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Determinants of Fungicide Spray Decisions for Wheat*

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Abstract: Yield responses to the application of foliar fungicides to wheat are highly variable. As the cost of treatment is known, some method of estimating future yield response is required if treatments are to be applied only when the value of the yield benefit will exceed the cost. Treatment decisions are often supported by disease thresholds, either formalised or developed by crop managers through practical experience. In farm practice, the proportion of yield that is due to fungicide treatment is not usually known, so the success of a spray decision is often judged by the level of disease later in the season. This paper presents data from field experiments in 1994 and 1995, showing that variation in the current level of visible disease (yellow rust and *Septoria tritici*) explains little of the variation in future effects of the disease on the host, and that the yield effect of a unit disease is not constant across sites and seasons. These findings suggest that traditional disease thresholds may be unreliable predictors of the need to spray and that estimating the success of a treatment decision by observing disease levels later in the season is prone to error. A 'negative threshold', defined as that level of disease below which an economically damaging epidemic cannot develop within a known time-period, may be more reliable. Below the negative threshold, treatment is not required. Above the negative threshold, other factors affecting the rate of epidemic development and sensitivity of the host to green leaf area loss need to be considered, in order to quantify the need for treatment. Measurements which reflect the crop's ability to intercept solar radiation may prove more reliable tools than percentage disease for judging the success of treatment decisions and, experimentally, for quantifying the effect of variation in risk determinants.

Key words: wheat, disease threshold, fungicide, decision

1 INTRODUCTION

In farm practice, decisions on foliar disease management are largely dependent on intuitive judgement of the predicted economic outcomes from a range of possible fungicide treatment inputs. These decisions imply that judgements are being made both about future disease development either if no action is taken or following different inputs, and about the effect on yield of those future disease levels. In making these judgements, growers, consultants and technical representatives pay attention to certain indicators of the current state of the crop and disease. Over many years of shared collective experience, these indicators have become enshrined in crop management lore, as useful guides to the need for fungicide treatment, and might be termed 'determinants of spraying decisions'. In some cases, these determinants have been formalised through research and their mechanisms explained: for example, the rainfall criteria for

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spread of *Septoria tritici* Rob. and Desm. spores;[1] and the definition of treatment thresholds for the rusts (*Puccinia striiformis* Westend. and *P. recondita* Rob. and Desm.) and powdery mildew (*Erysiphe graminis* D.C.).[2] More recently, considerable resource has been devoted to the development of immunoassay and nucleic acid diagnostic techniques[3] to detect and/or quantify visible or latent infections. A threshold—be it either an informal assessment of disease during crop walking or a formalised incidence level or diagnostic test output—attempts to define a level above which the yield response to control will economically justify treatment. The underlying assumptions are that measures of current disease explain a useful proportion of the variation in future disease development, and that the yield response from the control of a unit amount of that future disease will be reasonably consistent across sites and seasons. Credibility has been lent to the latter by the publication of disease/yield-loss equations for the major foliar diseases of wheat.[1,4] This paper reports results from work to quantify and explain variation in yield response to fungicide treatment, and test the assumptions described above.

2 VARIATION IN YIELD RESPONSE TO FUNGICIDE TREATMENT

Comparisons of fungicide-treated and untreated yields from field experiments show substantial variation in the magnitude of response. Figure 1 shows a frequency distribution of yield response to broad-spectrum foliar fungicide application. The data come from 14 randomised and replicated field experiments (similar to those reported by Clark[5]) conducted between 1991 and 1993. At each site, treatments were applied to four cultivars of winter wheat (Riband, Beaver, Hereward and Pastiche) in a factorial design, as one-, two- or three-spray programmes at growth stages (GS) 31–32, GS 39 and GS 59.[6] Responses to an application at GS 39 were measured against untreated plots, whilst those for GS 31–32 and GS 59 were measured by subtracting the yield of treatments including those spray timings from identical programmes without. Responses to a single spray timing varied between zero and 1·76 t ha^{-1}.

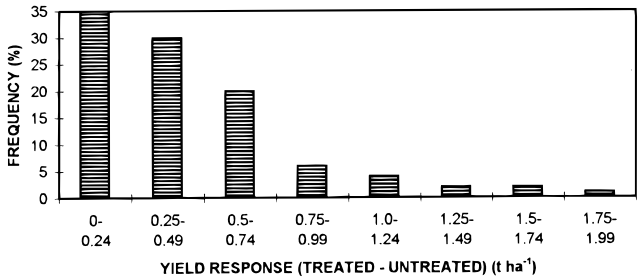

Fig. 1. Frequency distribution for yield responses to a single fungicide treatment, across a range of cultivars, sites, seasons and spray timings.

As the cost of a single fungicide treatment is approximately equivalent to the value of 0·25 t ha^{-1} of wheat, a 'treat or do-not-treat', decision requires prediction of which side of the economic response line that particular crop will fall for an application at that point in time. In commercial practice, fungicide dose is now routinely reduced from that recommended on the label.[7] If choices of appropriate dose[8] are to be made, finer judgements of response are required, as a decision to reduce the applied dose by say 25% is only economically justified if the yield response will not be reduced by more than 0·06 t ha^{-1} as a result.

In applied agronomy experiments, yield–response variation is often reduced by measuring responses to fungicide application on a specific cultivar at a specific growth stage. For example, selecting data from the set used in Fig. 1 for two cultivars of contrasting genetic resistance to disease[9] and one application timing (GS 59) reduces the range of variation (Fig. 2). This approach has value as a guide to decision making as it shows that (grain quality considerations aside), on average it is profitable to treat the disease-susceptible cultivar Beaver at GS 59 but not the more resistant cultivar Hereward. However, Fig. 2 shows that such 'crop management by averages' would result in 28% of Beaver crops being treated unnecessarily (28% of crops giving a yield response less than 0·25 t ha^{-1}) and 29% of Hereward crops not being treated, when on economic grounds they required disease protection (29% of crops giving a yield response greater than 0·25 t ha^{-1}).

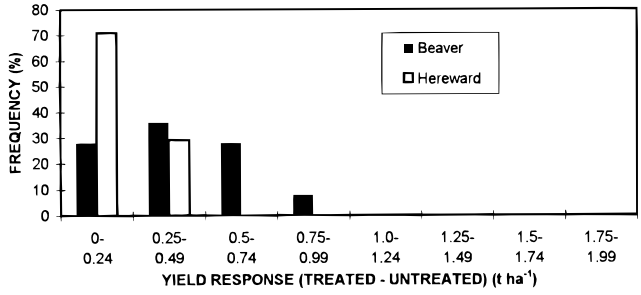

Fig. 2. Frequency distribution for yield responses to fungicide treatment at GS 59, on two cultivars of contrasting disease resistance.

For a given cultivar and spray-timing combination, variation in response to fungicide treatment arises through site and seasonal variation in disease severity[10] and the physiological state of the crop.[11,12] Crop managers aim to predict some of this variation through observation of the crop and disease.

3 CROP WALKING AND DISEASE THRESHOLDS

As fungicides are used to control disease, it seems inherently logical to monitor disease in crops, initially as a

guide to the need for treatment, and later as a measure of the success of that treatment. Post-treatment monitoring of crops forms an important part of the learning process. High levels of disease observed post-treatment may lead a crop manager to conclude that the fungicide programme applied was inadequate, whereas crops with low levels of disease are taken to indicate appropriate decisions, or excessive input. Many iterations of this learning loop enable crop managers to refine their responses to a wide range of crop and disease situations. The learning process is reinforced by sharing such experiences in discussion groups, and is particularly important because of the difficulty, in commercial practice, of associating site and seasonal variation in yield with disease-management decisions.

Several attempts have been made to formalise judgements on the need for fungicide treatment by defining thresholds. Some, such as those that form the basis of EPIPRE,[13] have been defined by fitting disease progress curves to sets of experimental data and relating the predicted area under the disease progress curve (AUDPC) to yield loss. Others have been defined by empirical interpretation of experimental data and consensus within an expert group, or by a combination of such approaches.[2,14] The practical application of much epidemiological theory, which could aid the process of threshold definition, has been inhibited by poor communication between mathematical modellers and applied pathologists. The common practice of expressing disease as percentage values of an unknown leaf area, rather than quantifying disease epidemics in relation to absolute values of crop canopy growth, may also have delayed progress.

The costs associated with crop monitoring to support the use of formalised or intuitive thresholds can be offset through more efficient crop management if: (i) current levels of disease explain a useful proportion of the variation in future disease severity, and (ii) future disease severity explains a useful proportion of the variation in yield loss (and hence response to treatment). If the former is not the case, the validity of thresholds is challenged. If the latter is not the case, the learning process described above is flawed.

4 TIMING OF TREATMENT DECISIONS

Figure 3, derived from data reported by Paveley *et al.*,[8] shows the relationship between the timing of a single fungicide application, dose (of active ingredient (AI) propiconazole, as the commercial product (CP) 'Tilt 250 EC', Ciba Agriculture), and the *Septoria tritici* AUDPC for the flag leaf of a crop of winter wheat, cultivar Riband. Dose is expressed as a proportion of the label recommended dose (0·5 litre ha^{-1} CP), and application timings 1 to 8 are at weekly intervals from 1 May 1991. For an individual leaf layer, the most effective disease

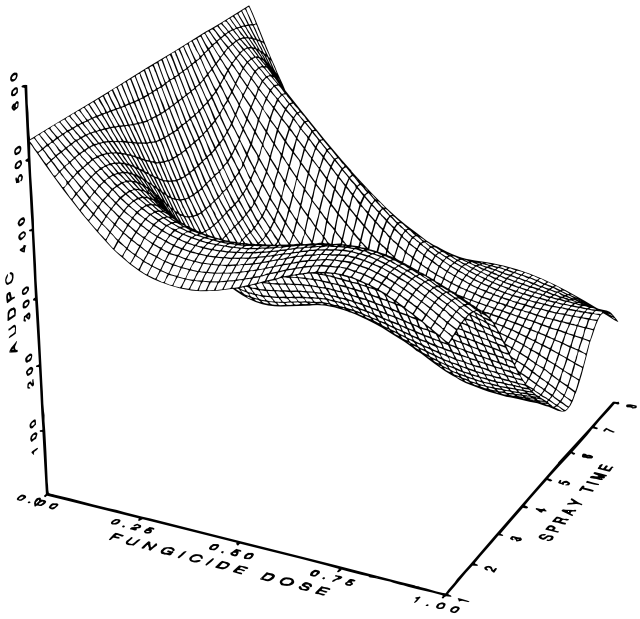

Fig. 3. Surface response for *Septoria tritici* AUDPC against fungicide dose and timing of application.

control at the lowest dose can only be obtained for a relatively short period after its emergence date. In the case of the flag leaf shown in Fig. 3, which was fully emerged at spray time 4 (22 May), optimum control was obtained around spray time 5 (29 May).

If similar response surfaces are drawn for successive leaf layers down the canopy, the optimal control timing, indicated by the valley in the surface, moves earlier along the spray–time axis at intervals roughly equivalent to the leaf emergence rate or 'phyllochron',[15] although the exact timing may be modified by the timing of rainfall events[16] in relation to leaf emergence. Similar relationships between degree of control and application timing have also been described for yellow rust (*Puccinia striiformis*).[8] Hence, for a given leaf layer, the decision to apply a fungicide needs to be made close to its emergence if the treatment is to be efficient.

The field experiments described below were designed to quantify: (i) the extent to which variation in visible disease severity lower in the canopy at the emergence of a leaf layer explains variation in subsequent disease severity and disease-induced loss of green area on that leaf layer and (ii) site and seasonal variation in disease/yield-loss relationships.

5 MATERIALS AND METHODS

5.1 Design and treatments

Field experiments were conducted at ADAS Terrington, Norfolk (targeting yellow rust), ADAS Rosemaund, Hereford and Bridgwater, Somerset (both targeting *S. tritici*), in each of 1994 and 1995 harvest years.

To generate variation in initial inoculum, field plots (minimum of 252 m^2 each, with four replicates at each inoculum level) of winter wheat (cv. Slejpner and Riband at yellow rust and *S. tritici* sites respectively) were either left untreated (ambient disease), or subjected to introduced inoculum on spreader plants (above ambient) or two levels of DMI fungicide treatment during the winter/early spring (below ambient). No inoculum-suppression fungicide treatment was applied closer than 60 days to the notional start date of the experiment, at which time a bioassay (using artificial inoculation with yellow rust spores) was performed on plants sampled from fungicide-treated and untreated plots, to confirm that the fungicides applied had degraded to inactive levels. Hence, any subsequent differences in epidemic progress could be attributed to differences in inoculum level, not to residues of the treatments applied to create the differences. All disease and green leaf area data presented here are taken from measurements after the bioassay was performed. Inoculum transfer between plots was minimised by separating plots with a minimum of 7 m of winter barley (yellow rust site) or non-susceptible wheat (*S. tritici* sites) guard. Work by Daniel *et al.*[17] suggested that inter-plot interference with yellow rust, unlike mildew, was minimal. Hence, within each experiment, inoculum was varied whilst all other aspects of crop agronomy, such as host resistance and weather, were constant.

To minimise the effects of non-target disease, oversprays of anilazine were applied at the yellow rust sites only. Non-oversprayed sub-plots (minimum 36 m^2, in a split plot design) were assessed as controls to ensure that the overspray was not affecting the natural development of the target disease. In practice, fungicide applications to control non-target diseases were either not required or had no significant effect on development of the target disease, and non-target diseases were present only at low levels, so all untreated data presented here were meaned across oversprayed and non-oversprayed sub-plots. To measure the effect of the target disease on the crop, further sub-plots received a broad-spectrum fungicide at GS 30, GS 32, GS 39 and GS 59 and acted as undiseased controls.

5.2 Disease and green leaf area assessments

Disease severity and percentage green leaf area were assessed on all leaf layers with greater than 25% green leaf area on 10 or 20 randomly selected tillers (*S. tritici* and yellow rust sites respectively) per sub-plot at 10- or 14-day intervals from GS 31 to GS 85 inclusive, using disease assessment keys.[18] Prior to GS 39, leaf layers were identified at each assessment by tiller dissection or leaf tagging, to enable disease and green leaf area progress curves to be plotted for each leaf layer from its emergence to senescence. As part of the standard assessment routine, leaf area was measured on each assessable leaf on two randomly selected tillers per sub-plot at each assessment date, using a grid printed on plasticised card, against which leaf length and width were measured. Leaf area was calculated *via* a form factor.[19] Green leaf area index (GLAI) values, defined as the number of units of planar area of green leaves per unit area of ground that they occupy, were calculated for each assessment date for each leaf layer *via* percentage green leaf area, leaf area per tiller and fertile tiller number per unit ground area. Healthy area duration[20] (HAD) values post-GS 39 were calculated by integration of the area under GLAI progress curves using the trapezoidal rule.

At the *S. tritici* sites, the number of spores per tiller was assessed at GS 31 in untreated sub-plots of each inoculum level main plot, by soaking a known number of tillers, from 20 plants per inoculum treatment, in a known volume of water and counting spores using a haemocytometer.

Plots were harvested by plot combine and grain yields adjusted to 85% dry matter.

6 RESULTS

6.1 Disease/yield-loss relationships

Figure 4 shows the relationships between AUDPC for the upper four leaves and yield loss. Regression equations are shown in Table 1. Although the regressions for each individual experiment are statistically significant and explain up to 95% of yield variance, the slopes vary by almost an order of magnitude between sites and seasons. Relationships between the effect of potential risk variables, such as initial inoculum, and disease-induced loss are likely to be more reliable if the measure of outcome is consistently related to yield. Measurement of AUDPC has shortcomings in this respect. Given that: (i) crop dry matter accumulation is considered to be proportional to intercepted photosynthetically active radiation[21] (PAR), (ii) the proportion of available PAR intercepted is related to the crop's green area index (the number of units of planar area of green leaves, stem and ears per unit area of ground that they occupy = GAI) *via* an equation

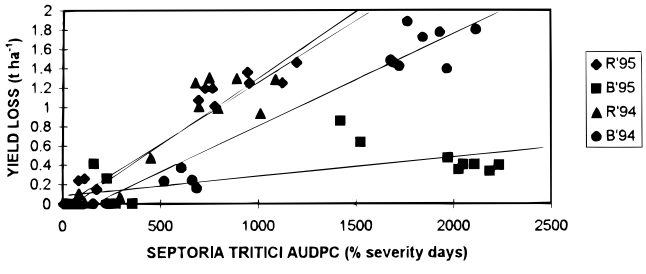

Fig. 4. Relationship between AUDPC and yield loss at Rosemaund (R) and Bridgwater (B), 1994 and 1995.

TABLE 1
Regression Equations for *Septoria tritici* AUDPC against Yield Loss (YL)

Site	*Year*	*Regression equation*	R^2(%)	*Probability*
Rosemaund	1995	YL = 0·0296 + 0·0013AUDPC	95·8	<0·001
Bridgwater	1995	YL = 0·0893 + 0·000184AUDPC	45·0	0.004
Rosemaund	1994	YL = − 0·0679 + 0·001 38AUDPC	88·4	<0·001
Bridgwater	1994	YL = − 0·279 + 0·001 02AUDPC	95·9	<0·001

derived from Beer's Law,[22] and (iii) percentage disease may not be consistently associated with loss of percentage green area, it is less surprising that measures of disease percentage on canopies of unmeasured absolute size relate inconsistently to yield loss. The limitations of measures of disease based on percentage assessments have been discussed in greater depth by Waggoner & Berger,[20] Gaunt[12] and Bryson *et al.*[11]

6.2 Effects of disease on the crop canopy

Figure 5 shows the growth and decline of GLAI of the upper four leaves of the canopy at the Bridgwater site in 1994. Assessment points were at 14-day intervals and the growth stages shown are to the nearest assessment date. Error bars represent 95% confidence limits (12 df). The difference in green area between the two curves represents the effect of disease prevented by the broad-spectrum fungicide programme described in Section 5.1.

The effect of fungicide application is to delay the onset of green area loss. Although fungicides were applied at GS 30 and GS 32, their effects were not expressed until after GS 39. For example, leaves 4 and 3 emerged at approximately GS 31 and 32 respectively, but the effect on yield of fungicides applied at or shortly after their emergence was expressed *via* loss of green leaf area towards the end of their potential eight- to ten-week life, between GS 39 and 77. Hence the treatment decision at the start of a leaf layer's life is temporally displaced from the expression of effect of that treatment, particularly when controlling the longer latent-period diseases such as *S. tritici.*

Taking the GLAI curves for the upper four leaves and integrating the area beneath them over time, from GS 39 onwards, provides a measure of the light-intercepting surface of the canopy, which has been termed the healthy area duration (HAD) by Waggoner & Berger.[20] R^2 values from Figs 6 and 7 suggest a relationship between HAD loss due to disease and yield loss that is consistent across sites and seasons and explains 80% or more of yield-loss variation. HAD loss and yield loss were calculated by subtracting untreated values from fungicide-treated values within each inoculum level at each site.

A key question therefore is: to what extent does variation in visible disease on older leaves at the emergence of a new leaf layer explain variation in the disease-induced HAD loss on the new leaf layer that will be suffered in the absence of treatment?

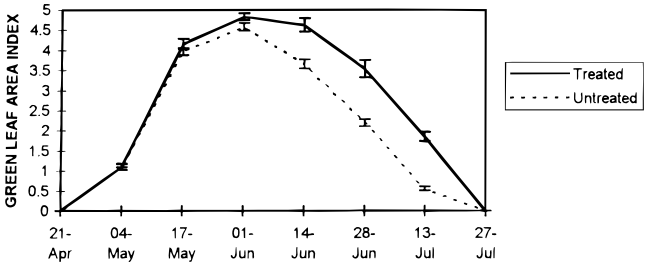

Fig. 5. Green leaf area growth and senescence (leaves 1 to 4) for fungicide-treated and untreated plots at Bridgwater, 1994. Growth stages 31, 32, 33, 39, 59, 71, 77 and 87 on dates 21 April to 27 respectively.

6.3 Inoculum–HAD loss relationships

To generate the data for Fig. 8, disease that would be visible during crop inspection at the emergence dates for each of the upper four leaf layers was quantified by

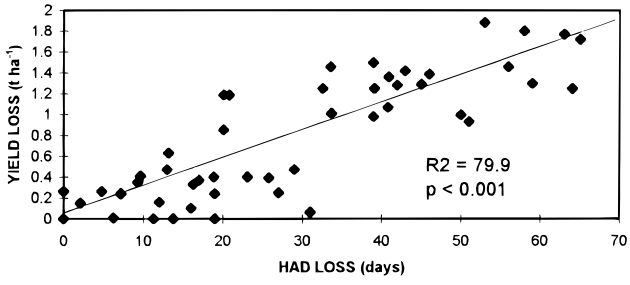

Fig. 6. Relationship of healthy area duration (HAD) loss to yield loss at Rosemaund and Bridgwater, 1994 and 1995.

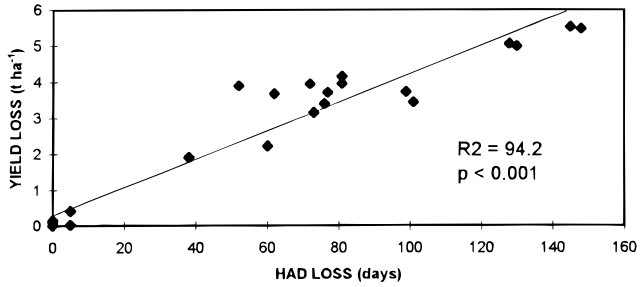

Fig. 7. Relationship of HAD loss to yield loss at Terrington, 1994 and 1995.

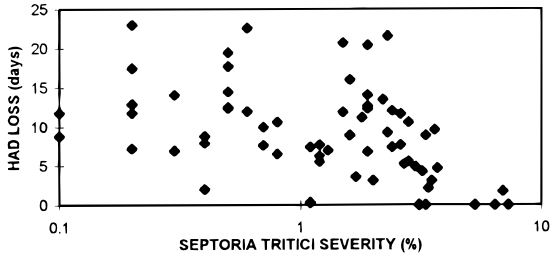

Fig. 8. Mean disease severity on all assessable leaf layers at the emergence dates for each of leaves 1 to 4 against HAD loss on each of leaves 1 to 4, Rosemaund and Bridgwater, 1994 and 1995.

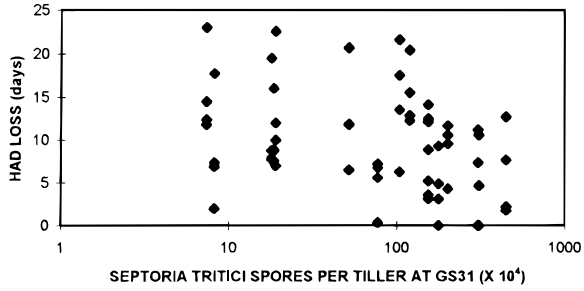

Fig. 9. Spores per tiller at GS 31 against HAD loss for each of leaves 1 to 4. Rosemaund and Bridgwater, 1994 and 1995.

calculating a mean severity figure across all the assessable leaf layers (i.e. the new leaf layer and the older leaf layers below it) present at each date. The disease present at the emergence of each new leaf layer was plotted as a scatter diagram against the HAD loss for that leaf layer, in each inoculum treatment at each site. Hence the location of one point on the graph is defined on the *y*-axis as the HAD loss (fungicide-treated minus untreated) suffered by one leaf layer throughout its life, within one inoculum level, at one site, in one season, and on the *x*-axis as the mean percentage disease severity across all assessable leaf layers, measured on the emergence date of that leaf layer, within one inoculum level, at one site, in one season. The highest *x*-axis values appear artificially low, as the means include leaf layers still within one pathogen latent period of their emergence date and hence showing no symptoms. Across the sites, seasons and leaf emergence dates tested, the range of disease severities embraced the range of normal experience in the UK.

6.3.1 Septoria tritici

Within the range of severities tested, there was no evidence that increasing levels of inoculum at leaf emergence were associated with an increase in subsequent disease-induced loss of GLAI.

One hypothesis to explain the lack of a relationship would be that viable spores on senesced leaf layers lower down the canopy were providing a viable inoculum source, the size of which may be unrelated to the severity of visible disease symptoms. To test this, spore numbers per tiller at GS 31 were plotted against HAD loss for each of leaves 1 to 4, in each inoculum treatment at each site (Fig. 9).

Within the range tested, initial inoculum in the spring did not predict the disease-induced HAD loss on the upper canopy. Regression analysis of the data in Figs 8 and 9 suggested that little of the variation in HAD loss was explained by variation in percentage disease at emergence or spores per tiller (R^2 values of 27% and 15% respectively), and negative relationships were suggested—contrary to expectation.

Low levels of visible disease were not always associated with low spore numbers. For example, a visual assessment of one treatment at the time of sampling for spore counting gave a mean severity of less than 0·2% *S. tritici*, associated with 190 000 spores per tiller.

6.3.2 Yellow rust

The yellow rust data presented in Fig. 10 show the mean severity across all the assessable leaf layers present at the emergence date for each of the upper four leaves, against the HAD loss for that leaf layer, in each inoculum treatment at each site (as for the *S. tritici*, Fig. 8 above).

At very low levels (mean severities less than 0·01%), inoculum appears to limit loss of HAD. At higher inoculum levels, HAD loss may or may not be high, dependent on other variables. In the most severe disease cases, over 60% of the total HAD of the upper four leaves was lost. HAD loss was not zero at zero severity. This may be explained by inoculum not being zero, but simply latent or below the sampling detection limit, or influx of spores from outside the plots.

Disease progress data for yellow rust have shown that, even on a susceptible cultivar, relative growth rates (r of Vanderplank[23]) measured on an individual leaf do not on average exceed 0·1 unit^{-1} day^{-1} under typical conditions in a disease-susceptible part of the country (Paveley, N. D., unpublished), which agrees with Zadoks' corrected r values.[24] If $r = 0{\cdot}1$, with 'compound interest', the disease will increase in severity by one order of magnitude every 25 days. Hence, over the typical 56-day life of a leaf layer, the disease has the potential to increase by just over two orders of magnitude. This supports the data shown in Fig. 10, as an

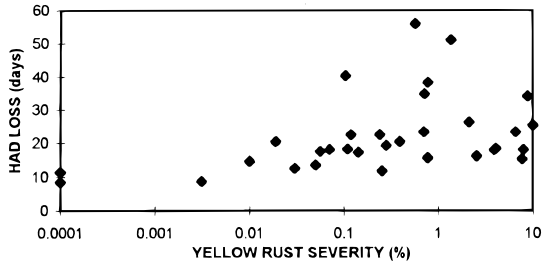

Fig. 10. Mean disease severity on all assessable leaf layers at the emergence dates for each of leaves 1 to 4 against HAD loss on each of leaves 1 to 4, Terrington, 1994 and 1995.

initial disease level of 0·1% increasing to 10% during a leaf's life could readily cause substantial damage to green area, whereas an initial level of 0·001% increasing to 0·1 would be unlikely to. Variation in r on a single cultivar ranging from 0·02 to (exceptionally) 0·2 (Paveley, N. D., unpublished), and earlier work on weather–disease relationships[25,26] and crop nutrition (Bryson, R. J., pers. comm.) suggest that variations in weather and host nutrition are predominantly responsible for the wide variation of outcomes, on one variety, where inoculum is not limiting.

Given further supporting data, a 'negative threshold' might be set. The definition of a negative threshold would be that level of disease below which an economically damaging epidemic cannot develop on the newly emerged leaf layer. Below the negative threshold, treatment would not be required and further monitoring would not be needed until the decision point at the emergence of the next leaf layer. Above the negative threshold, other factors affecting the rate of epidemic development (such as weather and nutrition) and sensitivity of the host to green leaf area loss would be considered, in order to better predict the need for treatment.

The critical influence of such low levels of disease in determining future disease progress raises sampling issues for field experimentation and crop walking. Experimentally, yellow rust pustules occupying 0·5% of a leaf's area are readily detectable during destructively sampled disease assessments (e.g. a 5 mm^2 pustule on a wheat leaf of 1000 mm^2). In the experiments reported here, the level of sampling, replication and assessable leaves per tiller meant that approximately 500 leaves were assessed for each point on the scatter diagram. Hence 0·001% must be considered the absolute limit of potential assessment sensitivity. During crop walking, substantially larger numbers of leaves can be scanned quickly, at the expense of ability to detect small lesions.

7 CONCLUSIONS

7.1 Measuring the effect of potential spray determinants and fungicide treatments

Measurements of percentage disease severity provide an inconsistent indicator of yield loss, even when integrated over time. In work reported by Thomas *et al.*[1] from four field experiments, the slopes of the steepest *S. tritici* disease/yield-loss regressions were 24% and 133% greater than the shallowest for leaves 2 and 3 respectively. Greater variation has been reported by Bryson *et al.*[11] for yellow rust, and here for both pathogens. Such variability is not surprising. For percentage disease to relate consistently to yield loss, subjective error in assessment of percentage disease would need to be small, percentage disease would need to relate consistently to percentage green leaf area (GLA) loss, and percentage GLA loss would need to relate consistently to the absolute loss of the light-intercepting green leaf surface (GLAI). Work by Parker *et al.*[27] suggests that the first of these requirements is unlikely to be satisfied, and the difficulty of differentiating necrosis due to disease from that caused by natural senescence compounds the error. Visual assessments of percentage green leaf area, from which GLAI may be calculated *via* measures of total leaf area index, may be less error-prone in this respect. More fundamentally, percentage loss of GLA cannot relate consistently to absolute GLAI loss, given that peak GLAIs for winter wheat crops in the UK can vary more than three-fold (Sylvester-Bradley, R., unpublished).

In contrast, measures of disease-induced GLAI loss, integrated over time (HAD), provide a measure that appears to relate consistently to yield loss across sites and seasons. HAD may therefore be a useful tool for pathologists who wish to relate variation in potential spray determinants to variation in loss, or assess the appropriateness of treatment decisions by observation of the crop after treatment. Use in farm practice may be limited until convenient techniques for leaf area index measurement are widely available. An area of crop kept free of disease would also be needed, against which comparisons could be made at key points during the season. Further improvements in prediction of yield loss might be obtained, at the cost of greater complexity, by taking account of variation in available PAR between and within sites and seasons, and at different leaf layers within the crop canopy. Separating the time and green leaf area components of HAD might also be beneficial, given that their effects on intercepted radiation are different.

7.2 The inoculum–risk relationship

In 1963, Vanderplank[23] stated that: 'Any reduction in the amount of initial inoculum inevitably reduces the final amount of disease, other things being equal. But other things are not always equal; and the effects of changes in the amount of initial inoculum may be small compared with the effect of other changes'. He observed that with potato blight (*Phytophthora infestans* (Mont.) de Bary) the effect of weather was much more striking than the effect of sanitation to reduce initial inoculum, as the value of r can change so much that it overshadows initial inoculum variation. Diseases capable of high r values are, therefore, less amenable to prediction from measures of current inoculum. Whilst *P. striiformis* and *S. tritici* are not capable of the extraordinary relative growth rates achieved by *Phytophthora infestans*, the data presented here suggest that prediction of future disease-induced green leaf area loss from current disease

severity is prone to sufficient variation for the accuracy of solely threshold-based fungicide treatment decisions to be questioned.

Theory suggests that predictive success will be positively associated with the accuracy of estimation of current inoculum, and negatively related to the extent of variation in *r*, and the time period over which the prediction is made.

7.2.1 Estimation and manipulation of inoculum

For the obligate pathogens, such as *Puccinia striiformis*, it is probably reasonable to assume that, as the visible disease is predominantly sporulating structures, the level of visible disease relates to the currently available inoculum within the crop. The focal nature of yellow rust epidemics, and inter-plot interference studies,[17] suggest that spore movement is predominantly by physical contact between infectious and neighbouring plants. Air-borne spread does occur, but the evidence presented here suggests that, even in a disease-prone part of the country at the peak of the epidemic, amounts of inoculum arriving from outside sources are sufficiently small not to cause substantial damage within the life of one leaf layer.

The quantification of negative thresholds, below which disease could not develop to damaging levels within a known time-period, would define the level of sampling required during crop walking or for diagnostic tests. Practical experience suggests that sampling techniques need to be quick and convenient if they are to be used, but this conflicts with the need to detect low levels of disease reliably.

For *S. tritici*, visible symptoms may relate poorly to available inoculum, as spores remain viable after leaf senescence. Immunoassay or nucleic-acid-based diagnostic tests have the potential to quantify the 'hidden' inoculum, but this may not be the most appropriate way to utilise such technology (see below).

The level of inoculum present at the start of rapid canopy expansion (*c.* GS 31) is highly variable between sites and seasons—in the experiments reported here, differences of two or three orders of magnitude were found. In contrast, manipulation of inoculum by the repeated use of fungicides during the winter generally provided a reduction of less than one order of magnitude. This, and the poor relationship between early inoculum and future damage to the crop, suggests that fungicide sprays during the autumn, winter or early spring to 'reduce inoculum' are likely to be ineffective, a conclusion supported by Thomas *et al.*[1]

7.2.2 Variation in relative growth rate (r)

It is unfortunate that an important period for fungicide decision making (GS 31 to GS 39) coincides with high potential for variation in *r*. During that period, the canopy is expanding rapidly and producing a new leaf layer approximately every 110 degree days.[28] As a result, the proportion of the canopy affected by disease varies both with the *r* of the epidemic and the growth rate of the canopy. Variation, for example in temperature, may alter the balance between epidemic and canopy growth, partly by altering the pathochron, defined by Beresford & Royle[29] as the number of phyllochrons per latent period. Leaf senescence at the base of the canopy sheds obligate pathogens, reducing their infectious period and, hence, progeny : parent ratio (a key determinant of *r*). Also leaf layers become more vertically separated during stem extension, making spore transfer efficiently of *S. tritici* (another key determinant of *r*) more dependent on rainfall events. Such effects probably explain the unexpected negative relationship shown in Figs 8 and 9, as in 1995 inoculum levels were generally higher than in 1994, but rainfall was lower; the effect of the latter outweighing that of the former.

Diagnostic techniques which could detect spores after arrival on a newly emerged leaf layer, or early in the latent phase, might cut out the *r*-varying step/s of spore transfer and infection, and hence improve prediction of future green area loss and the need for treatment.

7.2.3 Period of loss prediction

It is tempting to believe that the period of disease risk prediction required is only that between crop inspections. However, for a given leaf layer, the time period over which fungicides can most efficiently control disease is sufficiently short that, as time progresses following leaf emergence, any improvement in accuracy of prediction is associated with decreased ability to influence the epidemic efficiently by treatment. This process is repeated for each new leaf layer, so that the events that require prediction (HAD loss) always tend to remain one leaf-life ahead.

7.3 Fungicide treatment thresholds

Intuition suggests that a severely diseased crop should receive more protection than one that is only moderately infected. Within the range of data presented here, disease severity proved a poor predictor of future loss. Only at very low levels of disease did inoculum appear to be limiting for yellow rust (in agreement with work by Luo & Zeng[30]), and there is some evidence that levels of *S. tritici* inoculum below the lowest experienced in this work may also be limiting.[16] It may be, therefore, that the relationship between inoculum and risk is on, or close to, its plateau within the range of inoculum levels typically experienced on susceptible cultivars. Intuitive responses may need to be suppressed, particularly at early growth stages, when the crop has the capacity to 'grow away' from the disease.

It has often been difficult to prove an economic advantage from the use of thresholds, when compared

against prophylactic treatments that were appropriate to the cultivar/s used and applied at appropriate growth stages. In the light of the data presented here, pathologists might usefully consider whether this is because treatment thresholds based on severity or incidence on indicator leaf layers give the impression of 'working', not because disease on that leaf relates consistently to the severity of later disease and loss, but simply because one incubation period after emergence of, say, leaf 3 or 2 will typically take the crop to GS 33–39, when yield responses to fungicide application are at their highest.[5] Clearly, the use of a threshold will also tend to pick out correctly the more resistant varieties and reduce their treatment. However, it could be argued that, if the national variety resistance evaluation process (and monitoring of obligate pathogen virulence) is functioning correctly, it should not be necessary to incur the costs of inspecting over 200 000 UK wheat fields in order to confirm what is already known.

Negative thresholds, below which disease cannot develop to damaging levels within a known time interval, may be more reliable than 'traditional' thresholds. When the negative threshold is exceeded, variables controlling r (principally host resistance, nutritional state and weather)[31] and the sensitivity of the host to green leaf area loss could be assessed, to allow informed decisions to be made, whilst avoiding unnecessary fungicide use.

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