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Detection of tolerance of barley cultivars to infection by powdery mildew (*Erysiphe graminis* f.sp. *hordei*)

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Summary

Barley genotypes representing a wide range of resistance expressions and origins, from major resistance genes in modern cultivars to field resistances in landraces, were assessed for tolerance to disease under glasshouse and field conditions. A few genotypes were picked out as showing less yield loss than would be expected from the level of mildew infection. Genotypes showing more than the expected yield loss were also found. The potential use of tolerance as a breeding character is discussed.

Introduction

Tolerance to disease may be defined as the ability of a plant to yield well despite being infected by a level of disease that would normally be expected to cause severe yield loss. However, the term has been used in many contexts (Clarke, 1984; Mussell, 1981). Furthermore, the concept of disease tolerance has been little used as a breeding objective because of the difficulty of assessment, although a breeding programme in Hungary regards it as a selectable breeding character. (A. Mesterhazy, personal communication). Apparent differences in tolerance between genotypes in highly developed crops such as barley can sometimes be attributed to components of genetic resistance to disease development where these are not detected by the disease assessment methods used. Not all components of resistance can be detected by, for example, overall visual rating (Newton, 1990).

Wild plant species are generally tolerant of infection (Tarr, 1972; Ben-Kalio & Clarke, 1979), whereas crop plants suffer severe yield loss from relatively low levels of infection (Chester, 1950; James, 1964). Thus tolerance appears to be inversely related to selection for high yield (Clarke, 1984). This could be associated with more of the plant being used for assimilate production for yield. Where resistance is available, breeders have concentrated upon deploying it as it is generally easy to manipulate. Any tolerance that may have existed in the gene pool would gradually be lost in the same way that 'background' resistance has. Loss of tolerance may be particularly attributable to reductions in chlorophyll levels, rates of photosynthesis, altered translocation pattern and reduction in root:shoot ratio which are all found in crop plants but not in some wild plants (Clarke, 1984). In a balanced ecosystem there is little opportunity for highly adapted pathotypes to develop. Alternatively, tolerance can be regarded as a character or group of characteristics for survival in a balanced ecosystem, many components of which may be deleterious to economically important characteristics including high yield potential.

Yield potential of a cultivar may be inversely related to tolerance as high yield may be attributable at least in part to a higher proportion of assimilates being diverted to grain filling. However, Kosuge (1978) calculates that no more than 1–2% of total host carbohydrate is diverted by tobacco mosaic virus and Hancock & Huisman (1981) calculate that plants should be able to supply nutrients for pathogen growth out of the normal amounts lost due to leaching and that metabolite diversion therefore is unlikely to be associated with differences in tolerance between host genotypes. Furthermore, infection often leads to enhanced photosynthesis rate compensating for any assimilate drain. Much damage caused by pathogens is due to their metabolites

such as enzymes, growth regulators and toxins (Wheeler, 1975) and physical damage rather than diversion of host metabolites (Clarke, 1984). Therefore it is the ability of a host to compensate for loss of photosynthetic ability and thereby assimilate production capacity for yield that must be related to its tolerance capacity.

Detecting tolerance under field conditions requires the use of fungicide applications to obtain comparable control values, leading to possible confusion of tolerance with other effects. Fungicides could exclude the effects of saprophytes that cause induction of resistance reactions which, although not scorable as disease, result in yield reduction from the diversion of assimilates to fuel the resistance response in untreated plants (Smedegaard-Petersen & Stolon, 1981). Fungicides may also exclude the response to induction of resistance from avirulent pathogen inoculum pressure. Thus fungicides confound the effects of removal of infection with induced resistance reactions. Furthermore, fungicides often have stimulatory effects on plant growth (Newton & Thomas, 1987). To be of practical value tolerance must be determined as a field response and assessments must be made under field conditions. Glasshouse conditions can be used, however, both to control resistance induction and infection, and to subject cultivars selected for optimum yield under field conditions to non-optimal conditions.

Little & Doodson (1972) showed a correlation between NIAB disease assessment and yield loss due to disease. Given this correlation, cv. Proctor showed less yield loss than would be expected from its infection by disease although Rowe & Doodson (1976) suggest that it is less tolerant under severe disease pressure. Preliminary observations of trials grown at SCRI indicated that some genotypes selected as breeding lines appeared to show this characteristic. Therefore, two spore-proof glasshouse experiments, where inoculum could be controlled, and four field experiments were carried out to investigate the potential of disease tolerance as a breeding character.

Materials and methods

Experiment A - glasshouse

Five pairs of genotypes of barley containing identical powdery mildew resistance genes (Table 1); two resistant, Apex and MC20; and eight susceptible, Triumph and Tasman, Javelin and Egmont, Carnival and Midas, Golden Promise and Proctor, were grown as single plants in 15 cm square pots on sand beds with automatic watering. The experiment was grown in two blocks in two compartments of a sporeproof glasshouse, one block in each compartment being protected from disease by regular application of fenpropimorph (Mistral). Within each block each genotype was represented by 20 plants grown in a completely randomised design. The glasshouse was maintained at *c.* 20° C with an 18 h artificial light period each day. One compartment was kept free from disease contamination and was presumed also to be free from air-borne saprophytes. The other compartment was regularly infected with spores of a single strain of powdery mildew capable of infecting only the *Ml(La)+Mla12* or *Mla8* resistance genes (Javelin and Egmont or Proctor and Golden Promise respectively). Plants were individually harvested and the number of fertile tillers was recorded. Height, grain number and thousand kernel weight (TKW) were measured on the main stem and the plant yield was recorded.

Experiment B - glasshouse

Seven genotypes of barley were used, one fully resistant cv. Atem, four defeated major gene cvs viz., Golden Promise, Midas, Proctor and Triumph, and two breeding lines expressing partial resistance from crosses of partial resistant selections with cv. Golf (ITA) and cv. Regent (ITD) (Table 1). Ninety-six plants of each genotype were grown in the summer with no supplementary lighting in 15 cm square pots with three plants per pot in each of two sporeproof glasshouse compartments maintained at *c.* 20° C, one being kept free from incoming spores and the other infected with a mixture of mildew races containing virulence to all the major genes present in the barley genotypes except *mlo*. Plants were grown both as three-way mixtures consisting of one plant of each component in the same pot, and as a monoculture with all the same genotype in each pot, under drought stress or adequate watering, and in two types of compost to test the effects of these factors on yield components. Alternate rows of plants were drought stressed or adequately watered but otherwise pots were completely randomised within a surround of pots of cv. Atem as a guard.

Experiment C - field trial

Thirteen cultivars or breeding lines of barley were analysed, one fully resistant (cv. Atem), five expressing partial resistance from crosses of partial mildew resis-

Table 1. Barley cultivars and breeding lines used in all experiments

Genotype	Expt	Response[a]	Resistance genes[b]
Pallas	F	S	*Mla8*
Golden Promise	ABCEF	S	*Mla8*
Proctor	ABFE	S	*Mla8*
Hora	EF	S	*Mla12*
Midas	ABFE	S	*Mla6*
Golf	FDE	S	*Mlg (CP) (La)*
Regent	F	S	*Mla12+Mlg(CP)*
Triumph	ABCFE	S	*Mla7(Ab)*
Corgi	F	S	*Mlg (CP)*
Tyne	DEF	R	*Mla13 (La)*
Apex	AEF	R	*mlo*
Atem	BCF	R	*mlo (La)*
7163 (Hen Gymro) (71)	EF	Pr	Polygenic
7204B (Cornutum) (72)	EF	Pr	?
7526 (Abed 894) (75)	F	Pr	Polygenic + *Mlg(CP)*
9319 (Ethiopian) (93)	F	Pr	Polygenic
9855 (S. American 2.79) (98)	F	Pr	Polygenic
Hornings Sommergeste	F	Pr	Polygenic
Gloire Du Velay	EF	Pr	Polygenic
ITA (22044CoI92)	BCEF	Pr	Polygenic (+*Mlg(CP)*?)
ITB (22045CoI5)	C	Pr	Polygenic (+*Mlg(CP)*?)
ITC (22045CoI33)	CF	Pr	Polygenic (+*Mlg(CP)*?)
ITD (22048CoI15)	BCEF	Pr	Polygenic (+*Mlg(CP)* + *(La)*?)
ITE (22049CoI123)	CF	Pr	Polygenic (+*Mlg(CP)* + *(La)*?)
71/68/5	C	Pr	Polygenic
72/31R/9	C	Pr	Polygenic
75/7M/2	CF	Pr	Polygenic (+*Mlg(CP)*?)
93/38R/7	CF	Pr	Polygenic
98/59/2	CF	Pr	Polygenic
B87-105/1	EF	Pr	Polygenic (+*Mla7(Ab)*?)
B87-106/4	EF	Pr	Polygenic (+*Mla7(Ab)*?)
P02	F	S	*Mla3*
P03	EF	S	*Mla6*
P06	EF	S	*Mla7 (Mu2)*
P08b	F	S	*Mla9*
P10	EF	S	*Mla12 (Em2)*
P11	F	S	*Mla13*
P14	EF	S	*Mlra*
P15	F	S	*Ml (Ru2)*
P17	F	S	*Mlk*
P21	EF	S	*Mlg(CP)*
P23	EF	S	*Ml (La)*
P24	F	S	*Mlh*
Blenheim	D	S	*Mla12 (Ab)*
Camargue	D	S	*Mla13 (Ab)*
Doublet	D	S	*Mla7 (La)*
Natasha	D	S	*Mla12 (Ab)*

Table 1. continued

Genotype	Expt	Response[a]	Resistance genes[b]
Prisma	D	S	*Mla12 (Ab)*
Sherpa	D	R	*Mla13 + Mlu*
Tweed	D	S	*Mla9 + Mlg(CP)*
MC20	A	R	*mlo*
Tasman	A	S	*Mla7 + MlAb*
Javelin	A	S	*Ml(La) + Mla12*
Egmont	A	S	*Ml(La) + Mla12*
Carnival	A	S	*Mla6*

[a] S = susceptible; R = resistant; Pr = partially resistant

[b] Brown & Jorgensen, 1991.

tant selections with cvs Golf (ITA & ITB), Corgi (ITC), or Regent (ITD & ITE), five selections from crosses between sources of partial resistance and cv. Golden Promise (71/68/5, 72/31R/9, 75/7M/2, 93/38R/7 and 98/59/2), and two defeated major gene cvs, Triumph and Golden Promise (Table 1). Most of these genotypes were not tested in experiments D and E but some were included again in F. Plots measuring 1.9 × 1.22 m (excluding gaps) of each cultivar were sown at SCRI in 1988 in a three replicate split plot design using with and without desease control as the main plot. Seed was treated with triadimenol + fuberidazole (Baytan) to control seed borne diseases. The fungicide treatment consisted of a single spray of fenpropimorph (Corbel) + carbendazim (Bavistin) when mildew first started to appear on Golden Promise (GS30), followed by a single spray of propiconazole + tridemorph (Tilt Turbo) once mildew started to reappear (GS39). This gave adequate protection for the whole season. Plots were harvested with a plot combine and yield recorded. Mildew levels were assessed three times during the season.

Experiment D - field trial

Twenty cultivars or breeding lines of barley were used consisting of the genotypes used in experiment C except for Golden Promise and ITB, plus cvs Blenheim, Camargue, Doublet, Golf, Natasha, Prisma, Sherpa, Tweed, and Tyne (Table 1). The trial was carried out at SCRI using a design similar to that of trial C and assessed in the same way.

Experiment E - field trial

Twenty cultivars or breeding lines were used in experiment E: the fully resistant cv. Apex, three sources of partial resistance (Gloire du Velay, 7163 and 7204B), four breeding lines expressing partial resistance (B87-105/1, B87-106/4, ITA and ITD), the susceptible cv. Pallas and six near-isogenic lines of Pallas carrying different mildew resistance genes (P03, P06, P10, P14, P21 and P23) and five cultivars expressing varying degrees of resistance and possessing defeated major resistance genes (Golden Promise, Corgi, Regent, Tyne and Hora) (Table 1). Plots measuring 1.9 × 1.22 m (excluding gaps) of each cultivar were sown at SCRI in 1991 in three replicates in a split plot using no disease control, a programme of systemic fungicides (GS31 fenpropimorph (Corbel) + carbendazin (Carbate FL), GS36 tridemorph (Calixin) + propiconazole + tridemorph (Tilt Turbo), GS39 propiconazole + tridemorph (Tilt Turbo)) and a programme of non systemic tridemorph sprays (GS31 Ringer and GS39 Calixin) as the main plots. Seed was treated with carbendazim + thiabendazole + imazalil (Cerevax Extra) to control seed borne diseases and plots were scored and harvested as for trial C. In addition, 12 of the leaves subtending the flag leaf and 12 of the next leaf below were sampled at random from each of the control (no fungicide) plots and assayed for mildew biomass per unit leaf weight using an enzyme-linked immunosorbent assay (ELISA) (Newton & McGurk, 1991). The area under the disease progress curve (AUDPC) was also calculated.

Table 2. Main effects of mildew challenge on barley plant height, yield and yield components in glasshouse experiment A

Treatment	Height (cm)	Tillers	Grain number	TKW[1] (g)	Plant yield (g)
Mildew	89.25	3.463	16.49	54.99	2.394
No mildew	84.65	3.715	16.93	48.48	2.607
LSD 5%	0.74	0.123	0.06	0.86	0.095

[1] Thousand kernel weight

Experiment F - field trial

Experiment F was carried out in the same year as E but at a different site at SCRI. It consisted of the same 20 cultivars plus an extra 20 representing more of the same type of material (Table 1). Only two main plots were incorporated in the design, no disease control and the systemic fungicide programme, and ELISA data were not used but other trial details were the same as for experiment E.

Results

Experiment A - glasshouse

Despite generally low levels of mildew infection in this experiment, mildew attack had a significant effect on plant height, yield and its components. However, there were no significant cultivar × treatment interactions indicating that the effect of mildew attack was consistent irrespective of the effectiveness of the resistance genes carried by the cultivars. The principal effects of mildew challenge were reduced tillering and grain number but much of this effect was compensated for by increased TKW (Table 2). No cultivar effects were attributable to either common resistance genes or parentage. Plant height was consistently greater in mildew challenged plants.

There was a high correlation in the performance of each of the genotypes between yield loss due to mildew challenge and yield of the uninfected plants, whether the challenged plants were compared to the mildew challenged fungicide control ($r = 0.915$; $P<0.001$) or with the mildew non-challenged non-fungicide protected plants ($r = 0.745$;$P<0.001$). There was also a high correlation between the yields of genotypes, mildew challenged and non-challenged, when they were not fungicide protected ($r = 0.845$; $P<0.001$) but not when they were ($r = 0.390$; $P<0.15$ ns).

A linear regression was fitted between the mean yields of the blocks that were not protected by fungicides in the infected and clean compartments of the glasshouse. Midas and MC20 both showed markedly greater yield in the infected compartment than the clean compartment although no mildew developed on them indicating that tolerance may be a response to inoculum pressure as much as to disease. Of the cultivars which were infected by mildew, Javelin and Golden Promise were the most and least tolerant, respectively. As mildew levels were low the effect of infection relative to induction of resistance reaction was likely to be small. Thus the relationship between yield loss due to inoculum pressure, rather than disease, and potential yield is likely to be greater than the effects of assimilate drain due to mildew infection. The low mildew levels recorded are unlikely therefore to distort the overall conclusions substantially for the susceptible genotypes.

Experiment B - glasshouse

In experiment B there were significant effects of the compost, watering and disease on yield components of cultivars (Table 3). There were few cases where the performance of the mixtures differed from the mean of their components and no consistent pattern emerged. Although the relative performance of different genotypes and mixtures varied greatly with compost, for example plant yield, no particular genotype dominated these effects and there was no obvious advantage of mixtures overall. As the components of the mixtures could be identified separately they were added to the effects of the single genotypes overall. Again there was a correlation between yield under mildew challenged and non-challenged conditions ($r = 0.724$; $P<0.001$) and again cv. Midas, as well as cv. Proctor, yielded relatively highly when challenged and in this case infected, i.e. they were tolerant under glasshouse conditions. The criterion of the most marked deviation from the regression of yield loss on amount of disease was subsequently used to select genotypes of potentially high and low tolerance in all experiments. Whilst consistent deviation from the regression between yield loss and disease scores between experiments was regarded as the most useful criterion indicative of tolerance or non-tolerance, deviations of a least one standard error

Table 3. Effects of stress factors on yield components in glasshouse experiment B

	Mean height	Mean TKW	Mean weight
Compost J	64.36	36.19	0.71
Compost U	66.77	29.26	0.59
Drought	58.65	25.87	0.47
Adequate	72.49	39.59	0.83
Diseased	62.11	28.53	0.53
Clean	69.02	36.93	0.77
SED	0.95	0.77	0.02

Table 4. Yield responses to systemic fungicide treatment and mean mildew levels in field trials

Field trial	Fungicide response (%)	Mean mildew (%)
C	21%	11%
D	17%	18%
E	31%	15%
F	23%	15%

from the fitted regression line were considered most reliable.

Field trials

The overall yield responses to fungicide in field trials ranged from 17% to 31% reflecting the relatively susceptible nature of the genotypes used. This might also explain the high mean mildew levels in the main plots with no disease control, 11% to 18% (Table 4).

Experiment C - field trial

Among the 13 genotypes in this field experiment there were correlations of $r = 0.614$ ($P<0.05$), $r = 0.735$ ($P<0.05$) and $r = 0.720$ ($P<0.05$) between yield loss due to disease and the three mildew scores, so score 3 was used in Table 5. The genotypes which were most tolerant were 98 and ITA, and the least tolerant were 75 and Triumph.

A linear regression of infected yield on clean yield which passes through the origin means that tolerance does not vary with yield level. If a linear regression were to intercept the Y axis above the origin it would suggest that the relationship is curvilinear with a greater loss in yield due to disease as the fungicide protected yield (yield potential) increases. Our data does not provide a sufficient range of points to test the curvilinear relationship so the intercept of a linear regression with the Y axis was used to infer the effect of yield potential upon disease tolerance. Over the yield levels we have observed, the linear regression between infected and clean yield was found to intercept the Y axis close to the origin. Because the deviation of the intercept from the origin was negligible and not significant (Table 5) it can be concluded that tolerance does not differ with yield level in this particular trial.

Experiment D - field trial

The mildew was apparent relatively early in the season on the 20 genotypes in field experiment D but the conditions were less favourable for later development and the best correlation between yield loss due to disease and mildew was with the second score ($r = 0.458$; $P<0.05$). The genotypes which were most tolerant were Blenheim and Tweed, and one of the most non-tolerant was Triumph again but also ITA. The regression coefficient of the mildew infected yields of the entries on their fungicide protected yields was + 0.616, cutting the Y axis at + 195 which did not significantly differ from zero.

Experiment E - field trial

A mean of the three mildew scores was correlated with yield loss in this experiment as, individually, they all showed good correlations with yield loss. The correlation coefficient between mildew and yield loss calculated from the systemic fungicide treatment in experiment E was highly significant (0.927 ($P<0.001$)) and still significant when using the non-systemic fungicide data (0.790 ($P<0.001$)). The most tolerant genotypes were P14 and B87-106/4 and the least tolerant were ITA and P03. The regression coefficient of the infected on the clean yields of the entries was + 0.839 cutting the Y axis at - 113 which was not significantly different from zero.

The mean ELISA values from all the control plots were used to correlate with yield loss calculated from both the systemic and non-systemic fungicide protected plots. The correlation between the mean of the three mildew visual scores and the ELISA mean was 0.551 ($P<0.01$). The correlation coefficients between

Table 5. Summary of potentially tolerant mildew genotypes of barley identified in field and glasshouse experiments

Experiment	Tolerant	Non-tolerant	Correlation of yield loss with mildew cf + fung	Coefficient of regression of untreated on treated yield	Y axis intercept (g)
Expt A (G-hse)	**Midas**[#], MC20 & Javelin	G. Promise	–	0.660	
Expt B (G-hse)	**Midas & Proctor**	–	–	0.525	
Expt C (Field)	ITA & 98	**Triumph** & 75	0.720	0.800	+ 7
Expt D (Field)	Blenheim & Tweed	**Triumph** & **ITA**	0.458	0.616	+ 195
Expt E (Field)	**P14** & **B87-106/4**	**P03** & **ITA**	0.927 0.790[##]	0.839 0.790	- 113
Expt E (ELISA)	**P14**, Pallas **&Proctor**	G. du Velay & **P03**	0.470 & 0.475		
Expt E (AUDPC)	**P14** & **B87-106/4**	**P03**	0.914 & 0.787		
Expt F (Field)	**P14** & 75 **B87-106/4** G. du Velay	93, Corgi & H.S'g'te	0.801	0.881	- 129

[#] Bold indicates genotypes which appear more than once as tolerant or non-tolerant

[##] Non-systemic fungicide control

the ELISA mean and yield loss were 0.470 ($P<0.05$) and 0.457 ($P<0.05$) when using the sytemic fungicide and non-sytemic fungicide data, respectively. The most tolerant genotypes were P14, Proctor and Pallas and the least tolerant were P03 and G. du Velay (Table 5).

The correlations between the AUDPC scores and yield loss were 0.914 ($P<0.001$) and 0.787 ($P<0.001$) when using the systemic fungicide and non-systemic fungicide data respectively, and the correlation between the AUDPC and the visual scores using either the mean of three or four was 0.976 and 0.999 ($P<0.001$), respectively. The most tolerant genotypes were P14, and B87-106/4 and the least tolerant was P03 (Table 5).

Experiment F - field trial

The correlation coefficient between the mean of the three mildew scores and yield loss for experiment F was 0.801 ($P<0.001$). The most tolerant genotypes were again P14 and B87-106/4 but also Gloire du Velay and 75. The least tolerant were Hornings Sommergeste, 93 and Corgi. The regression coefficient of the infected on the clean yields was 0.881 (Table 5) cutting the Y axis at - 129 which was not significantly different from zero.

Discussion

Field trials E and F included 20 genotypes common to each trial and used near-isogenic lines to determine whether any particular resistance gene had effects on tolerance. These two trials gave the best correlations between mildew scores and yield loss due to disease. In both trials, genotypes P14 and B87-106/4 were identified as being tolerant. Neither genotype had previously been available for trialling. Among the non-tolerant genotypes Triumph and ITA were identified twice in

two field trials but ITA was also identified as being tolerant in another field trial and as neither in trial F. As these trials were carried out in different years this indicates a potentially large environmental interaction in the expression of tolerance.

Using ELISA as a measure of mildew infection P14 was again shown to be tolerant and P03 non-tolerant. Proctor and Pallas were also picked out as tolerant, Proctor having been identified as tolerant in glasshouse experiment B and by Little & Doodson (1972) in their field trials. Gloire du Velay was identified as non-tolerant by the ELISA technique but as tolerant in trial F by conventional scoring techniques. Thus the biomass of Gloire du Velay must be greater than visual assessments indicate in the same field trial, which could be explained in terms of denser more branching colony morphology. There is clearly no evidence of greater than expected visual mildew, indeed experiment F indicates a trend to the contrary. In addition to Proctor both glasshouse tests identified Midas as being tolerant. The effects of the high mean mildew levels found in these field experiments may have obscured the detection of many potentially tolerant genotypes as found by Rowe & Doodson (1976), and may mean that tolerance is only truly effective at low disease levels and may therefore be suited to low input systems.

Calculation of the AUDPC in trial E revealed the highest correlations between yield loss and infection using either three or all four scores for the calculations and either the systemic or non-systemic fungicides as yield controls. Thus in this trial all three methods of disease assessment identified genotype P14 as the most tolerant and P03 as the most non-tolerant. B87-106/4 was also identified as tolerant from the mean visual scores and the AUDPC. Therefore as an assessment of yield loss due to disease, AUDPC appears to be a good method of reflecting the overall effect although single scores in mid-season also correlate highly and identify the same tolerant and non-tolerant genotypes.

These variations highlight the problems in identifying tolerance as the measure of tolerance of any one genotype can only be expressed relative to the other entries in the trial. Clearly the composition of the trial can influence the identification of tolerant genotypes. A solution may be to form the regression of yield loss on mildew level from a set of control cultivars and express tolerance as deviations from that regression.

Some genotypes do rank among the least or the most tolerant more frequently than would be randomly expected. It is also clear that there are environmental factors which affect tolerance expression differentially in different genotypes. The fact that the near isogenic line P14 which carries the *Mlra* gene was identified as tolerant suggests that tolerance is a character associated with certain genes and perhaps some resistance genes. However, it does not appear to be consistently associated with any of the other genes represented, nor with partial resistance in general.

No overriding relationship between tolerance and yield level was detected. The points at which the regression lines between yield when fungicide protected and when infected cut the Y axis did not differ significantly from zero, but they varied from + 195 g to - 129 g per plot, highlighting not only a problem which may be attributed to the genotypes used in each trial but also indicating that the environment may affect this relationship. In the absence of the normal levels of saprophytes on the leaves (Smedegaard-Petersen & Tolstrup, 1987), the mildew spores could have induced a proportionately larger defense response in cultivars resistant to the mildew population used than was expected. Inoculum pressure may affect the relationship not only between yield when infected and uninfected by pathogens, but also the tolerance to disease. The observations of the behaviour of cv. Proctor would also seem to suggest this (Little & Doodson, 1972; Rowe & Doodson, 1976).

The increase in TKW with mildew challenge in the first glasshouse experiment was unexpected as mildew generally results in a reduction in TKW when measured on samples of grain harvested from plots. This experiment is different in that TKW was measured on the main stem of single plants and, given the decrease in the number of tillers, one might expect some form of compensation through an increase in TKW might be expected. It is interesting to note that this compensation has favoured development of the main stem, for if all the heads had the same grain number and weight as the main stem the plant yield would have been 3.140 g under mildew challenge but 3.049 without mildew challenge. There was, therefore, a greater reduction in grain number and/or grain weight of the other tillers under mildew challenge. Such effects may reflect the timing and extent of mildew attack rather than any general phenomena but do illustrate the ability of the plant to divert assimilates in the presence of disease.

Triumph and Golden Promise were included in glasshouse and field experiments along with partially resistant material. The original sources of partial resistance were land races or wild barleys (Jones & Davies, 1985; Asher & Thomas, 1987) and therefore

their derivatives used in these experiments might be expected to carry other characters including tolerance if it is a character more common in wild genotypes. If tolerance is related to inoculum pressure then it will be compounded by resistance expression. Neither Golden Promise nor Triumph showed any tolerance, Triumph even being classified as non-tolerant. However, many partially resistant selections, which are closer to the non-selected material, also showed no evidence of tolerance.

This work provided no evidence to suggest that high apportionment of assimilates to grain yield in high yielding genotypes was deleterious to tolerance. Sabri and co-workers found that photosynthesis was less affected by mildew infection in wild oats compared with cultivated oats resulting in greater tolerance in wild oats (N. Sabri, P.J. Dominy & D.D. Clarke, personal communication). Thus tolerance may be unrelated to assimilate drain to the pathogen but instead related to resistance to metabolic disruption whether caused directly by the pathogen or by induction of various resistance responses and is thus potentially related to inoculum pressure.

The principal mechanisms which are responsible for conferring tolerance remain obscure. There is therefore considerable potential for detailed study of plant response to infection in terms of chlorophyll metabolism, photosynthesis rates, resistance mechanism induction, inoculum pressure, assimilate distribution and developmental changes.

The tolerant genotypes were not found at the extremes of the yield distribution and thus may be assumed to be characters not causally related to yield. Thus as the potential for tolerance is apparently not related to selection for high yield, this character is a practical breeding objective.

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