REVIEW

A Plant Pathologist on Wheat Breeding with Special Reference to Septoria Diseases

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Abstract: This review has a personal, plant pathologist's outlook on plant breeding. It touches upon some generalities, among which the "three stages" of plant breeding, participatory plant breeding and biotechnology in plant breeding. It delves deep into modern molecular studies on leaf blotch (anamorph *Septoria tritici*) and glume blotch (anamorph *Septoria nodorum*) of wheat. Epidemiological knowledge of the teleomorphs *Mycosphaerella graminicola* and *Stagonospora nodorum* has progressed with great strides. Consequences for applied plant breeding slowly become visible.

Keywords: glume blotch; leaf blotch; molecular epidemiology; *Mycosphaerella graminicola; Septoria nodorum; Septoria tritici; Stagonospora nodorum*

My life as a plant pathologist had two episodes of intensive contacts with plant breeders. The first episode was at the onset of my career when I researched the epidemiology of yellow rust (*Puccinia striiformis*) on wheat in close collaboration with farmers and plant breeders (Zadoks 1961). The second episode was at the end of my career when I served as a member of the Committee on Genetic Modification (COGEM) advising the Dutch Minister of the Environment about the registration of genetically modified crops (Zadoks & Waibel 2000). In between, I often had the opportunity to muse upon resistance and resistance breeding (Zadoks 1972, 2002; Parlevliet & Zadoks 1977).

When I began to work as a graduate in 1956, the scene was dominated by Dr Willem Feekes, naturalist, wheat physiologist and plant breeder. He was a visionary man and he had a keen eye for good wheat varieties (he represented some German wheat breeders in the Netherlands) though as a wheat breeder he was not overly successful. He introduced the "Feekes scale" for the recording of wheat growth stages (Feekes 1941), later adapted as the Decimal Code for use in chemical control, plant breeding and computer applications (Zadoks *et al*. 1974). Feekes' great contribution was to bring European wheat breeders together in a loose co-operative network of which the successful "International Yellow Rust Trials" (Zadoks 1961; Stubbs *et al*. 1974) was but one of the many materialisations (Zadoks 1985).

I was a member of the COGEM during ten years of which five years as the chair of the sub-committee on plants. The major concern was environmental safety of genetically modified crops. Trained in classical population genetics I had to work hard to become sufficiently knowledgeable in molecular genetics without ever having done laboratory bench work. Modern molecular genetics still hides many secrets for me but I feel familiar with the major issues and results.

Three stages of resistance breeding in crop plants

I see three stages in the history of resistance breeding of which one stage has yet to come. The first is breeding for vertical resistance, the second

is breeding for horizontal resistance, the third is breeding to promote symbionts and natural enemies of pests and diseases (Zadoks 2002). I will briefly discuss these stages.

Stage 1. The story of breeding for *R*-genes, usually conferring complete resistance by means of single dominant genes, is well known. The failure to breed potatoes for resistance to potato late blight (*Phytophthora infestans*) by using single *R*-genes from *Solanum demissum* is a good example of solid breeding work annihilated by the mutability of the pathogen. However, success stories exist too (Eenink 1976). Present day control of wheat black (stem) rust (*Puccinia graminis*) in the USA is due, at least in part, to the stacking of *R*-genes. Today, *R*-gene resistance is still easy to use and at least temporarily effective.

Stage 2. The disappointment with *R*-gene or "vertical" resistance led to a search for alternatives. These were found in new concepts such as "incomplete" (Zadoks 1972), "partial" (Parlevliet 1979) or "horizontal" (Vanderplank 1963; Robinson 1976) resistance. The usual hypothesis is that several genes, each with small effects, together provide a broad-spectrum resistance which may be incomplete but operates more or less equally against all known races of a pathogen. Theory is advanced, as became apparent at two international conferences on durable resistance (Jacobs & Parlevliet 1993; Govers *et al*. 2002), but practice lags behind because of technical problems in plant breeding.

Stage 3. When attacked by insects or mites, "plants cry for help" (Dicke *et al*. 1981, 2004; Dicke 1996), secreting pheromones with attract predators or parasitoids. As variations in pheromone production exist among plant varieties, pheromone production is a selectable character. Similarly, variation in mycorrhizal density exists, another interesting selectable character (e.g. Bertheau *et al*. 1980). I surmise, without any experimental evidence, that in the case of fungal plant diseases the relative favourableness of the phyllosphere for antagonistic organisms is a variable and hence selectable character. What about selecting wheat lines that favour rhizosphere-inhabiting *Pseudomonas* bacteria which induce resistance (van Loon *et al*. 1998)? I have not yet seen any published examples of stage 3 resistance breeding.

The actual situation with wheat is between stages 1 and 2. Minor genes for resistance seem to be common, but since major genes for resistance are epistatic over minor genes, we usually do not know how many minor genes are present in a variety and to what effect. There is evidence that "broken" *R*-genes, that is *R*-genes matched by new virulence genes (to use old-fashioned parlance), may have a "residual" effect (Zadoks 1961) not unlike to the effect of a minor gene. The "gene-for-gene" hypothesis of major genes was extended to minor genes (Parlevliet & Zadoks 1977) and evidence was provided by Parlevliet (1979).

Today's challenge, septoriosis

Septoriosis in wheat is caused by either one of two different fungi, *Stagonospora nodorum* (anamorph *Septoria nodorum*) which causes glume blotch, and *Leptosphaeria graminicola* (anamorph *Septoria tritici*) causing leaf blotch (Cunfer & Ueng 1999; Lucas *et al*. 1999). The typical *nodorum* symptom, shrivelling of the nodes followed by stem breaking, was and is relatively rare. Both fungi cause leaf flecks and eventually leaf decay and both may affect the heads, but only *nodorum* causes severe damage to the heads. Septoriosis as a collective concept is today's challenge for wheat breeders.

A hundred years' history of septoriosis (Zadoks 2003) shows that it becomes serious about every 25 years, once per human generation. Severe epidemics (possibly of *tritici*) occurred somewhere in Europe just before 1900, whereas heavy *nodorum* epidemics were experienced around 1930 and again after 1950. *Tritici* went to the nearly worldwide attack around 1970 and it is still a fearsome fungus. Whereas Shipton's review (Shipton *et al*. 1971) ignored the epidemiological function of the teleomorphs, the European Handbook of Plant Diseases (Smith *et al*. 1988) stated for both fungi that new infections are caused by ascospores. This statement is, however, too simple (McDonald & Linde 2002).

Nowadays, both fungi can survive the off-season as teleomorphs and can infect new crops by ascospores, but mating, recombination and formation of ascospores with new genetic properties can also occur during the wheat growing season. Short and medium distance dispersal occurs by ascospores. Long distance dispersal occurs by ascospores and by anamorphs on seed, primarily with *nodorum* but probably also with *tritici*. This statement is supported by the high genetic uniformity of fungus populations over large distances, even worldwide, relative to the within-field genetic uniformity. In *nodorum*, early fall infestation often consists

of small clonal foci suggesting the effectiveness of seed transfer and short-distance conidiospore dispersal (Shah *et al*. 2001).

Several factors conspired to promote septoriosis since 1970 (i.a. Shaw & Royle 1993), apart from favourable weather (Pietravalle *et al*. 2003). Of course, *nodorum* feels at home in the moist shaded mountain valleys of the Alpine region. *Nodorum* feels fine on late sown crops and even more so on spring sown crops. A *tritici* infection can be heavy and uniform on early fall-sown crops as I could see myself in Dutch wheat fields measuring 60 hectares. In the course of time the dosage of nitrogen fertilisers increased gradually, which was appreciated by both fungi. Very high dosages, however, seem to favour *tritici* above *nodorum* (Zadoks, unpublished). Similarly, tillage operations in the course of time became more sloppy, with less attention to field hygiene. Chemical weed control provides survival opportunities to the fungi on dead plant material. More standing wheat straw survived the summer with its suite of *tritici* and *nodorum* inoculum. Reduced tillage left more infective straw on the ground. Undersowing wheat with green manure crops strongly promoted the summer survival of and fall infections by the two anamorphs ("green bridge effect" – Zadoks 1984). Nonetheless, these historical trends do not suffice to explain the worldwide septorioses of the recent decades.

The unthinkable has to be thought, wheat breeding as a decisive factor. Indeed, wheat breeding has an immense effect on wheat disease epidemiology. Think of Egypt, plagued by black stem rust (*Puccinia graminis*). When this problem was eliminated by aggressive wheat breeding in American style, brown leaf rust (*P. recondita*) became severe. Having eliminated that problem the table was set for yellow stripe rust (*P. striiformis* – Mohamed 1963). The high pressure on wheat breeders may have led to sharply targeted haste work neglecting other potential threats. The USDA (1953) stated explicitly that the intensive resistance breeding against wheat rusts had led to the incorporation of susceptibility to septoriosis. Brown *et al*. (2003) took the matter one step further by suggesting that the use of one single variety (Heines Peko was mentioned in an oral presentation) as a resistance donor against yellow rust was at the root of all *tritici* problems in the United Kingdom. Indeed, I believe that the plant breeders are a major cause of the secular changes in wheat disease epidemiology with its periodic outbursts of septoriosis, roughly once per human generation. The trend toward dense crops with short stature is good for the septorias (*i.a*. Danon *et al*. 1982; Simón 2003).

Molecular septoria epidemiology

Host and pathogen populations respond to mutual selection pressure. Pathogen genetics is very advanced in comparison with host genetics of septoria resistance (Kema *et al*. 2003). I will discuss some aspects of pathogen genetics leaving aside the topic of host genetics. To this purpose I distinguish genes under selection pressure, such as genes for virulence, from selection-neutral genes, as identified by e.g. RFLP.

Fungal genes under selection pressure

The existence of differential interaction and physiologic specialisation in the septorias was denied until 1970 (Shipton *et al*. 1971). Eyal *et al*. (1985) presented good evidence for the existence of a major divide in the world's *tritici* population, separating a bread wheat from a durum wheat population. Kema *et al*. (1996a) showed differential interactions within each of these two populations and he gave the populations varietal rank. The disease parameters necrosis (N) and pycnidia (P) were under different genetic controls. A typical gene-for-gene system was identified (Kema *et al*. 1996b, 2000; Brading *et al*. 2002), for the time being with one resistance gene only.

Ahmed *et al*. (1996) demonstrated within-season adaptation of local *tritici* populations to their respective hosts, indicating selection for aggressiveness as well as cultivar specific but supposedly non-monogenic virulence. The variety Gene, selected for its high vertical resistance, rapidly became so diseased that its cultivation was discontinued (Cowger *et al*. 2000). Obviously, wheat breeders must avoid that risk by leaving single vertical resistance genes unused. Typical physiological races are now found in *tritici* (McCartney *et al*. 2002).

The response of the wheat hosts to the selection pressure exerted by the adaptive pathogen usually is a gradual loss of productivity. The phenomenon was demonstrated clearly, but without specifying diseases, in the 1970s (de Jong 1981). The potato researcher Niederhauser introduced the term "erosion of resistance" (McDonald & Linde 2002). Mundt *et al*. (2002) stated explicitly that "erosion

of resistance against leaf blotch" in wheat did occur. Presumably, such erosion of glume blotch resistance may occur too but I have seen no clear evidence yet.

Selection neutral fungal genes

Leaf blotch. *Mycosphaerella graminicola* is rapidly becoming the *Drosophila* of the fungi, or at least the *Arabidopsis* of the *Sphaeriales*, that large fungal order with so many plant pathogens of economic importance. Among the great achievements are (1) the genetic map with location of mating type and avirulence loci, (2) the molecular population genetics, and (3) the study of ABC transporters.

Kema *et al*. (1996c) applied a RAPD-based tetrad analysis to eight isolates supposedly from one ascus. Three classes of polymorphic patterns were distinguished. The isolates were grouped into three pairs with one related isolate and one deviant isolate which was discarded. Pairwise crosses were made *in planta*. Segregation for growth type on agar pointed to a bipolar heterothallic mating system. In the field the teleomorphic cycle could be completed within five weeks. Ascospore discharges were registered in spring and summer. Hence, polycyclic teleomorphic epidemics are possible so that the picture of a strict separation between the establishment of an epidemic by ascospores and the clonal build-up of the epidemic by pycnidiospores needs revision. Plant breeders should remember that continuity in pathogenicity may not exist and that next year's fungal population need not resemble this year's population. The postulate of quantitative resistance of wheat against leaf blotch is ready for revision. The apparent quantitativeness of resistance may in fact result from a large number of small differential interactions (Kema *et al*. 1996b). Is the conceptual framework of Parlevliet and Zadoks (1977) still appropriate or do we need a new one?

The creation of a genetic map of *tritici* (Kema *et al*. 2002) with 282 markers in 23 linkage groups including the mating type locus *MAT* on linkage group 16 and the first avirulence locus *AVR1* on linkage group 22 is an outstanding achievement. It opens the road to the study of evolutionary trends in the family of the *Mycosphaerellaceae* and possibly in the order of the *Sphaeriales*.

Frequency of mating, resulting in teleomorphs, increases with the intensity of the epidemic, the intensity being furthered by host susceptibility and wet weather (Cowger *et al*. 2002b). We may readily assume that the more mating occurs, the more recombination will take place with development of new aggressive (Cowger & Mundt 2002a) and/or fungicide-resistant strains. The species *Mycosphaerella graminicola* is in a stage of rapid evolution.

Molecular analyses of leaf blotch at different scales of distance have shown remarkable results (McDonald & Martinez 1990). At the smallest scale, the mm scale (that of the single pycnidium), different isolates are identical as it should be expected. At the cm scale (that of the lesion) coinfection by up to six haplotypes was found (Linde *et al*. 2002). At the 1 m scale (the scale of the "spot" or "focus") haplotype diversity was rather low, suggesting that clonal multiplication from founder haplotypes occurred. At the 10 m scale (the field scale) haplotype diversity became high, practically as high as the regional diversity at the km scale. The region may be quite large stretching over 750 km in the USA (Boeger *et al*. 1993). At the intercontinental scale genetic identity between populations is high (Zhan *et al*. 2002) though gene flow and genetic drift may not yet have reached an equilibrium (Linde *et al*. 2002).

The leaf blotch populations of the Mediterranean region seemed to be fairly uniform, irrespective of the difference between *durum* and *aestivum* subpopulations. Note that we still talk about nuclear haplotype diversity characterised by alleles of anonymous, supposedly selection-free genes. However, the pace of evolution differs between nuclear and mitochondrial DNA. Mitochondrial DNA is not necessarily evolutionary neutral. At the level of mtDNA specialisation occurred in the Mediterranean basin for *durum* versus *aestivum* populations (Zhan *et al*. 2004). Specialisation in a "selective sweep" was followed by conservation as suggested by the remarkably low mtDNA diversity within both the *durum* and *aestivum* populations.

Glume blotch. Surprisingly, the genetic situations of glume blotch and leaf blotch do not differ so much in view of their wide-spread random mating systems. A genetically diverse founder population leads to high in-field diversity distributed on a fine scale (McDonald & Martinez 1990; McDonald *et al*. 1994). In-field haplotype diversity may increase during the season by two other processes, immigration and hybridisation. Immigration occurs by wind-borne ascospores and splash- or wind-borne pycnidiospores. Hybridisation occurs

when the disease intensity reaches the level that different mating types meet *in planta*. The diversity generating processes in the glume and leaf blotch fungi may differ in timing and intensity and vary according to location and historical period. Genetic uniformity of early glume blotch foci suggested clonal descent from single anamorphic seed infections (Shah *et al*. 2001) whereas the high diversity and relative uniformity of the total Swiss *nodorum* population (Keller *et al*. 1997a, b) pointed to an ascospore-mediated gene flow over hundreds of kilometres. Gametic equilibrium indicated random mating at least within populations.

Has classical plant breeding an answer?

In my early days I listened to endless talks about breeding strategy, talks without issue. One strategy is to build up a gene pool, plant it in the breeding block, and make crossings. Over the years, such material will have been exposed to so many influences that there is great safety in the material. Vertical genes may have been eliminated gradually and horizontal resistance may accumulate. This idealistic picture will be disturbed by the necessities of the day, e.g. short stature or yellow rust resistance, which require the introduction of new and unknown material with equally new and unknown effects, effects often with long delays as e.g. *septoria* sensitivity. In worn-out terminology, new germ plasm may pervade the carefully constructed gene pool with pernicious results.

Breeding for horizontal resistance has no standard recipe yet, see Euphytica (2002), Vol. **124**, No. 2, entirely devoted to the subject of Durable Resistance. Elimination of vertical resistance, so that the hypostatic horizontal resistance becomes visible, is recommended but the required virulence genes of the pathogens (plural!) are not usually available. Artificial infection of selection material in greenhouse and field partly compensates the deficiency, but unfortunately such artificial infection is inevitably done with old pathogen genotypes even if the stock of pathogen genotypes is replenished every year. The results do not predict any future eventualities caused by new genotypes which in both our septorias are generated constantly by recombination.

The usual safeguard of the breeder is multilocational testing, including testing in "hotspots" in the breeder's target area (e.g. Czekia, Europe) and beyond. How many years should be spent on multilocational testing? And how many breeding lines should be tested everywhere? The more the better, but the numbers of locations, years and breeding lines add to the breeder's expenses to which there is a limit. The decision of the "6th International Symposium on *Septoria*/*Stagonospora* Diseases of Cereals", Tunisia 2003, to try and organise international trials for septoria resistance with the help of CIMMYT (Mexico), in the same vein as the long abolished "International Yellow Rust Trials", is a step forward to multilocational and multiyear testing.

Chemical control of the two septorias is feasible and common, though resistance against specific fungicides may appear (De Waard 1997). Within-season population monitoring of the septoria populations could help to decide which fungicides to avoid. Population monitoring could also help to decide on the composition of the population to be used for artificial inoculation. Plant breeders once showed the tendency to rely on pesticides and to give up resistance breeding against chemically controllable diseases. Today plant breeders must respond to the public pressure to avoid pesticides. They are expected to create varieties with sufficiently high resistance to grow them without pesticides, at least in a "normal" season.

The question remains to answer whether artificial inoculation of test nurseries should be done with one potent mixture of fungus genotypes or with a limited number of carefully selected fungal genotypes. It is my feeling that the former approach may provide unreliable information because selected genotypes may be lost by intergenotype competition whereas new genotypes may appear by within-season hybridisation. The latter approach provides some protection against these undesirable effects and creates far more information including some information on specificities of pathogen virulence and host resistance, but the costs multiply with the number of pathogen genotypes tested.

Genetic modification and other alternatives

Genetic modification of wheat is perfectly feasible and not expensive (Pellegrineschi *et al*. 2000). Ignoring for the moment the aversion to genetic modification of the European public, we may ask what modifications are interesting to enhance resistance of wheat to the septorias. In my view genes for resistance taken from other cereals or

grasses should not be used as they will probably act as single vertical genes. In this context we remember the dramatic effects of the interspecific crosses between rye and wheat. The transfer of a minor piece of rye chromosome to wheat yielded a resistance to the brown and yellow rusts "which should last forever". In the course of time the resistance was overcome by either pathogen and in retrospect these resistances acted as simple vertical *R*-genes (Zadoks & Bouwman 1985). By the way, transfer of resistance genes against *tritici* from durum to aestivum wheat, advocated by some, will probably lead to a disillusion because the nuclear genotypes of the *tritici* populations on *T. durum* and *T. aestivum* are very similar.

More interesting are genetic modifications which enhance the host's defences by stimulating e.g. pathogenesis related (PR) proteins (Jongedijk *et al*. 1995; van Loon 1997). In doing so we choose the road of horizontal resistance, to use this outdated term once more. Early selection by means of genetic and proteomic chips seems feasible. Whether such a selection leads to the desired result remains to be seen.

A simpler biotechnical approach avoiding genetic modification is the search for QTLs for partial resistance to septoria. The process is costly and the testing has to be repeated in at least two successive years because of interactions of putative QTLs with the environment. Whether these QTLs will really protect the wheat against septoria remains to be demonstrated.

Alternative possibilities are cultivar mixtures and participatory plant breeding. Cultivar mixtures have proven effectiveness in reducing septorias (Jeger *et al*. 1981; Belhaj Fraj *et al*. 2003). Participatory plant breeding with adaptation of breeding lines to the requirements of local farmers is a new trend in developing countries (Cleveland & Soleri 2002), which might be interesting for special problem areas in Europe. Both approaches may conflict with the existing EU regulations on variety registration and uniformity and they may be rejected by wheat merchants and wheat processors who prefer large and uniform lots of wheat.

Postscript

These are the musings of a long-retired plant pathologist who often worked in close contact with plant breeders. Due to lack of knowledge and modesty these reflections have an autobiographic

stint. Thanks are due to Drs Hanisova, Horcicka and Sip for inviting me to write this paper. Such writing would have been impossible without the generous invitation by the organising committee of the "6th International Symposium on *Septoria /Stagonospora* Diseases of Cereals" to present the keynote lecture in Tunis (Tunisia, December, 2003) from which parts have been used in the present paper.

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Souhrn

Zadoks J.C. (2004): **Šlechtění pšenice orientované na odolnost k braničnatkám z pohledu fytopatologa.** Czech J. Genet. Plant Breed., **40**: 63–71.

Studie přináší osobní pohled fytopatologa na vývoj šlechtění rostlin. Týká se jednak obecných trendů rezistentního šlechtění ve třech etapách, šlechtění s podílnickou účastí pěstitelských firem a biotechnologií ve šlechtění. Pozornost je soustředěna především na molekulární studie, které v současné době probíhají u braničnatky pšeničné (anamorph *Septoria tritici*) a braničnatky plevové (anamorph *Septoria nodorum*) na pšenici. K výraznému pokroku došlo na úseku epidemiologických studií u teleomorfních stadií hub *Mycosphaerella graminicola* a *Stagonospora nodorum*. Výsledky tohoto výzkumu se však v praktickém šlechtění prosazují jen pomalu.

Klíčová slova: braničnatka plevová; braničnatka pšeničná; molekulární epidemiologie; *Mycosphaerella graminicola; Septoria nodorum; Septoria tritici; Stagonospora nodorum*

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