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Progress towards wheat varieties with resistance to fusarium head blight

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Abstract

Many breeders are urgently trying to improve genetic resistance to fusarium head blight. Within a set of 17 varieties tested in 4 countries resistance seems to be stable across pathogen species and location and a reduction in disease appears to result in a proportional reduction in deoxynivale (DON) content. Breeding is a compromise between achieving improvements in yield, quality and resistance and many characters are technically difficult to handle, sometimes making progress slow. New technology is likely to increase the efficiency of this process and we hope to see better resistance to fusarium head blight in Europe within the next 10 years.

1. Introduction

Damage to the yield and quality of small grain cereals (principally wheat, barley, rye and maize) by *Fusarium* spp. and associated fungi is a problem recognised from early in the last century (Jenkins, Clark, & Buckle, 1988; McMullen, Jones, & Gallenberg, 1997; Snijders, 1990). The disease, fusarium head blight (FHB) is widespread, occurring in North and South America, Europe and Asia where moist weather occurs during the growing season, particularly if this comes at flowering and grain filling. Infection became particularly serious in the early 1980s and 1990s in North America and the late 1990s in Europe although it has occurred every year in some areas (notably Southern Germany).

2. The causal fungi

In wheat fusarium head blight is caused by several species of *Fusarium* (*F. graminearum*, *F. culmorum*, *F. avenaceum* and *F. poae*) and by *Microdochium nivale* (see Parry, Jenkinson, & McLeod, 1995). *Fusarium* spp. produce a wide range of mycotoxins, important for their potential effects on animals and humans (see Ellner, 2000; Smith, Lewis, Anderson, & Solomons, 1994). Three important groups are the trichothecenes, zearalenone and its derivatives and the fumonasines (see D'Mello et al., 1966). Many trichothecenes have been identified but most attention has focussed on those of type A (T-2 toxin, Ht-2 toxin and others) and type B (nivalenol, NIV; deoxynivalenol, DON and its acetyl derivatives 3-ADON and 15-ADON). Within Europe the FHB pathogens principally produce DON and NIV (for summary see D'Mello, MacDonald, & Placinta, 1996). *M. nivale* does not produce toxin.

3. Variation in resistance

Genetic variation in resistance to the disease is well recognised in most parts of the world (Bai, Plattner, Desjardins, & Kolb, 2001; Miedaner, Reinbrecht, Bahle, Schneider, & Geiger, 1999; Snijders, 1990). Variety registration procedures in a number of countries assess genetic resistance to fusarium head blight and minimum standards for resistance are in place (Bundessortenamt, Beschreibende Sortenliste, 2000, Hannover; NIAB, UK Recommended Lists of Cereals, 2000). Differences in resistance can be readily demonstrated between commercially grown varieties, as in an inoculated, irrigated experiment carried out at the Monsanto Cambridge site in 2000 (Table 1).

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Table 1
Amount of fusarium head blight on winter wheat varieties after artificial inoculation with *Fusarium culmorum*; 6 replicates, irrigated, Cambridge 2000

Variety	Date inoculated[a]	Assessed at (°C days)			Average disease[b]
		350[c]	400	450	
95NYP1256	143	0.2	0.3	1.5	1
Sumai#3.2	141	0.2	0.3	1.7	1
Zhefeng	141	0.5	0.6	1.7	1
Ning 7840	148	1.8	1.8	3.2	2
Patton	148	0.7	2.5	6.4	3
Heyne	152	0.6	3.2	9.2	4
Jagger	143	0.3	5.7	9.9	5
Freedom	150	3.9	5.8	12.3	7
Cockpit	160	2.7	7.4	13.7	8
Piko	164	1.6	9.9	20.3	11
Soissons	152	3.4	8.0	21.9	11
Ludwig	160	3.9	12.0	20.0	12
Kraka	164	3.6	11.9	21.8	12
AC Winsloe	154	4.0	8.6	24.7	12
Greif	160	7.3	10.5	20.2	13
Frelon	154	6.0	9.2	23.1	13
Petrus	160	3.0	19.1	24.3	15
Achat	164	2.8	17.3	27.8	16
Astron	164	6.1	15.7	26.8	16
Batis	164	5.0	21.5	33.3	20
Isengrain	154	10.9	17.3	35.6	21
Huntsman	160	10.9	26.4	35.5	24
Tremie	152	12.7	22.7	40.9	25
Smart	164	7.3	27.9	47.1	27
Savannah	164	8.7	28.8	50.5	29
TP1689/-/-/18	164	13.1	31.5	45.5	30
Fruhgold	157	12.1	29.9	49.6	31
Semper	164	8.8	34.3	49.6	31
Ritmo	164	18.6	32.7	43.2	31
Rialto	160	15.5	36.6	58.0	37
Contra	160	15.6	38.1	58.8	37
Shango	164	19.7	39.6	61.2	40
Consort	164	15.3	44.7	69.5	43
Tower	164	13.1	46.7	70.0	43
Equinox	160	26.4	55.1	68.8	50
Hanseat	160	30.8	54.6	73.6	53
Charger	160	30.6	65.8	80.4	59
5% LSD			12.5 (within date)		7.2

Varieties inoculated individually at anthesis and assessed for disease by thermal time. Inoculated with a mixture of two *F. culmorum* isolates.
[a] Days after 1 January.
[b] Percentage ear area with symptoms of FHB.
[c] Average of daily maximum and minimum temperatures summed over days.

Wheat varieties are most susceptible at the flowering stage, growth stage (GS) 61–69 (Tottman & Broad, 1987), so it is important when critically comparing varieties to inoculate at a consistent stage of growth. In this way lines with different maturity, often from different geographic regions, can be compared. In this experiment disease was then assessed at defined intervals after inoculation (350, 400 and 450 °C days), which accounted for average temperature as well as time to minimise inaccuracies due to changing average temperatures during disease development.

Large differences in resistance were observed. In these very disease conducive conditions up to 60% infection was observed on some UK varieties (Charger) whereas the widely grown German variety Batis had 20% disease. Several lines were even less diseased (Sumai#3, etc.) but most of these are low yielding and/or poorly adapted to northwest Europe. Their resistance is currently being incorporated in our, and a number of other, breeding programmes throughout the world.

4. Inheritance of resistance

At least tree types of genetic resistance have been described. Type I, resistance to initial infection, Type II,

resistance to spread of the pathogen (Schroeder & Christenson, 1963) and Type III, resistance based on the ability to degrade mycotoxin (Miller, Young, & Sampson, 1985). Resistance is inherited quantitatively and is affected by environmental conditions making it often difficult for the researcher to define reactions. However, a number of genes conferring resistance to the pathogen, and DON degradation, have been described (see Ban, 2000) from different gene pools. In addition many of these genes have been shown to be additive and it is likely that many more await discovery.

5. Stability of resistance

A number of pathogens can cause fusarium head blight, particularly in Europe where *F. graminearum*, *F. culmorum*, *F. poae* and *M. nivale* have all been implicated (see Parry et al., 1995). For resistance breeding it is important that resistance to one pathogen confers resistance to the others. This seems to be the case as reported in the literature (Mesterhazy, 1977; Snijders & Van Eeuwijk, 1991) and in a recent collaborative experiment done between ourselves, The Institute for Agrobiotechnology at Tulln, Austria (Professor P. Ruckenbauer) and CEBECO Zaden BV, The Netherlands (Dr H. De Jong) as part of the EU Concerted Action FAIR programme CT-4094 (acronym ''Mycotochain'').

Seventeen varieties from five different European countries were tested in five locations, some with artificial infection and some with natural infection. Disease estimates demonstrated that, in general, varieties ranked similarly at the different sites (Table 2) although some varieties (e.g. Semper) showed some inconsistency across sites. The validity of this observation is being checked.

In addition in Austria varieties were tested against six separate pathogens that have the potential to cause FHB (*F. culmorum*, *F. graminearum*, *F. avenaceum*, *F. poae*, *F. sporotrichoides* and *F. subglutinans*). Only three species caused substantial disease on the susceptible lines (see Table 2) and again variety ranking was similar. These results are in agreement with those reported by Van Eeuwijk et al. (1995) who similarly tested varieties covering a range of resistance to different pathogens in different regions of Europe.

6. Relationship between disease and mycotoxin content of grain

Specific end use requires grain free from mycotoxins so it is important to the grower, and plant breeder, to know that decreased disease in the field leads to decreased mycotoxin content in the grain. Several studies have show this to be the case with either natural epidemics (Wosnitza, personal communication) or with

Table 2
Amount of disease on heads of winter wheat at five locations in Europe; artificial infection in the UK and Austria, natural infection in Germany and The Netherlands, 2000

	Cambridge, UK	Tulln, Austria			Bavaria 1	Lelystadt, Netherlands	Bavaria 2
	F.c.[a]	F.c.[a]	F.g.[b]	F.a.[c]			
Petrus	15	14	15	5	0	1	3
Batis	20	18	15	8	0	3	3
Contra	37	46	42	22	7	11	5
Hanseat	53	76	62	34	16	23	5
Consort	43	32	46	19	16	9	6
Charger	59	76	76	58	19	28	6
Equinox	50	62	46	29	13	11	6
Achat	16	26	32	15	3	4	2
Ludwig	12	19	24	15	3	4	3
Semper	31	29	42	16	1	4	4
Tower	43	21	20	7	0	3	3
Bercy	–	83	83	66	12	30	5
Ritmo	31	54	58	19	14	13	4
Soissons	11	22	21	15	4	7	4
Shango	40	54	62	24	17	14	5
Tremie	25	58	58	46	9	17	7
Isengrain	21	26	26	22	10	6	6
5% LSD	8.8				0.47		

[a] *Fusarium culmorum.*
[b] *Fusarium graminearum.*
[c] *Fusarium avenaceum.*

artificial infection (Bai et al., 2001; Miedaner et al., 1999), the latter often when disease levels, and mycotoxin content, are high. The relationship can also be inferred by the variation in mycotoxin content seen in harvested crops from a region. Different crops will have been subjected to a range of disease pressures depending on the environment and will have had varying fungicidal control programmes.

7. Wheat variety production

Wheat varieties are not widely adapted so varieties are bred for specific regions or environmental conditions. For commercial success in Europe a variety needs high yield, appropriate quality and a mix of resistance to between five and eight major diseases (Fig. 1).

Disease frequency in a particular area will vary from field to field and year to year, but in general growers, agronomists and the appropriate national registration authorities recognise which diseases are important for that region. They are also able to judge the level of resistance that is needed to reduce damage to acceptable levels, with fungicides if appropriate.

8. The breeding pipeline

The wheat breeder is attempting to combine high yield, appropriate quality and disease resistance in the same variety. To get this he crosses two varieties, each of which contains some of the traits he is aiming to combine, and then selects within the progeny for the desired combination in successive years, each year retaining

Fig. 1. Diseases of wheat—Europe.

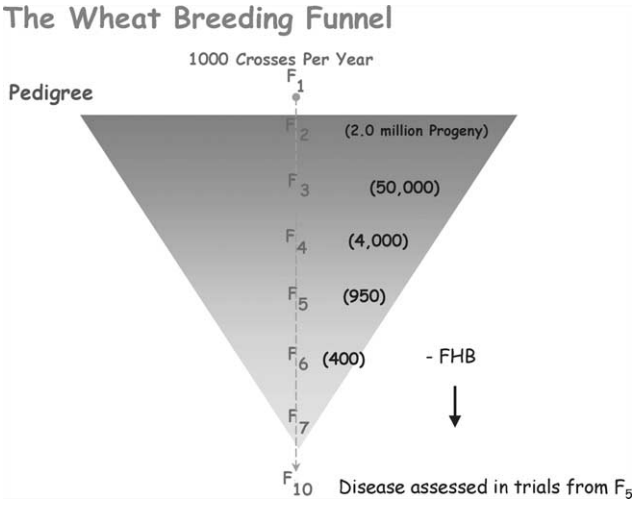

Fig. 2. The wheat breeding funnel.

only those lines of potential value. Some characters are relatively easy to identify (height, ear size, etc.), others such as disease resistance require more effort (introduction of the disease if not already present), and still others may not be directly measured until later when he has sufficient seed available. The breeder therefore 'funnels' the material (Fig. 2).

Selection for resistance to fusarium in our programmes is begun only at about half way thought the development process at present because it is technically difficult for at least two reasons. First, environmental variation across the field/breeding nursery is likely to be significant making it difficult to differentiate the resistant lines from the 'escapes'. Second, and perhaps more critical, is the danger that grain harvested for re-sowing the following year may be infested with fusarium thus leading to establishment problems. Consequently, selection only begins when enough seed is available to establish a dedicated nursery.

9. What has been achieved?

Within Europe the range of resistance available in commercial varieties differs between countries. In general, varieties from the UK are more susceptible than those from France and Germany (Table 3). Clearly there is overlap between countries and this analysis says nothing about which varieties are successful commercially, and indeed whether disease prone areas, such as the south of Germany, only grow the more resistant varieties. However, this snapshot does show that German and French breeders have achieved a better standard of resistance than their UK counterparts, probably because they have seen a greater need for resistance, over a number of years, in their regions than UK breeders.

Table 3
Amount of disease on winter wheat artificially inoculated with *Fusarium culmorum*, Cambridge 2000

Variety		Average disease[a]
Petrus	Germany	15
Astron	Germany	16
Batis	Germany	20
Smart	Germany	27
Contra	Germany	37
Soissons	France	11
Frelon	France	13
Isengrain	France	21
Tremie	France	25
Shango	France	40
Savannah	UK	29
Rialto	UK	37
Consort	UK	43
Equinox	UK	50
Charger	UK	59

Varieties grouped by country of origin.
[a] Mean of 6 replicates, 10 heads per plot and 3 assessment dates.

10. Future prospects

For the future there is an urgent need for better resistance to meet both farmer and end user needs. Three strategies can be envisaged; continued emphasis on conventional disease selection, deployment of molecular markers for resistance and the possibility of using transgenic resistance.

10.1. Conventional selection

We are trying to combine resistances from within the current German material to 'stack' the resistance factors and to introduce the better resistance from lines such as Sumai#3. Effective selection in the presence of the disease at key stages in the breeding process is essential for success.

10.2. Molecular markers

The ability to identify the 'fingerprint' of specific genes can offer considerable advantages in breeding. This 'fingerprint' of key genes which describes the resistance in Suma#3 has been identified (Sixin, Anderson, Stack, & Frohberg, 2001). Use of these gives several advantages; the ability to identify the plant we want without using the pathogen; the test is not tissue specific and allows one to do the indirect testing for resistance at any time of the year. In addition high throughput technology would allow much more material to be handled.

10.3. Transgenic resistance

Resistance to disease is the result of a chain reaction in the plant. When a pathogen encounters a plant there is a recognition process that leads to a resistance response. If this response is slow or weak, susceptibility results. We aim to understand this process, and then modify the plant response so that we invoke a multiple defence shield that will hopefully provide broad based and durable resistance to fusarium.

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