Relations between light leaf spot (*Pyrenopeziza brassicae*), pod canopy size and winter oilseed rape yield in England

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ABSTRACT

Mechanisms of yield loss from light leaf spot (*Pyrenopeziza brassicae*) and effects of tebuconazole fungicide regimes were examined in winter oilseed rape at Rothamsted in 1997/98. There was no evidence that light leaf spot decreased plant populations. The best light leaf spot control was with routine or autumn/spring applications of tebuconazole, with responses greater for cv. Bristol (susceptible) than cv. Capitol (resistant). Cv. Capitol had a greater pod green area index (GAI, estimated from light interception measurements) than cv. Bristol and routine tebuconazole treatments produced largest GAIs for both cultivars. Cv. Capitol yielded more (mean of all treatments 3.70 t ha^{-1}) than cv. Bristol (3.23 t ha^{-1}). Routine fungicide treatments most increased yield over the untreated (by 1.29 t ha^{-1} for cv. Capitol and 1.93 t ha^{-1} for cv. Bristol). Yield was positively related to pod GAI in June, indicating that the main mechanisms of yield loss from light leaf spot were reductions in numbers of pods and canopy GAI (which reduced capacity to capture light).

Key words: Light leaf spot, *Pyrenopeziza brassicae*, canopy size, yield, tebuconazole fungicide

INTRODUCTION

Light leaf spot (LLS, *Pyrenopeziza brassicae*) can cause yield losses in winter oilseed rape of >1 t ha^{-1} and cost UK producers >£30M *per annum* (Fitt *et al.*, 1997). Mechanisms of yield loss are not clear and the LLS epidemic cycle within crops is complex. Initially the disease is localised and may cause over-winter loss of plants. Crops usually compensate for low plant populations by increased branching, unless large disease patches develop. Reduction in green area index by leaf spots may limit ability of the vegetative canopy to provide assimilate for reproductive structures. Growing points of flowers and branches may also be infected and stunted. Although oilseed rape is able to compensate effectively for widely differing pod densities, with similar yields possible between 4000 and 12,000 pods m^{-2} (Lunn *et al.*, 2001), limitation of pod numbers and green area during pod filling is a potential yield loss mechanism. During stem extension and flowering, LLS epidemics can develop on stems and pods. Reduced photosynthetic capacity of pods due to LLS infection, and premature pod shatter cause yield loss, since almost all seed matter is produced in a 6 week period in June and July by pod hull and branch photosynthesis. The triazole fungicide tebuconazole, used to control light leaf spot, also has plant growth-regulating effects, reducing height and lodging to increase yield by >0.5 t ha^{-1} (Lunn *et al.*, 2002). This work was done to assess the mechanism of yield loss due to the disease and investigate disease control and PGR effects of tebuconazole.

MATERIALS AND METHODS

Cultivars Bristol and Capitol, with LLS resistance ratings of 2 (susceptible) and 8 (resistant), respectively, were grown at Rothamsted in 1997/98. Tebuconazole treatments were applied at various dates. Plant numbers m^{-2} were assessed in November and January. Monthly, % leaf, stem and pod area affected by LLS were recorded by assessing white spore masses after incubation at 5-10°C for 2-5 days (Fitt *et al.*, 1998). Phoma stem canker, downy mildew and alternaria were also assessed. Light interception was measured in early June, after flowering, with a Sunscan ceptometer (Delta T, Burwell, Cambs.). Incident photosynthetically-active light radiation (*I*, µmol photons m^{-2} s^{-1}) was measured above the canopy and light transmitted by the pod canopy (*T*) at the base of the pods. The percentage of incident light intercepted (i.e. absorbed and reflected) by the canopy was calculated as (*I*-*T*)*100/ *I*. Light extinction through a

canopy approximates Beer's Law, giving the equation $(1-F) = e^{-k\mathrm{GAI}}$. F is the fraction of light intercepted (i.e. $(I-T)/I$), k the extinction coefficient and GAI the green area index (area of green material per m^2 ground). Assuming k = 0.66, green area indices of different pod canopies were estimated. Areas of healthy and diseased canopy were then calculated from GAI and % area LLS values. Plots were harvested on 22-24 July 1998 and yield (at 10% moisture) determined.

RESULTS

Plant populations did not differ between treated and untreated plots; establishment was *c.* 50% with a population of *c.* 60 plants m^{-2} in November and January. Light leaf spot development in untreated plots started in November and reached a maximum (20% leaf area affected) in late January, with more on Bristol than Capitol. Only routine sprays prevented development of leaf lesions (until May). On Bristol, October and November full rate sprays delayed appearance of disease and reduced leaf area affected. The December spray reduced the area affected. Half-rate applications also delayed development of LLS but only slightly decreased disease severity. The spring half-rate spray reduced % areas with LLS only in plots sprayed in December. By May, only the routine spray and December/March split application had decreased LLS on leaves. On Capitol, there was less disease and fewer treatment differences. October, November and December full rate applications delayed the epidemic. Only routine and December/March sprays reduced % LLS in May. For Bristol, routine sprays delayed development of the pod phase most. In other treatments, *c.* 15-40% of the pod area was affected, with indications that spring sprays reduced the pod area affected. On Capitol, there was less LLS on pods than on Bristol, but no treatment effects. For other diseases, there were no treatment differences.

Analysis of variance showed significant effects (P<0.001) of cultivar and fungicide treatment on total canopy size with a significant (P=0.047) cultivar x fungicide interaction. Capitol had a greater GAI than Bristol. Untreated Bristol and Capitol had the smallest canopies (*c.* 1.8 and 3.2 units, respectively), whilst the routine treatments produced the largest canopies for both (*c.* 4.1 and 4.6, respectively). On Capitol, fungicide treatments other than routine increased canopy size little. On Bristol, autumn full rate sprays produced larger canopies than untreated plots, whilst treatment including spring sprays produced largest canopies. Bristol had a greater proportion of canopy diseased than Capitol, except in the routine treatment. Treatments with spring applications had less diseased canopy area than those with only autumn sprays. For Capitol, fungicide treatment had little effect on the proportion of canopy diseased. Light interception results also showed differences (P<0.001) due to cultivar and fungicide treatment, with a significant cultivar x fungicide interaction (P=0.019). Capitol intercepted more light than Bristol. Untreated Bristol intercepted least light (72%); routine spraying gave the highest light interception (92%), close to that for routine-sprayed Capitol (94%). Fungicide treatment did not affect light interception in Capitol, but light interception by Bristol was better in sprayed than unsprayed treatments and in treatments including spring applications.

Capitol yielded more than Bristol. Routine treatment gave greatest yield increases for both Bristol and Capitol (+ 1.93 and + 1.29 t ha^{-1}, respectively). Other spray treatments gave increases from 0.36 – 1.25 and 0.26 – 0.89 t ha^{-1} for Bristol and Capitol, respectively. Greatest yield increases came from treatments including full or half-rate applications in spring (March-April). On average, disease control by autumn-spraying increased yields by 0.41 and 0.37 t ha^{-1} for Bristol and Capitol, respectively, and the spring application by 0.53 and 0.43 t ha^{-1}. Some PGR effects were noted, with shortening of plants in plots that received full rate applications in March and April. Full control of disease by routine spraying yielded a further 0.99 and 0.49 t ha^{-1} compared to the autumn/spring split applications for Bristol and Capitol, respectively, although this could also include some PGR effects. Regression analysis showed yield was positively related to canopy size (Figure1) and radiation interception. Canopy size accounted for 60% of the variance in yield, according to the equation Yield (t ha^{-1}) = 0.49*(Pod canopy GAI in June) + 1.88.

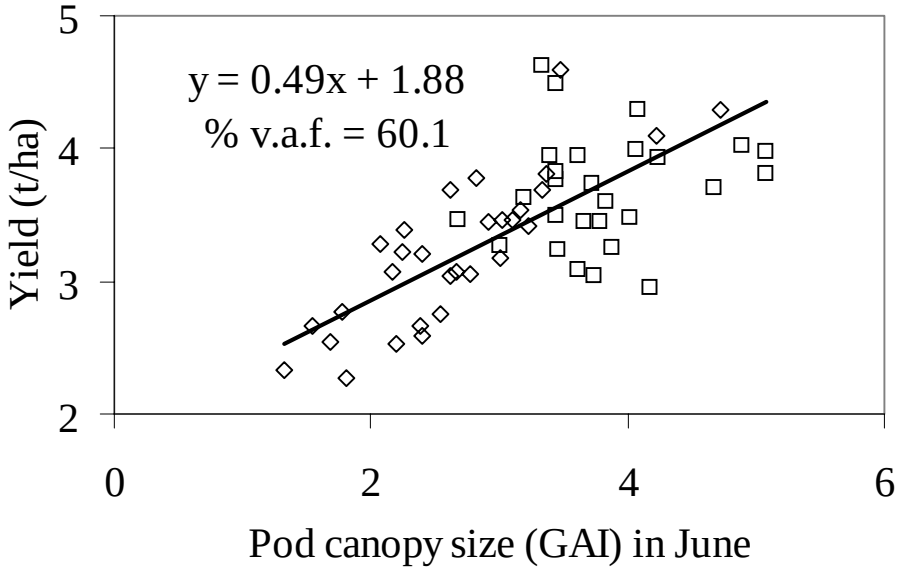

Figure 1: Relationship between pod canopy size (green area index) in June and yield (t ha^{-1} @ 90% DM) of winter oilseed rape (cvs Bristol ◊, Capitol □) at Rothamsted in 1997/98.

DISCUSSION

This work confirms that light leaf spot can cause yield losses >1 t ha^{-1} in resistant cultivars and up to 2 t ha^{-1} in susceptible cultivars. No evidence for yield loss due to winter kill was found in this experiment, although this can be a factor in severe conditions (Baierl *et al.*, 2002). There was evidence that the main impact of LLS infection on yield was to reduce pod canopy size and thus light interception, reducing numbers of pods and seeds and assimilate availability for seed filling. The effect on canopy size was greater on the susceptible cultivar Bristol than on the resistant cultivar Capitol, and Bristol was thus more responsive to fungicides. With complete control of disease by routine fungicide application, canopy GAI could be almost doubled compared to untreated controls, with >15% extra incident light intercepted. There were also differences in the levels of disease on the pods that could have affected yield by reducing pod photosynthesis during pod filling. Although fungicide sprays in autumn delayed onset of LLS infection and reduced maximum leaf area affected, strategies involving spring applications appeared to give better control at the critical phases, increasing canopy size and reducing pod infection. However, plant-growth-regulating effects could not be separated from disease effects in this experiment.

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