SPREAD OF LIGHT LEAF SPOT (PYRENOPEZIZA BRASSICAE) IN OILSEED RAPE CROPS IN THE UNITED KINGDOM

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INTRODUCTION

Light leaf spot of brassicas, caused by Pyrenopeziza brassicae Sutton et Rawlinson, is as one of the most important diseases of oilseed rape in the United Kingdom (Anon. 1986). Until recently it was thought that the disease was spread by splash-borne conidia (Rawlinson et al 1978), thus restricting the rate and range of disease spread. However, the teleomorph of the fungus was found in an oilseed rape crop in the U.K. in 1986 (Lacey et al 1987). When the sexual stage of the fungus is present in the crop many ascospores, which can be efficiently dispersed by wind, may be released (McCartney et al 1986; McCartney and Lacey 1990).

Wind dispersed ascospores of P. brassicae have the potential of spreading light leaf spot considerable distances and may be involved in disease spread from field to field. Indeed, in the autumn, ascospores can be released from infected volunteer plants after harvest (McCartney and Lacey 1990) and thus may be the source of initial infection of newly sown crops. However, little is known of the influence of ascospores on disease spread within infected fields. We report here the results of five years of disease monitoring in infected oilseed rape crops at Rothamsted Experimental Station. We also report the results of measurements of disease spread within infected crops.

MATERIALS AND METHODS

Five crops were studied in consecutive seasons (1985-86 to 1989-90). All crops were sited at Rothamsted Experimental Station and the sowing dates, plot sizes and cultivars used are shown in Table 1. The experimental plots were rectangular and, except for the 1985-86 crop, they were within larger fields also sown to oilseed rape. In 1985-86 the rest of the field contained cereals. The husbandry of the crops is described elsewhere (McCartney and Lacey 1990; McCartney and Lacey 1991). No fungicides were used, but herbicides and/or insecticides were applied when required (McCartney and Lacey 1990; McCartney and Lacey 1991).

To encourage disease development chopped oilseed rape straw, taken from a crop of the same cultivar and known to have been infected with P. brassicae, was scattered over each plot one or two months after sowing. In 1985, 86, 87 and 89 the whole plot area was inoculated with oilseed rape straw. In 1988, the outer edge of the plot, consisting of a strip 2m wide, was inoculated (Fig. 1). Infected straw was also scattered on another strip, 2m wide, which divided the plot in two across its shorter side, so forming two sub-plots.

Table 1. Summary of experimental plots.

Season	Variety	Sowing date	Inoculation date	Plot size (m)
1985-86	Jet Neuf	4 Sept.	30 Oct.	70x120
1986-87	Jet Neuf	3 Sept.	6 Oct.	20x200
1987-88	Jet Neuf	16 Sept.	6 Nov.	51x51
1988-89	Cobra	7 Sept.	25 Oct.	45x90
1989-90	Cobra	22 Aug.	13 Nov.	40x40

Disease progress in the inoculated areas of all plots was monitored by assessing the fraction of leaves, stems and when appropriate pods, showing light leaf spot symptoms. Assessments were done every three or four weeks from just before inoculation until June the following year. In all seasons except 1988-89 disease was assessed from a randomly chosen sample of 10-15 plants from the plot. In 1988-89, five plants was chosen at random from seven sample areas in inoculated strips as shown in Fig. 1.

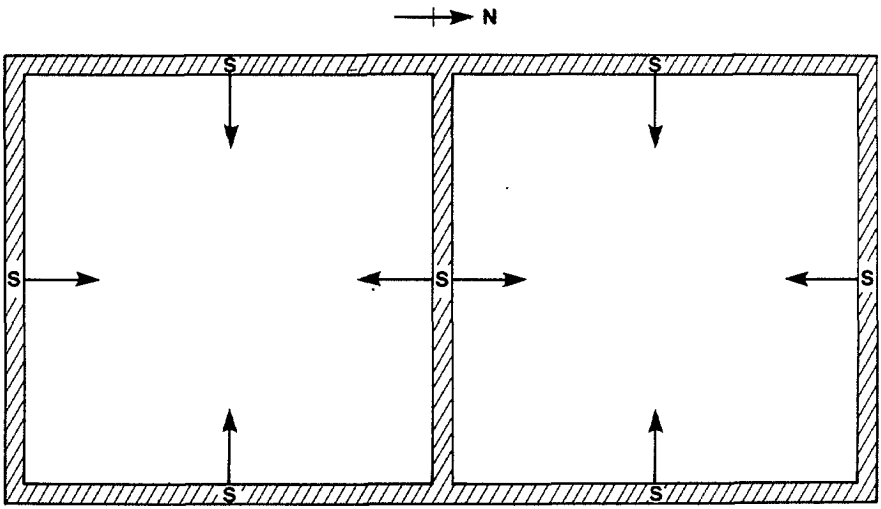

Fig. 1. Schematic diagram of the 1988-89 plot. Areas inoculated with infected straw are shown hatched and sample areas are indicated by "S". The directions of disease gradient assessment are shown by arrows.

Disease spread from infected to non-infected plants in the crop was measured in the 1988-89 and 1989-90 seasons. In 1988-89 disease was assessed at different distances from the edges of the infected strips towards the centre of the two sub-plots (Fig. 1). At each assessment eight disease gradients were measured: two pointing in each of the four directions, north, south, east and west. In 1989-90 the whole plot area was inoculated therefore gradients were measured away from the edge of the plot. Disease was assessed at 1, 3, 10 and 30m from the centre of each side of the inoculated plot. In both seasons a sample of five plants was randomly chosen from each sample area, which consisted of a 0.5m wide strip about 5m long parallel to the edge of the inoculated area. Disease on each plant was assessed as described above.

RESULTS

The seasonal progress of disease for each of the five seasons is illustrated in Figure 2. Patterns of disease development were similar for all seasons, except 1986-87, but the onset of the epidemic differed between seasons. Symptoms first appeared on leaves as early as mid-November (1986-87) or as late as mid-January (1985-86), between one and two months after the plots had been inoculated. In three of the five seasons it then took about three months for all plants to show disease symptoms, this usually happened by the end of March. In 1989-90 the epidemic took only two months to reach 100% incidence on plants. In 1986-87, 100% incidence occurred about one month (mid December) after the first appearance of symptoms. Disease incidence on leaves increased more slowly than on plants. Except for 1986-87, maximum disease incidence was reached between three and four months after symptoms appeared. Incidence on leaves reached 100% in only one season (1987-88), in the others maximum disease incidence on leaves was about 70%. The percentage disease incidence on leaves declined after May as new leaves appeared.

The change in disease incidence , measured as percent leaves infected, with distance from the inoculated crop for seasons 1989-89 and 1989-90 is shown in Figure 3. There was little difference between disease patterns for the different directions away from the source so all incidence measurements at each distance have been combined in the figure. There was a marked difference in the patterns of disease development between the two seasons. In 1988-89 there was a decrease in disease over the first 5m from the inoculated crop from January until the end of May when measurements stopped. At greater distances, disease incidence appeared to change little with distance, but, values increased with time and by May were about 60% of to those in the inoculated plot. A similar decrease in disease with distance was observed in the winter of 1989-90 but by mid-February disease incidence at distances up to 30m from the edges of the plot was similar to that within the plot (Fig. 3) and no noticeable disease gradient was apparent. From about mid February disease incidence outside the plot increased with time in a similar pattern to that within the plot and reached a maximum of about 80% infected leaves by the end of April.

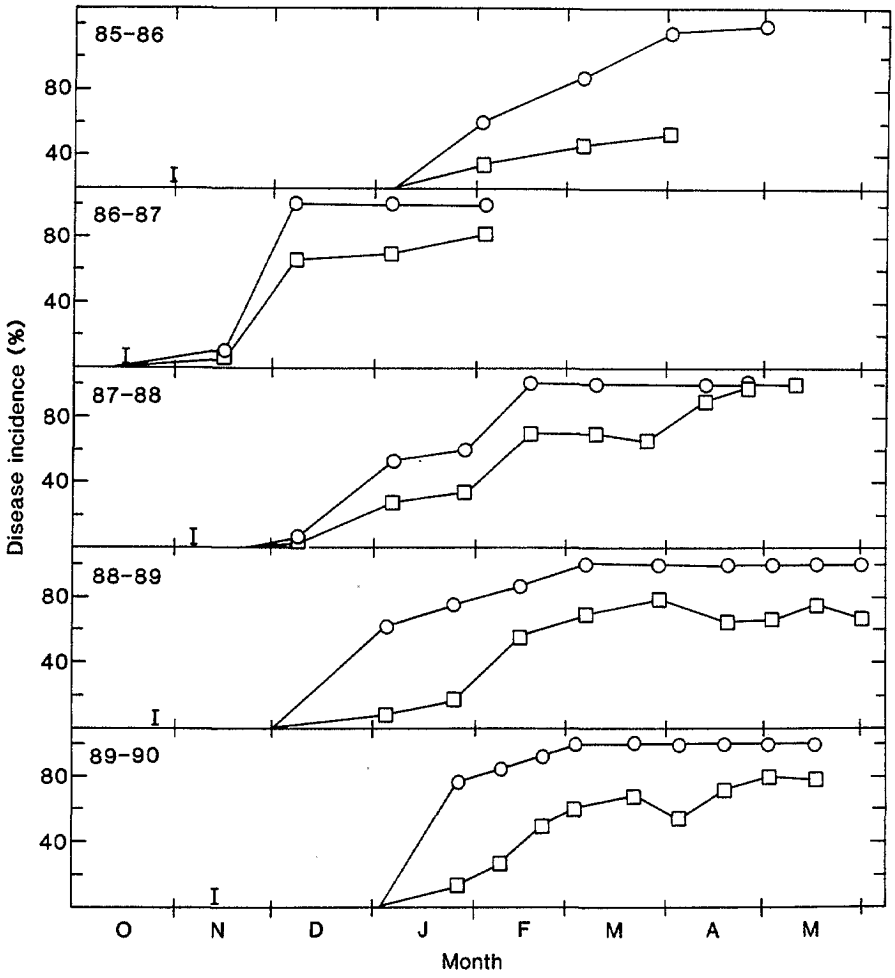

Fig. 2. Disease progress curves of the five experimental plots. Disease is expressed as % infected plants (○) and leaves (□). The date of inoculation with infected straw is shown by "I".

DISCUSSION

The experimental plots in this study were exposed to two sources of inoculum (i) the infected straw spread to promote disease development; (ii) natural background inoculum (possibly ascospores). The time between inoculation and the onset of visible symptoms differed between seasons which may reflect differences in potency of the infected straw, in the amount of background inoculum present or differences in weather. In 1989-90, the development of the epidemic outside the plot was similar to that within the plot suggesting that the straw was not a particularly effective inoculum source. The date of appearance of lesions also differed between seasons. The earliest appearance was in mid-November and the latest mid-January. Except for 1986-87, the rate of development of the subsequent epidemic was similar for each season, although the weather patterns were different (the winters of 1988-98 and 1989-90 were both mild). Thus, although crops exposed to P. brassicae inoculum in the autumn may not show symptoms until January, in most years weather may not limit disease development once the epidemic starts.

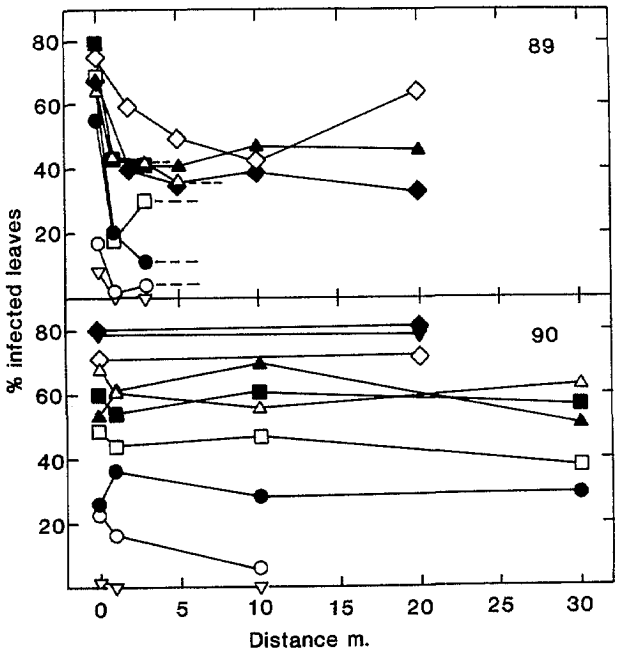

Fig. 3. Change in disease with distance from the inoculated plots. Disease is expressed as % leaves infected.

▽ - 4/1/89 6/12/89; ○ - 25/1/89 24/1/90; ● - 14/2/89 8/2/90
□ - 6/3/89 21/2/90; ■ - 29/3/89 5/3/90; △ - 19/4/89 21/3/90
▲ - 3/5/89 4/4/90; ◇ - 17/5/89 18/4/90; ◆ - 31/5/89 2/5/90
▲ - 16/5/90

The pattern of disease spread away from the inoculated areas observed in the 1988-89 season is not typical of disease spread from a single line or point source of inoculum. However, is what may be expected when there are two sources: the inoculated strip and a spatially uniform background. In these conditions disease would be expected to decrease with distance from the source until a uniform background level was reached. The 1988-89 results suggest that the inoculated strip had little influence on disease at distances greater than about five metres. When background levels were subtracted, disease decreased by half in less than a metre from the strip. Dispersal distances for splash-borne spores (eg. P. brassicae conidia) are usually shorter than this (Fitt et al 1987), but air-borne spores such as P. brassicae ascospores would probably travel further (Fitt et al 1987; McCartney and Lacey 1990). Thus, the disease gradients observed were probably mainly due to the dispersal of splash borne conidia and not ascospores. Indeed, spore trapping showed that most ascospore production occurred after disease incidence reached a maximum (McCartney and Lacey 1990). In 1989-90, decrease in disease with distance from the inoculated area was noted only at the beginning of the epidemic. The artificial inoculum may have been small and so the disease induced was quickly overtaken by that induced by background inoculum. In both 1988 and 1989 there appeared to be sufficient background inoculum available in the autumn to initiate disease epidemics in the late winter and early spring outside the inoculated crop. Thus, light leaf spot in commercial crops exposed to natural background inoculum may exhibit similar development patterns as observed in our inoculated plots. Although, when background inoculum levels are small the timing and the final intensity of infection may be different.

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