

INVITED REVIEW

CLASSICAL PLANT BREEDING FOR DURABLE RESISTANCE TO DISEASES

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SUMMARY

Classical plant breeding has produced many improvements to crop cultivars, including disease resistance. It is a dynamic process in which the objective is usually the improvement of many characters simultaneously, including disease resistance. Some introduced resistances failed rather soon after its introduction, because of evolution to virulence in the pathogens; others did not fail in this way and remained effective despite widespread and prolonged use, thus being identifiable as providing durable disease resistance. Examples of durable resistance achieved by classical breeding are given, and show that there is no single genetic model for durable resistance and also, no typical phenotype by which to identify it. It is the durability that initially allows the identification of it. Disease resulting from introduction of genetic male sterility in maize is described and used to illustrate risk in the use of widespread genetically uniform material. Such risk will be associated with disease resistance introduced by biotechnology just as much as it has been by classical plant breeding. Resistance introduced by biotechnology will only be shown to be durable after widespread and prolonged testing.

Key words: genetics, fungal disease, virus disease, linkage, phenotype.

INTRODUCTION

Numerous cultivated plant species were improved for human use during the 20th Century by what has come to be known as conventional or classical plant breeding. This includes the generation of variation, usually by crossing different lines and producing the subse-

quent generations, during which selection for improved plant types is practised. Having generated the variation and selected it, an important component of classical breeding is the extensive testing of the material in the possible environments in which it is to be grown, in comparison with existing cultivars, to demonstrate that improvements have been achieved. For inbreeding crops, re-establishment of genetic stability is also needed, and this requires the cultivation of a number of generations to obtain a high degree of homozygosity. For some crops, this may not be necessary as vegetative propagation is possible, as with potatoes, or the crop is long-lived, such as trees, and the selected specimen may be used for a long time, or, alternatively, genetic variability may be tolerable for some crops and is sometimes used as a defence against diseases.

An important point to note about classical breeding is that, often, the objective is to change many characters simultaneously, resulting in a large number of plants that must be examined for selection. As an example, in the winter wheat breeding programme at the Plant Breeding Institute, Cambridge, in the 1960s and 1970s, more than 1500 crosses per year were made in glasshouses. The F1 generation was produced in the glasshouse, and the F2 plants were planted in long rows, with individual plants at 10 cm intervals, which amounted to a total row length of more than 200 km. During June and July all the more than 2 millions plants in the F2 alone were examined and appropriate plants were selected, in addition to further selection of F3 families and subsequent generations. Characters that were important were yield; agronomic characters (height, strong stems, response to fertilizers etc.); disease resistance; yellow rust; powdery mildew; eyespot; septoria diseases; fusarium earblight. For some lines, bread-baking quality was a further important character.

Inoculation of plants to spread disease was carried out for yellow rust, septoria diseases and fusarium earblights. Irrigation was used to enhance the environment for infection. Obligate pathogens, such as *Puccinia striiformis*, the cause of yellow rust, were favoured by high nitrogen in the soil, which was applied to increase the power of the test.

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At the end of 8 or 9 generations, perhaps three or four lines per year were considered suitable for submission to the national trials system and many of the crosses never produced lines suitable for submission. By the time a new cultivar was fully tested, it could be 11 or 12 years from the original cross and a few more years before it was distributed and more before it became widely grown, if successful (Bingham, 1981).

One important point to be made is that plant breeding is a numbers game (R. Riley, personal communication). No perfect cultivar can be produced, and all new cultivars have both strengths and weaknesses. This is a point that needs to be made in relation to any process that leads to the introduction of a single new character. A choice needs to be made as to which cultivar or cultivars are adequate to be considered for improvement by a single character. An important aspect of this question is how many cultivars need to have the new trait to avoid the risks of genetic uniformity of crops over wide areas, with the inherent risk of simultaneous failure. This is referred to at the end of this chapter.

BREEDING FOR DISEASE RESISTANCE AND IDENTIFICATION OF DURABLE DISEASE RESISTANCE

Many positive attempts to introduce disease resistance have been made by plant breeders and pathologists. Information has been generated about the genetic basis of resistance, thus helping to understand the ease with which genetic resistance can be exploited. Resistance genes have been introduced from related species using elaborate crossing and selection procedures (Knott and Dvorak, 1976).

Early attempts to introduce resistance to rust diseases in wheat led to initial success, followed by failure because new forms of the pathogen evolved, with the ability to grow on cultivars into which resistance genes had been incorporated. Such failures of introduced resistance have continued to the present day, and are still continuing, important new virulence having been identified this year in the black stem rust pathogen *P. graminis* in Africa (Z.A. Pretorius, personal communication). This is virulence for *Sr31*, a gene that had remained effective in widely grown CIMMYT wheats for many years. There has been a long succession of new races of *P. striiformis*, the yellow rust of wheat pathogen, leading to reduced resistance and withdrawal of some cultivars from commercial use (Table 1). The table shows changes in the UK, but similar changes have resulted in epidemics, not only in the UK, but also across the world. Changes have continued to the present and there have been some notable epidemics in re-

cent years in Australia and New Zealand and in several Middle Eastern countries including Iran (personal observations 1995, 1996).

Table 1. Races of *P. striiformis* occurring on wheat in the UK from 1952 to 1979 and cultivars affected. All the cultivars became too susceptible for commercial use.

Year	Cultivars affected	Race name
1952	Nord Desprez	40 E8 ^a
1955	Heines VII	32 E160
1966	Rothwell Perdix	37 E123
1968	Cama, Maris Envoy	41 E136
1969	Maris Beacon, Maris Nimrod	104 E137
1971	Joss Cambier	104 E137 type 2 ^b 41 E136 type 2
1972	Maris Ranger Maris Bilbo	108 E141 104 E137 type 3
1973	Maris Nimrod, Joss Cambier	41 E136 type 3
1973	Clement Kinsman	232 E137 108 E141 type 2
1979	TL363/30/2, Hobbit	41 E136 type 4

^a Nomenclature according to Johnson *et al.*, 1972.

^b Variation occurring outside the differential set listed as 'type x'.

Notwithstanding such failures, fortunately, breeders persevered and, by good fortune, occasionally produced cultivars that remained resistant during widespread and prolonged cultivation, even in areas where other cultivars had failed rapidly. Such resistance can be described as durable (Johnson, 1983). Studying these examples I recognised that they were diverse. To cut a long story short, there was no single resistance phenotype and no single genetic basis for durable resistance. It was important to recognise this, because some writers claimed that resistance that would remain effective would be some form of incomplete resistance under polygenic control (Vanderplank, 1975). The very suggestion that resistance had to be under polygenic control was something of a disincentive to breeders to use it.

However, across a range of crops and diseases, durable resistance could be phenotypically complete or incomplete, and under simple or complex genetic control. It is also important to note that incomplete resistance is not necessarily durable, certainly not in resistance to yellow rust of wheat (Johnson, 1983).

One last critical point that should be noted is that durable resistance is first recognised by the performance of widely grown cultivars. This makes investigation of such cultivars important and their exploitation in the breeding of new cultivars valuable. However, the

recognition of resistance as durable does not guarantee its future performance, particularly after genetic re-assortment has occurred in the breeding process.

EXAMPLES OF DURABLE RESISTANCE

In this short account, it is not possible to describe in detail the many examples of durable resistance to different diseases and how they have been managed in breeding to produce new cultivars that have also performed similarly.

DURABLE RESISTANCE TO BLACK STEM RUST. *Sr2* is a single recessive gene for adult plant resistance that has provided durable resistance in many wheats, in Australia, the USA and in CIMMYT wheats. It was derived from an 'Emmer' (tetraploid) wheat, via the cultivar 'Hope'. This gene has been successfully transferred to many new wheat cultivars by classical breeding, assisted by an unusual linked character known as pseudo black chaff (Hare and McIntosh, 1979).

Sr26 was derived from *Agropyron elongatum* and was used in Australian wheat breeding, giving a high level of resistance and remaining effective in many Australian wheat cultivars in the New South Wales stem rust-prone area. It may be associated with a limitation on yield, but in the Australian conditions, this was less important than the rust resistance it provided. (McIntosh *et al.*, 1995).

Sr31 is a gene carried on a rye chromosome segment translocated to wheat many years ago, on the 1BL-1RS chromosome. This rye segment carries resistance to yellow rust (*Yr9*) and leaf rust (*Lr26*) both of which provided resistance that was not durable. In contrast, the resistance to stem rust remained effective for many years during widespread cultivation, and could reasonably be described as durable. The occurrence of virulence, suspected over recent years, was confirmed unequivocally this year, as mentioned above. This supports the notion that, even after prolonged success, resistance cannot be guaranteed to last permanently (McIntosh *et al.*, 1995). Perhaps the recognition that this resistance performed as though under control of a single gene should have led to the expectation of eventual failure, but after many years, both *Sr2* and *Sr26* have remained effective.

RESISTANCE TO THE EYESPOT DISEASE OF WHEAT. 'Cappelle Desprez', a French bread wheat, was recognised to possess a measure of resistance to eyespot caused by *Pseudosporrella herpotrichoides*. Cytogenetic investigation showed that a large part of this resistance

was carried by chromosome 7A (Law *et al.*, 1975) and the resistance was exploited successfully at the Plant Breeding Institute, despite being a difficult character to score. This was obviously assisted by the resistance being largely controlled by a single chromosome, although there is no evidence that it is a single gene. The resistance was durable and remains effective today, despite some variation in pathogenicity in the eyespot pathogen, particularly in relation to wheat, rye and other grasses (Scott *et al.*, 1978).

New resistance to eyespot from Aegilops ventricosa. New resistance to eyespot was detected in *Aegilops ventricosa* before the Second World War. Attempts were made to introduce it to wheat in the 1960s and were successful in France, requiring a cross between tetraploid and hexaploid (bread) wheats. Cultivars such as 'Rendezvous', possessing the resistance, were not released until the 1980s. The resistance was shown to be carried by chromosome 7D and can be combined with the resistance from 'Cappelle Desprez' (Worland *et al.*, 1988). The *A. ventricosa* resistance is linked to an endopeptidase marker and selection for resistance can be achieved by observation of the endopeptidase isozyme marker (McMillin *et al.*, 1988). Thus, it can be exploited by a combination of classical breeding and the use of a genetic marker. Although the pathogen is variable in pathogenicity, there are, as yet, no reports that this resistance has been overcome.

RESISTANCE TO POWDERY MILDEW IN BARLEY. Many genes for mildew resistance introduced into barley, but the gene *mlo* has been widely used now in western Europe in many spring barley cultivars, and the resistance has lasted up until now, more than 20 years, thus qualifying it for the description as durable (Jørgensen, 1992). This is another example of successful exploitation of durable resistance by classical breeding.

RESISTANCE TO YELLOW AND BROWN RUST OF WHEAT. It is important to note that adult plant resistance and slow rusting resistance are not diagnostic for durable resistance to yellow or brown rust of wheat (Johnson, 1983). For both diseases, durable resistance has been observed in a large number of cultivars. In CIMMYT and related wheats, the linked genes *Lr34* and *Yr18* control incomplete or partial resistance and durable resistance to brown and yellow rust, respectively. The linked character of leaf-tip necrosis has been used to assist in their selection. European wheats with durable resistance to yellow rust have a range of levels of disease resistance (Table 2), and it appears to be under rather complex genetic control, not including the

gene *Yr18*, except in 'Besostaja 1' wheat. Despite the apparent genetic complexity, the resistance has been transmitted in breeding programmes and new cultivars with durable resistance have been produced. There is no space in this chapter to describe these in detail. Another gene that may provide durable resistance to leaf rust has been identified as *Lr46* as one of two genes present in the CIMMYT cultivar 'Pavon 76' that had displayed durable resistance (Singh *et al.*, 1998). Combining *Lr34* and *Lr46* might be a useful strategy. Molecular markers for these genes would be very useful, but have not so far been identified.

Table 2. Percentage leaf area infected with yellow rust in a heavily inoculated trial June 1980, among cultivars with durable resistance to yellow rust, and country of origin.

Cultivar	Origin	Percentage leaf area infected
Hybride de Bersée	France	5
Bouquet	France	25
Cappelle Desprez	France	37
Caribo	Germany	27
Holdfast	UK	6
Hybrid 46	UK	25
Little Joss	UK	3
Luke	USA	22
Maris Widgeon	UK	4
Nugaines	USA	13
Starke II	Sweden	17
Desprez 80 (susceptible)	France	80

RESISTANCE TO VIRUS DISEASES. Two interesting examples may be mentioned.

Resistance to Potato Virus X (PVX). The first is resistance to potato virus X. The gene *Nx* provided resistance to several races of potato virus X, and the gene *Nb* also gave resistance to other races. The combination of the two genes combined these effects and gave resistance to three groups of the virus races. Despite the occurrence of the Group 4 isolates with virulence for the gene combination, such races remained rare and did not prevent the successful exploitation of the *Nx* gene to provide durable resistance (Howard, 1978).

Resistance to Tobacco Mosaic Virus (TMV) in Tomatoes. Resistance to TMV was controlled by the genes *Tm-1* and *Tm-2*, but in both instances was rapidly overcome. When the gene *Tm-2²* was introduced it remained effective in glasshouse tomatoes, despite the de-

tection of some isolates of the virus with virulence, which seemed to have a limited ability to survive and spread (Hall, 1980).

There are many other examples of durable resistance, under either simple or more complex genetic control. Many examples have been so successful that they are rarely mentioned and can be forgotten about. In an interesting example in recent years, resistance to the *Phylloxera* aphid was essential to survival of the European wine industry in the last century, and resistant rootstocks from North America provided the solution. *Phylloxera* was so successfully controlled that American grape breeders forgot about it, and produced some cultivars that suffered from *Phylloxera* in the 1980s.

These examples illustrate that durable resistance to many diseases and insects has been found and successfully exploited by the techniques of classical breeding.

RISKS OF INTRODUCING NEW CHARACTERS AND OF FORGETTING OLD ONES. This chapter will finish by mentioning problems that have occurred in plant breeding and must be considered in the context of classical breeding, and equally in the context of resistance and other characters introduced by biotechnology.

Resistance of maize to Helminthosporium maydis. Maize hybrids were recognised as being superior in performance, and much energy was expended in detassling maize lines to prevent them producing pollen, to ensure cross-pollination. In the 1960s genetic male sterility was found in Texas cytoplasm. With the use of restorer genes, this economic method of producing maize hybrids was rapidly adopted in the USA. In the mid 1970s, maize cultivars with the Texas cytoplasm were severely attacked by *Helminthosporium maydis*, which had previously been considered a minor disease. Although the cytoplasm was in many different cultivars, all were more or less equally susceptible, and world grain prices were affected by the severe epidemic (Day, 1977). Two points can be made. First, despite the use of the Texas cytoplasm in different maize lines, from the disease point of view they were a large-scale monoculture. The other point to note is that the character introduced had no apparent implications for disease resistance. This is very important in the idea of introducing particular genes, by whatever mechanism and for whatever character, uniformly into many cultivars.

Many who work with molecular techniques know, perhaps, not very much about plant breeding, and the diverse qualities needed to produce successful new cultivars. They should, however, be aware of the risks of

genetic uniformity. Cultivars developed by biotechnology will need to be tested as stringently as any other cultivars and, for disease resistance, the resistance introduced by biotechnology will not always be durable. To find out which ones are durable they will have to be tested widely and for a long period of years in an environment favourable to the disease.

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