Leptosphaeria maculans (stem canker) on winter oilseed rape in England: some relationships between plant and pathogen development

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ABSTRACT

At Rothamsted during 2000/01 and 2001/02 field experiments were done using the fungicide Punch C (flusilazole + carbendazim) at three different timings during the autumn to find the optimum time for controlling stem canker epidemics on winter oilseed rape. Plots were regularly sampled to assess the effect of fungicide timing on both the phoma leaf spot and stem canker phases of the disease. In addition, ten plants in each of three untreated plots in these experiments were marked soon after emergence. Development and loss of individual leaves and the number of phoma leaf spot lesions occurring on each leaf was monitored through the season on these marked plants. This paper explores relationships between plant development (leaf production and loss) and pathogen progress (phoma leaf spotting and stem canker) and the consequences these may have on optimising fungicide timing for control of this disease.

Key words: oilseed rape – phoma – stem canker – leaf development

INTRODUCTION

Leptosphaeria maculans (phoma stem canker) is the most damaging disease of winter oilseed rape in England, causing estimated losses of more than £30M in recent years (Defra survey results: http://www.csl.gov.uk/prodserv/cons/crop/survey/osrintro.cfm). It is a monocyclic disease with two distinct phases. During autumn and winter, air-borne ascospores cause leaf lesions (the phoma leaf spot phase); from these lesions the pathogen grows down petioles to the crown of the plant, causing cankers to develop at the stem base during spring and summer (the stem canker phase). Previous studies on the epidemiology of *L. maculans* have investigated the relationship between plant and pathogen development and have generally found that early phoma leaf spot causes severe stem canker (Hammond and Lewis 1986, Sun et al 2000). Effects of fungicide timing on different phoma and stem canker epidemics have been studied and the need for autumn fungicide applications confirmed (Gladders et al 2001). However there have been few studies where plant and pathogen development and fungicide timing have been investigated together. This paper presents the results of two field experiments where these three elements were investigated and discusses the implications for disease control.

MATERIALS AND METHODS

In 2000/01 and 2001/02 field experiments were done at Rothamsted on winter oilseed rape (cv. Apex) to investigate the effects of fungicide timing on development of phoma leaf spot and subsequent stem canker epidemics. Experiments were sown on 23 August 2000, and on 14 August 2001, and they were harvested on 23 July 2001 and 18 July 2002. In both experiments Punch C (carbendazim + flusilazole) was applied on three occasions in the autumn. The first application was made when c. 10% plants had phoma leaf spotting (9 October 2000 and 22 October 2001) and the second and third applications at c. monthly intervals thereafter. Ten plants/plot were sampled c. monthly during both seasons and assessed for phoma leaf spot and for stem canker severity (score 0-4). In addition, ten plants in each of three untreated plots in these experiments were marked soon after emergence and monitored c. weekly from October to February. Each week, any new leaves were marked and the number of phoma lesions on each leaf, together with the number of new leaves and the number of leaves which had fallen off, was recorded. Stem canker severity was assessed on these marked plants just prior to harvest using the same 0-4 scoring system.

RESULTS

In both seasons the first seven leaves were produced within 60 days of sowing without loss of older leaves. Thereafter, production of new leaves occurred at approximately the same rate as the loss of old leaves. The rate of loss of old leaves was similar in the two seasons, but the rate of development of new leaves was lower in 2001/02 (probably because of a dry autumn period and a severe epidemic of powdery mildew). Thus, the mean number of leaves/plant was 7.8 from 60 – 160 days after sowing in 2000/01 and 6.5 in the same period in 2001/02. Mean stem diameter in July 2001 was 3.1cm and in April 2002 it was 1.9cm. Although measurements of stem diameter were made two months later in 2001 than in 2002, this is unlikely to account for all the observed difference. In both seasons the time that each leaf was present on the plant (i.e. from leaf production to leaf fall), was progressively longer for each new leaf that developed. For example, in both seasons leaf number four was present on at least half the marked plants for c. for 50 days, while leaf number ten was present for c. 90 days.

Table 1.	Effect	of fungicio	le timing oi	n phoma	leaf spot	t and ster	n canker	development

Fungicide timing		Stem canker score						
		(0-4)						
	November		January		February		June	
	2000/01	2001/02	2000/01	2001/02	2000/01	2001/02	2000/01	2001/02
Untreated	33.5	10.3	33.3	4.2	14.7	33.3	2.0	2.0
$F1^{1}$	1.3	3.1	33.3	1.1	9.1	43.3	1.3	0.6
$F2^2$	-	-	20.5	1.1	8.6	43.3	0.9	0.5
F3 ³	-	-	31.6	2.4	4.6	23.3	0.9	0.4
SED	2.8	2.4	3.3	1.7	3.6	14.2	0.2	0.2
df	4	4	12	14	14	14	14	16

¹F1 applied on 9 October 2000 and 22 October 2001

²F2 applied on 6 November 2000 and 19 November 2001

³F3 applied on 14 December 2000 and 8 January 2002



Figure 1. Number of lesion-days contributed by each leaf number from first true leaf (leaf 1) on 30 marked plants in 2000/01 (\Box) and 2001/02 (\blacksquare). (Leaves 11 -13 not assessed in 2000/01)

In 2000/01 a severe phoma leaf spot epidemic developed, with a higher percentage of leaves/plant affected (Table. 1) and higher numbers of lesions/leaf (maximum mean number of lesions/leaf 2.8 in 2000/01 and 2.0 in 2001/02). In 2000/01 the highest number of lesions occurred on leaf 6 (mean 6.5 lesions/leaf on 30 November) and in 2001/02 on leaf 4 (mean 1 lesion/leaf on 23 October). By multiplying the number of lesions on each leaf by the number of days each lesion was present on the plant, the number of lesion-days for each leaf was

calculated. Lesion-days for each leaf were far higher in 2000/01 than 2001/02 (Fig.1). In both seasons leaves 1 to 3 had generally fallen from the plant before phoma lesions were observed. In 2000/01 the number of lesion days was greatest on leaves 7-10, while in 2001/02 the number of lesion days was similar on leaves 4 -10.

In 2000/01 the first fungicide application (F1) was made when >50% of leaves 2-6 were present, the second (F2) when >50% of leaves 4-10 were present, and the third (F3) when >50% of leaves 8-14 were present. The corresponding leaf numbers for 2001/02 were 3-9 (F1), 6-12 (F2) and 8-14 (F3). In both seasons, although F1 was applied when >50% of the most severely affected leaf was present, and although this treatment significantly reduced the early phoma epidemic, F2 and F3 were more effective against stem canker, particularly in 2001/02 when phoma leaf spot was less severe (Table 1). Despite the greater severity of phoma leaf spot in 2000/01, the stem canker score on untreated plants in June was the same in both seasons. However, in July the mean stem canker score on the 30 untreated, marked plants was 3.2 in 2000/01 and 2.7 in 2001/02.

DISCUSSION

Although the phoma leaf spot epidemic was more severe in 2000/01, the final stem canker score on the 30 marked plants was only 12% higher. This was probably due in part to the larger number of leaves/plant in 2000/01 giving rise to more robust plants with thicker stems which were better able to withstand the stem canker phase. In both seasons the first fungicide application (F1) was made when >50% of the most heavily infected leaf layer was present (leaf 4 in 2000/01 and leaf 7 in 2001/02), while F2 and F3 were applied when most or all of these leaves had fallen from the plant. F1 was very effective in reducing early phoma leaf spot and yet, in both seasons, F2 and F3 were more effective against the stem canker phase. Punch C has protectant and curative activity against L. maculans and will therefore reduce phoma leaf spot infections that occur both before and after it is applied. Later leaves are produced as temperatures in the autumn are falling and stay on the plant for longer periods. As a result, although these later leaves may not be as heavily infected as earlier leaves, they contribute more lesion-days. This, together with the curative activity of Punch C, may explain the apparent contradiction between phoma leaf spot and stem canker control observed in these experiments. Growers are advised to apply fungicide when 10% of plants have phoma leaf spot. However, at Rothamsted, in seasons with both severe (2000/01) and moderate (2001/02) stem canker epidemics, Punch C was more effective when applied 1-2 months after this time.

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