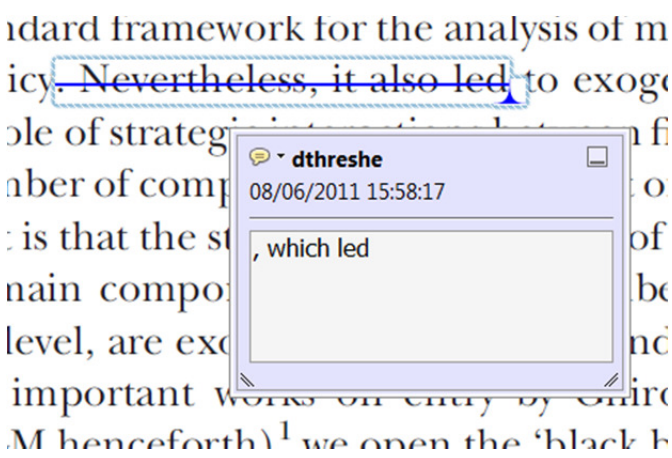

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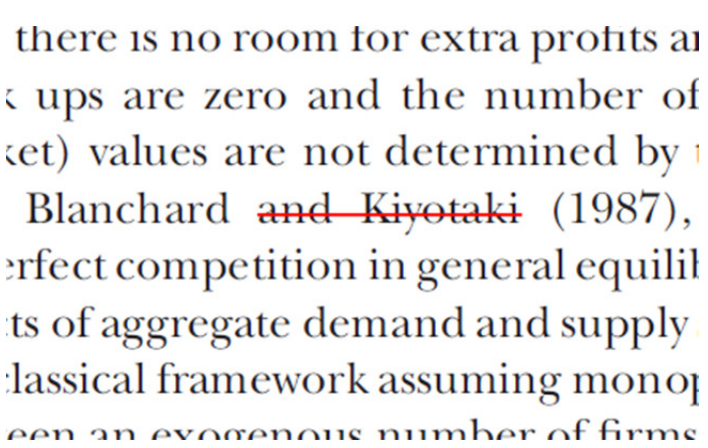

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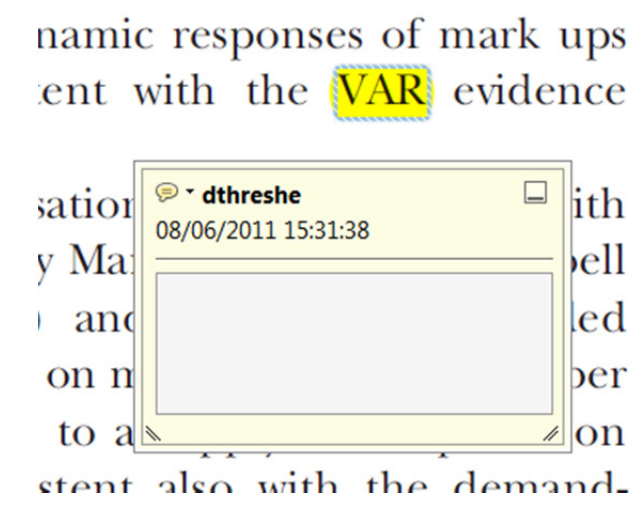

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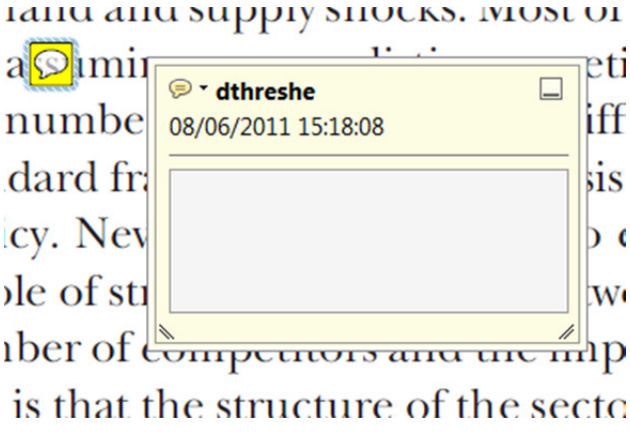

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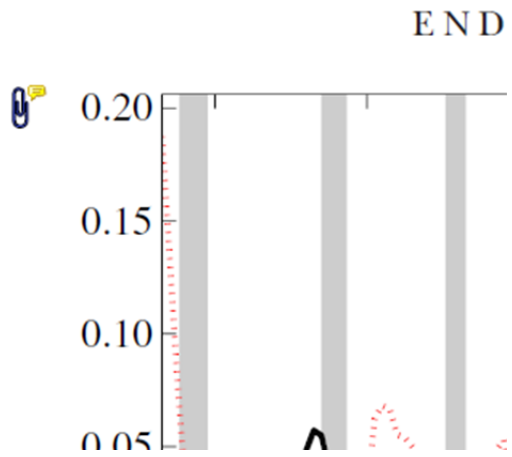

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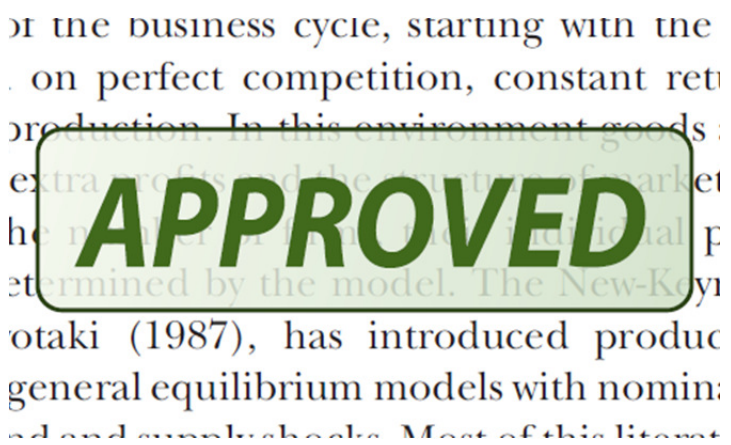

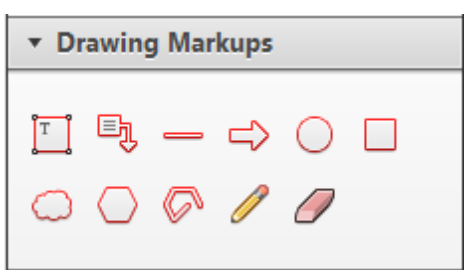

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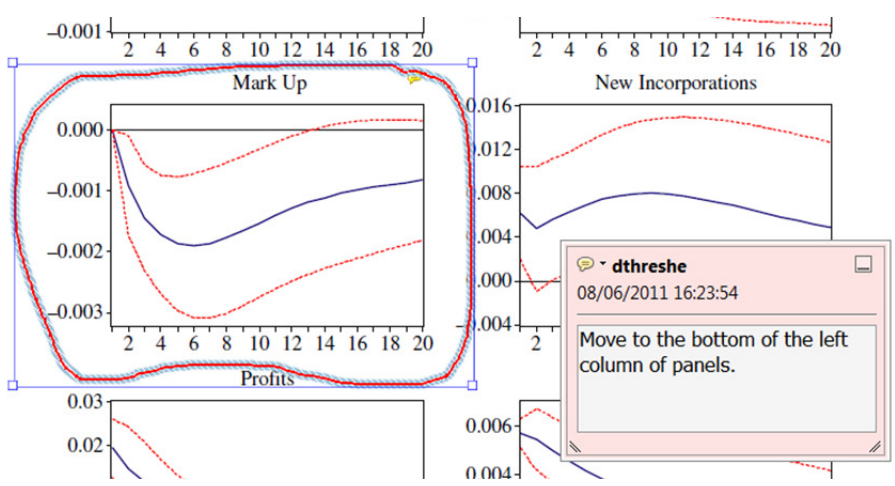

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