Understanding Field Resistance Mechanisms for Improved Control of *Septoria tritici*

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Abstract

Field resistance can contribute to reductions in epidemic progress of *Septoria tritici* in winter wheat. Crop growth and canopy architecture are identified as key mechanisms of field resistance. Near isogenic lines differing for dwarfing (*Rht*) and photoperiod sensitivity (*Ppd*) genes, enabled the study of target traits in a constant background of genetic resistance. Epidemics were consistently earlier in shorter crops across a range of environments. The mechanism for this effect was identified as an increase in spore arrival due to a reduction in the required dispersal distance between infective tissue and emerging upper culm leaves.

Keywords: *Septoria tritici*; winter wheat; dwarfing genes; photoperiod sensitivity; disease escape; disease control

INTRODUCTION

Leaf blotch (*Septoria tritici*) is the most serious foliar pathogen of winter wheat in Western Europe (POLLEY & THOMAS 1991). Severe yield loss is associated with epidemics occurring on the upper canopy leaves. Inoculum of *S. tritici*, occurring on basal leaves, is spread to the top of the canopy by splashy rainfall. This mechanism of dispersal is commonly accepted to be a key limit on disease progress (SHAW & ROYLE 1993). Quantitative host resistance is incomplete, so strong reliance is placed on the use of fungicides to control disease. However, some cultivars that have low genetic resistance sometimes exhibit less disease than resistant varieties, suggesting a 'field resistance' effect. A relationship between *S. tritici* severity and final plant height has been shown by several authors (e.g. TAVELLA 1978). This relationship has often been attributed to a possible linkage or pleiotropy of the genes controlling height and resistance. An alternative explanation could be the presence of a disease escape mechanism. Crop growth, canopy architecture, and rates of stem elongation and leaf emergence, have been shown to affect the risk of inoculum transfer onto the upper leaves (LOVELL *et al.* 1997a). However, quantification of the effect of these traits on disease progress was confounded by differences in genetic resistance of the cultivars studied. This paper describes work to identify and quantify mechanisms of field resistance, in order to assist improvement of crop phenotypes through breeding or agronomy.

MATERIALS AND METHODS

Near isogenic lines of winter wheat cultivars Mercia, Huntsman and Cappelle-Deprez, which varied for genes conferring different magnitudes of crop dwarfing or photoperiod response, were grown in replicated field experiments at three sites (ADAS Rosemaund, ADAS High Mowthorpe and LARS-Long

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Ashton), in England. Measurements of crop growth and structure (LOVELL *et al*. 1997a) and proximity of infectious lesions to emerging culm leaves (LOVELL *et al*. 1997b) were recorded twice weekly from the start of stem extension. Foliar disease was assessed weekly on 10 randomly selected main tillers per plot. The proportional areas of leaf lamina covered with pycnidia of *S. tritici*, and green leaf were estimated. Non target diseases were controlled with fungicides with no significant activity against *Septoria*.

RESULTS

***Rht* genes**. Compared to the commercial cultivar (*rht, ppd*), dwarfing genes reduced the rate of stem extension (RoSE, cm/day-degree). Within sites, rate reductions associated with *Rht* genes were similar across the three cultivar backgrounds (Table 1). Whilst there were variations in both RoSE and final plant height across sites, proportional reductions were broadly maintained. At all sites, distance between infectious lesions and the emerging leaves was significantly reduced in the shorter dwarfed lines throughout the period of stem extension (Figure 1). Dwarfing caused earlier development of epidemics (Figure 2). The effect of dwarfing on the relative magnitude of epidemics on the final three leaf layers was found to be consistent across sites.

***Ppd* genes**. Photoperiod sensitivity affected emergence dates of upper culm leaves. Whilst differences in phenotype were consistent between the two varietal backgrounds, they were not consistent between sites, presumably due to differences in sowing date and thermal time accumulation, factors that would affect vernalisation. At Long Ashton, crop maturation was advanced by approximately 200 and 100 degree days for *Ppd*1 and *Ppd*2 respectively compared to the commercial (*ppd*) line. These thermal time differences coincide with the phyllochron, so that *ppd* lines were one and two leaves behind *Ppd*2 and *Ppd1*,

Table 1. Rate of stem extension (cm/day-degree) for field grown near isogenic lines of winter wheat sited at Long Ashton, UK in 2000. Figures in brackets show rate relative to the non-dwarf commercial background

Background	*rht, ppd*	*Rht*1	*Rht*2	*Rht*3	*Rht*10	*Ppd*1	*Ppd*2
Mercia	0.135	NA	0.103 (76%)	0.051 (38%)	0.033 (24%)	0.123 (91%)	0.136 (101%)
Huntsman	0.168	NA	0.110 (65%)	0.070 (42%)	NA	NA	NA
C. Deprez	0.155	0.126 (81%)	NA	0.061 (39%)	NA	0.134 (87%)	0.155 (100%)

NA = not applicable (lines not tested)

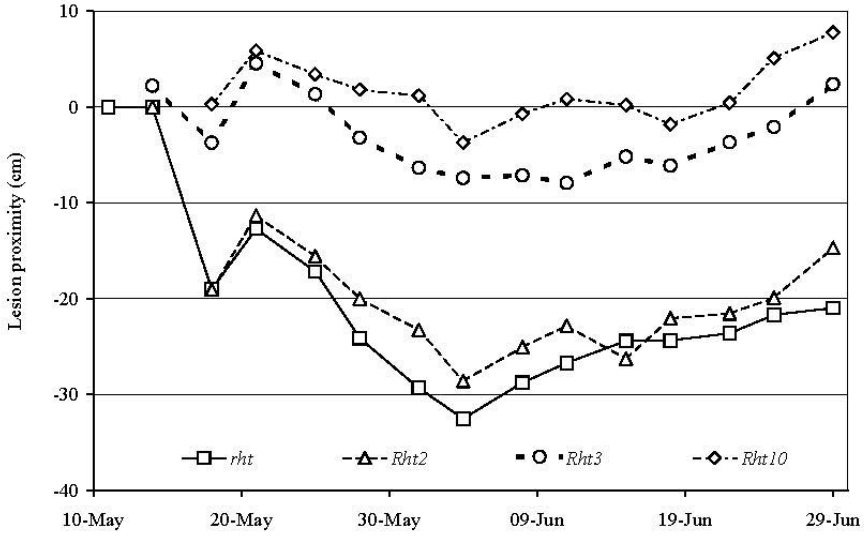

Figure 1. Mean vertical distance between emerging leaves and highest sporulating lesions, for near isogenic lines of cultivar Mercia sited at High Mowthorpe in 1999, during the period of stem extension. Negative values indicate that inoculum sources are at a vertical distance below the emerging leaf

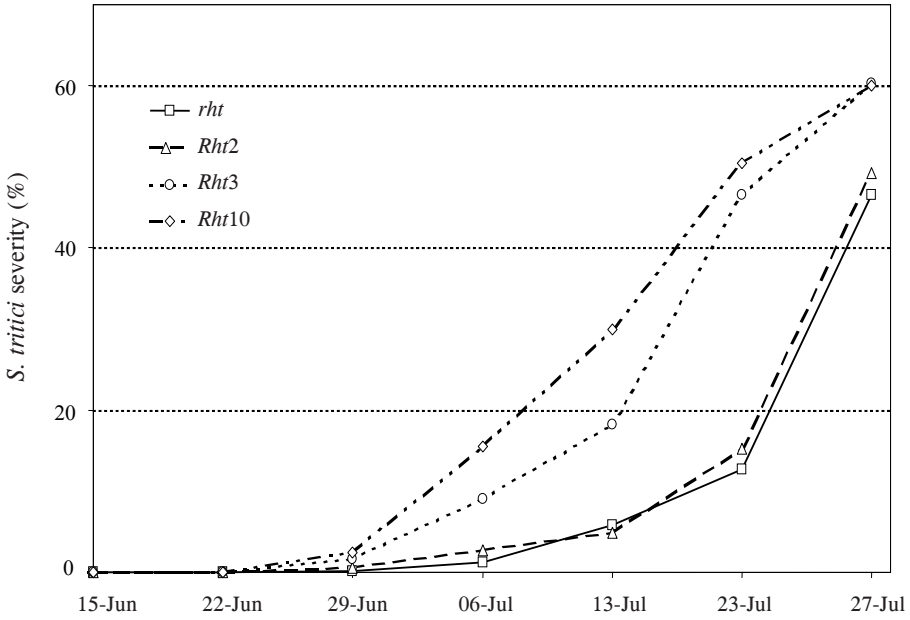

Figure 2. Progress of *S. tritici* epidemics on near isogenic lines of Mercia at High Mowthorpe 1999

respectively. Therefore, the rainfall pattern during the period of emergence and expansion of the upper three leaves differed considerably between lines (Figure 3). A reduction in RoSE of approximately 10% was detected for lines with *Ppd*1 when compared to both *ppd* and *Ppd*2 lines. Disease progress on individual leaf layers differed between lines with epidemics starting earlier on the more advanced lines.

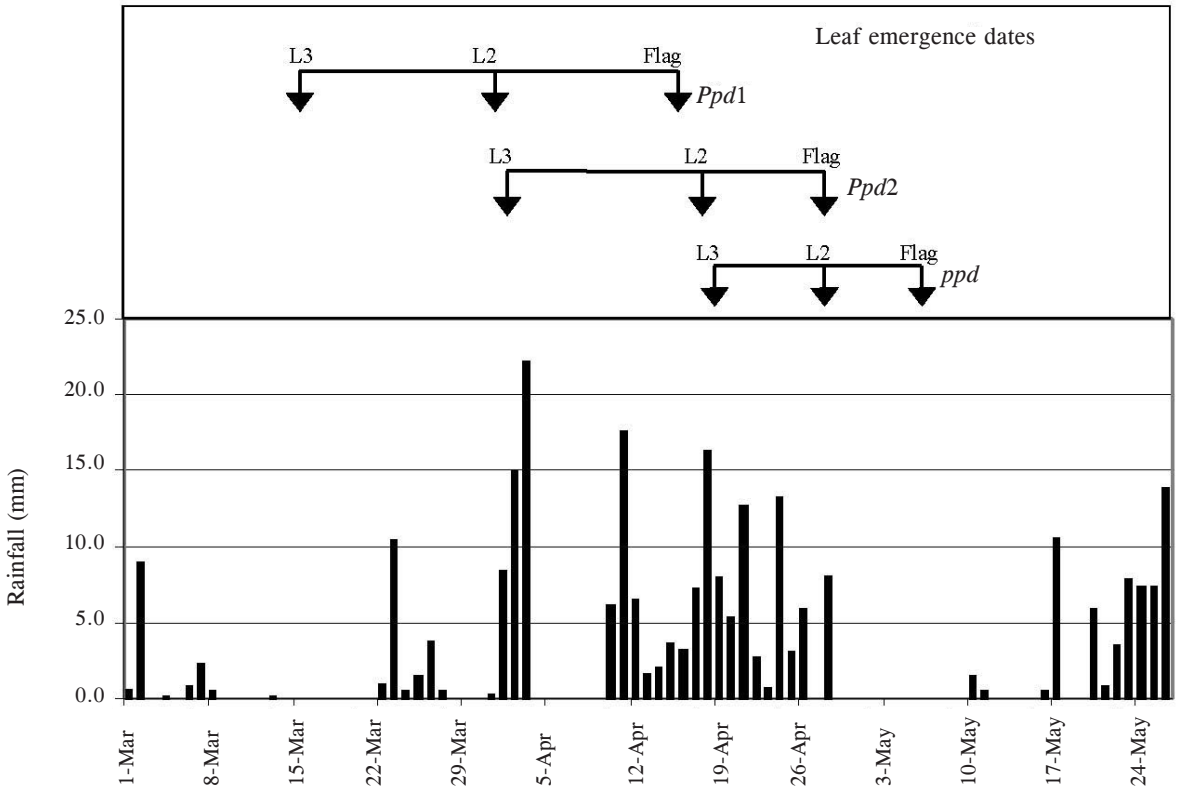

Figure 3. Rainfall pattern and crop development, at Long Ashton, for near isogenic lines of Mercia that differed for photoperiod sensitivity genes. Arrows indicate the approximate date of leaf emergence, identified as the first visual sighting of the leaf tip

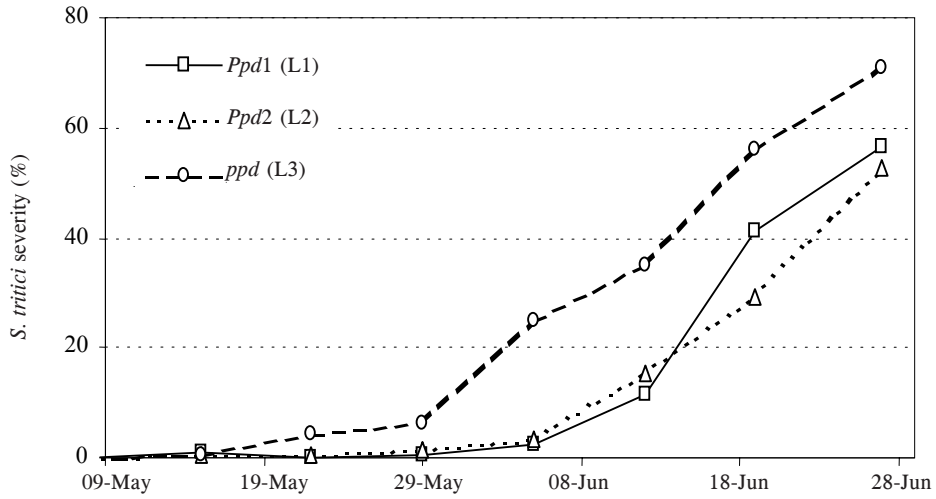

Figure 4. Progress of *S. tritici* epidemics, at Long Ashton, on isogenic lines of cultivar Mercia that differed for photoperiod sensitivity genes (*ppd*). Leaf layers shown differ between lines, *Ppd*1 (Flag leaf), *Ppd*2 (Leaf 2) and *ppd* (Leaf 3), but emerged at similar dates and were therefore exposed to the same environmental conditions during their development

Comparing epidemics on leaf layers that emerged at approximately the same date at Long Ashton, and thus shared the same environmental conditions, revealed that disease progress was earlier on the *ppd* line and that both *Ppd*1 and *Ppd*2 lines had similar epidemics (Figure 4).

DISCUSSION

Resistance to *Septoria tritici* is expressed in field crops through the combined effects of disease escape and resistance. In this context, escape is defined as a reduction in spore transfer to the upper canopy leaves. Due to the long latent period of the pathogen, the upper canopy leaves often grow away from inoculum located on basal leaves during stem extension. However, in lines with dwarfing genes, the rate of stem extension is reduced and therefore the distance of separation between inoculum sources and the emerging upper canopy leaves (inoculum proximity) is reduced. In the case of the semi-dwarf lines (*Rht*1 and *Rht*2) this reduction causes a small increase in eventual disease. However, for dwarf lines (*Rht*3 and *Rht*10) lesions were often observed above the height of the emerging leaves so that rainsplash was unnecessary for dispersal of spores to the emerging leaves. Consequently epidemics were earlier and more severe than in the non-dwarf lines. The maximum displacement of epidemics was of the order of one latent period, an effect similar in magnitude to that given by a well-timed application of triazole fungicide (PAVELEY *et al.* 2000). Consistency of this effect across sites/seasons suggest that there is no major interaction between the environment and disease escape conferred by crop height.

Photoperiod (*Ppd*) genes had a large effect on the maturity of the crop and thus on the environmental conditions to which leaves were exposed to during their emergence and expansion. The rainfall pattern during the period of leaf emergence, a time at when the leaf is generally closest to inoculum sources, is critical to determining the epidemic progress on a given leaf layer. Disease severity on a given leaf layer was generally higher on the more mature lines due to the longer period of exposure. It is common, when analysing severity data from crops that differ for maturity, to adjust for this effect by regressing data with date of heading (e.g. VAN BEUNINGEN & KOHLI 1990). However, whilst this method can aid identification of resistance when comparing large numbers of lines, it may only be appropriate if infection conditions are either constant over time or vary progressively (rainfall seasonality). Rainfall patterns within these studies were sporadic, therefore, we compared epidemics on leaves that shared the same environmental conditions during the period of emergence and expansion. Using this method, comparisons of the top three leaf layers at Long Ashton revealed that disease progress was more advanced on the later maturing *ppd* line, but that epidemics on *Ppd*1 and *Ppd*2 lines were similar. These differences could possibly be

explained by differences in height between the lines at the time of leaf emergence. Although the *Ppd*1 line was more advanced than *Ppd*2, leaves compared were at approximately the same height due to the dwarfing affect of the *Ppd*1 gene. However, the *ppd* line was approximately 15 cm shorter, increasing the likelihood of spore arrival on newly emerged leaves. The relative differences in epidemics, between these lines, was consistent over the upper canopy leaves and was similar to that expressed between dwarf and non-dwarf lines.

Plant breeders have tended to select for shorter crops because they provide benefits to biomass partitioning and spikelet fertility, and have a reduced risk of lodging. However, in addition to increases in *S. tritici*, there is evidence that ear infections by *Stagonospora nodorum* and *Fusarium* spp. may also be increased on shorter crops. Prospects for rational crop design therefore depend upon the development of quantitative frameworks to balance trade-offs between field resistance against disease, lodging resistance and yield, when defining crop ideotypes (DONALD 1968).

The apparent lack of an interaction between height and environment, with respect to the effect on epidemic development, suggests the effects measured here may be consistent across a wide geographical range. However, defining the benefits for changing crop maturity date is more difficult. In regions where rainfall is seasonal, later maturing crops may escape disease if rainfall decreases through the latter parts of the season. For the UK, where rainfall is sporadic and evenly distributed through the season, it is unlikely that differences in maturity, of 1–2 weeks, will provide a consistent and significant effect on epidemic progress.

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