REVIEW

Genetic Basis of Oat Resistance to Fungal Diseases

JOSEF ŠEBESTA[1], HYWEL W. RODERICK[2], SRBOBRAN STOJANOVIĆ[3], BRUNO ZWATZ[4], DON E. HARDER[5] and LUCIANA CORAZZA[6]

[1]Research Institute of Crop Production, Prague-Ruzyně, Czech Republic; [2]Institute of Grassland and Environmental Research, Aberystwyth, The United Kingdom; [3]Centre for Small Grains, Kragujevac, Yugoslavia; [4]Federal Office and the Research Centre for Agriculture, Vienna, Austria; [5]Cereal Research Centre, Agriculture and Agri-Food Winnipeg, Manitoba, Canada; [6]Plant Pathology Research Institute, Rome, Italy

Abstract

ŠEBESTA J. *et al.* (2000): **Genetic basis of oat resistance to fungal diseases**. Plant Protect. Sci., **36**: 23–38.

Race specific and race non-specific resistance and tolerance of oat genotypes to fungal diseases are characterized. Race specific resistance of oat to fungal diseases is conditioned by single dominant, partially dominant, less by recessive or incompletely recessive independently inherited genes but also by genes in interaction, e.g., by complementary genes. Race-non-specific resistance (slow diseasing) and tolerance are mostly conditioned by recessive genes with additive effects. It is supposed that molecular markers associated with desirable traits will increase the efficiency in disease resistance. Markers based on DNA or endosperm protein polymorphisms were used for identification of crown rust and stem rust resistance genes. Problems of genetic control of oat diseases is discussed. In Canada and in Europe the oat breeding for resistance to crown rust and stem rust is oriented for the production of multigenic cultivars. Owing to high variability of powdery mildew and deficiency of major resistance genes the combination of races specific and adult plant resistance (APR) governed by additive genes is very desirable.

Key words: oat; fungal diseases; resistance; tolerance; molecular markers; *Puccinia coronata* f. sp. *avenae*; *P. graminis* f. sp. *avenae*; *Erysiphe graminis* f. sp. *avenae*; *Phaeosphaeria avenaria* f. sp. *avenaria*; *Pyrenophora avenae*

Oat is considered to be an important cereal crop despite the considerable decline in the world oat (*Avens sativa* L.) acreage in the last few decades. The nutritional value of oat grain and its beneficial effects on human and animal health are now well known and accepted (ŠEBESTA *et al.* 1995a, b; ŠEBESTA & SÝKORA 1974). However, oat is subject to a number of diseases worldwide that are assumed to be major limiting factors in oat production (HARDER & HABER 1992; ŠEBESTA 1971, 1987, 1991, 1992, 1993, 1994, 1995, 1996, 1997, 1998). The most feasible and economic means to control significant diseases, and thus contribute to the stability of grain yield and quality, is by manipulating inherant plant resistance (ŠEBESTA 1991a. b; ŠEBESTA & ZWATZ 1980; ŠEBESTA & HARDER 1983; ŠEBESTA *et al.* 1995a, b).

A. sterilis, the hexaploid progenitor of cultivated oats, was recognized as an excellent donor of new desirable genes many years ago. Though oats are a classic example of a crop species with a demonstrably narrow gene pool, and introgression of genes from *Avena* spp. with other ploidy levels has been of very limited value, *A. sterilis* has proved to be a treasure-house of genes for broadening the cultivated oat gene pool (FREY 1994). Every trait studied so far has contributed useful genes, e.g., resistance to crown rust (*P. coronata* f. sp. *avenae)*, stem rust (*P. graminis* f. sp. *avenae)*, powdery mildew (*Erysiphe graminis* D.C. f. sp. *avenae* Em. Marchal) and tolerance to barley yellow dwarf luteovirus (BYD), as well as grain protein and oil content and vigour traits. Studies conducted on *A. sterilis* itself indicate this species posesses immense genetic variability in other traits such as leaf and panicle characteristics, photoperiod and vernalization respenses, and beta-glucan content (FREY 1994). The alleles from *A. sterilis* can be diverse or complementary to those alleles present in the *A. sativa* gene pool and as emphasized by FREY (1994), oats may survive as a crop only because of the genes introgressed from *A. sterilis*.

Transfer of some of these traits, including resistance, have been commercially important. The high yielding North American oat cvs. Hamilton and Sheldon have inherited both cytoplasmic and an unknown proportion of nuclear genes from *A. sterilis*. In the 1970s, experimental oat lines with 20–22% groat protein content derived from alleles from *A. sterilis* were developed. Though none of these were released as cultivars directly, their use as parents has been a factor in the general elevation of groat protein content in the past 15 years (FREY 1994). During

*The financial support of the Czech National Agency for Agricultural Research (Grant No. EP 7243) is very acknowledged.

the same time period, experimental lines have been developed with 50% higher groat oil content than either *A. sativa* or *A. sterilis*; however, new cultivars with high groat oil content have not been released to date (FREY 1994). In recent years, resistance from *A. sterilis* to crown rust (*Puccinia coronata* f. sp. *avenae* Eriks.) and BYD has been deployed in commercial cultivars in the USA and Canada.

This review focuses on genetic basis of oat resistance to crown rust, stem rust and powdery mildew in particular as clarified especially in the recent research.

Terms and definitions

Plant breeders are interested in description of resistance in terms of its expression, known inheritance and effectiveness. According to BROWDER (1985) resistance, including escape, is a characteristic of a host plant limiting the damage produced by disease. Basically, the resistance of cereals to diseases is usually classified as race specific (vertical) and race non-specific (horizontal) (VANDERPLANK 1968).

Race specific resistance is usually characterized by a visible hypersensitive response. It is effective against some races (isolates) of the pathogen but not against others.

Race non-specific resistance is supposed to be effective against all races of the pathogen. HARDER & HABER (1992) proposed to use the term race non-specific for the rate-limiting epidemiological types of resistance such as slow rusting. The rate of multiplication of a pathogen is decreased by reduced receptivity (infectibility) of a cultivar, longer latent period and reduced rate and duration of sporulation of pathogen (HARDER & HABER 1992; KOCHMAN & BROWN 1975; LUKE *et al.* 1981; WILCOXSON 1981). However, according to HARDER & HABER (1992) the terms specific (vertical) and non-specific (horizontal) characterize forms of resistance which probably do not exist in such explicit terms.

Disease tolerance (FREY *et al.* 1977; SCHAFER 1971; GAUNT 1981) is a property of a cereal cultivar that enables it to compensate for an attack by the pathogen to a certain extent. So that if two cultivars are equally susceptible to a particular diseases but the yield reduction is less with one than the other it is described as being more tolerant (ŠEBESTA 1980a, b). The tolerance might be different from rate-limiting resistance by a combination of yield/kernel weight determinations and spore production (HARDER & HABER 1992; POLITOWSKI & BROWNING 1978).

Quantitative assessments can be made by measuring the generation time which often indicates **partial resistance** to pathogens such as rusts and mildew (JONES & CLIFFORD 1978). A partial resistance results in the relatively slow development of fewer, smaller pustules of a compatible type from a given quantity of inoculum with little or no evidence of a necrotic host response. Such partial resistances are called **slow rusting** or **slow mildewing** types describing epidemiological consequences of resistance (JONES & CLIFFORD 1978).

Inheritance of Specific Resistance

The ***specific resistance*** of oat to fungal diseases is mostly conditioned by a single dominant or partially dominant gene, less by recessive or incompletely recessive genes and by genes in interaction (complementary genes) (BAKER & UHPADHYAYA 1967; ŠEBESTA 1979a, b; SIMONS 1985; SIMONS *et al.* 1978). The degree of dominance was found to be dependent on the genetic background and also varies with races (isolates) of the pathogen (ŠEBESTA 1979a, b; SIMONS 1985).

The dominance and the independent inheritance enable simple manipulation and the transfer of disease resistance genes to agronomically valuable cultivars (SIMONS 1985). Linkage or allelism commonly occur among crown rust and stem rust major resistance genes as well (MARTENS *et al.* 1968; MCKENZIE *et al.* 1965a, b, 1968a, b, 1970; SIMONS *et al.* 1978; WONG *et al.* 1983).

OSLER and HAYES (1953) found no evidence for linkage between crown rust (*Pc*) and stem (*Pg*) resistance genes and genes for date of heading, number and length of basal hairs, percentage of lower florets awned, strength of awns and plumpness of seed. Also, in a study by KIEHN *et al.* (1976) the genes for seed colour and awn character did not appear to be linked to the *Pc*-genes in *A. sterilis*. WONG *et al.* (1983) could not find an association between crown rust resistance and some floret characters in *A. sterilis*.

However, the *Pg11* gene for stem rust resistance was found to be associated with yellow-green plant colour, weak straw and reduced grain yield (HARDER *et al.* 1971), and linkage between a *Pc*-gene for crown rust resistance and low yield was found in *A. sterilis* (SIMONS 1979). On the other hand, FREY and BROWNING (1971) found *Pc*-genes for crown rust resistance associated with significant yield increase in *A. sterilis*.

HUMPHREYS and MATHER (1996) used parent-offspring regression to estimate heritability for beta-glucan content, groat percentage and the resistance to crown rust in oat. The populations used were derived from two crosses, Nova × Marion QC and Sylva × Marion QC. Marion QC was used as a parent because other research had shown that it was relatively high in beta-glucan content, a trait for which heritability had not previously been estimated. Nova and Sylva are similar in adaptation to Marion QC, and Sylva may be a source of general resistance to crown rust. Random F_6 and F_7 progeny were grown in replicated field trials. Heritability estimates for the two grain quality traits were based on regression of F_6 values on F_5 values, F_7 values on F_6 values, and F_7 values on F_5 values, and found to be between 0.27 and 0.45. The highest estimate was the one based on the F_6 and F_7 generations of Sylvia × Marion QC; 0.45, compared to estimates of 0.32 or less for earlier generations of the same cross and to estimates of 0.35 or less for all generations of Nova × Marion QC. Heritability estimates for groat percentage were all between 0.23 and 0.32.

The F_6 and F_7 generations were evaluated for resistance to crown rust resistance. The Sylva × Marion QC cross seemed to segregate for heritable resistance (h^2 = 0.31) but the Nova × Marion QC cross did not (h^2 = 0.07). Several lines from the Sylva × Marion QC cross had low symptoms in both the F_6 and F_7 generations. There were strong genetic correlations among the traits.

Allelism of major genes for resistance is of importance as the resistance conferred by allelic genes cannot be combined in the same line. The majority of studies have shown that genes for resistance to crown rust in unrelated oats are generally at different loci. Exceptions, however, have been found (UPADHYAYA & BAKER 1965).

GREGORY and WISE (1994) investigated the inheritance of resistance to an isolate of crown rust, race 276, in four diploid oat lines. F_3 families derived from *A. strigosa* (*A. nuda*) C.I.2630 × *A. nuda* C.I.9009, *A. wiestii* C.I. 1994 × *A. strigosa* C.I.3815 and C.I.2630 were inoculated at the seedling stage and evaluated 9–12 days later. Segregation of the F_3 families indicated that C.I.2630 and C.I.3815 each possess one dominant gene that conditions resistance to race 276. The alleles conferring resis-

tance occur at two different loci, separated by 17 cm on the same chromosome, and appear to be complementary. C.I.3815 may have an additional recessive resistance allele at a different locus on the same chromosome, 19 cm from its other resistance gene locus.

It is also supposed that combined genes might be additive in effect (FINKNER 1954; ŠEBESTA – unpublished). The significance of such transgressive segregation under field conditions has been investigated by JONES and RODERICK (1986b) in oat powdery mildew resistance

Use of near-isogenic lines in analysing race-specific pathogen interrelations

The topic has been reviewed by MAC KEY (1992) with particular reference to cereals under the following headings: gene-for-gene types of plant resistance; genic inventories of pathogen populations; and general fitness of virulence genes.

Examples cited include rice and *Xanthomonas oryzae*, oats and *Puccinia coronata/P. graminis* and wheat and *Erysiphe graminis*. The review explores the reasons why different pathogens may behave differently in the same environment and the same pathogen show different patterns in different ecological niches.

An understanding of these reasons, which can be gained by the use of near-isogenic lines, is seen as essential for proper planning of resistance breeding. It is argued that virulence genes may have pleiotropic functions, that the need for host alternation in addition to the dikaryotic/diploid constitution favour a preadaptive accumulation of virulence genes, and that breakdown of a newly introduced resistance may depend on preadaptation as much as on a mutational event.

Peculiarities in inheritance of race specific disease resistance

Complete and incomplete dominance for crown rust resistance has been found in the oat cv. Delphin (ŠEBESTA 1978, 1979a, b). The resistance was found to be conditioned by two complementary genes and showed complete dominance to eight races, the F_2's segregating in 9:7 ratio. On the other hand, the same genes behaved differently in relation to two other races, the F_2 segregating in 5:11 ratio. The double heterozygotes were resistant to the former race group, but susceptible to the latter. Further experiments showed that the same genes were responsible for the resistant reaction to all avirulent races (ŠEBESTA 1979a, b). The incomplete dominance of complementary genes observed by BAKER and UPADHYAYA (1967) in the cv. Bond and in cv. Delphin could be explained by assuming a heterozygotic constitution for pathogenicity in some races of the pathogen.

Race specific resistance to crown rust conditioned by major and minor genes was found in the oat line *Pc 50-2*. In the reselected lines *Pc 50-2* and *Pc 50-4*, giving identical immune reactions to many crown rust races, an unusual different reaction to the Polish crown rust isolate 7-77 P was found. Whereas *Pc 50-4* was highly susceptible and severely attacked, *Pc 50-2* showed a reproducibly very low occurrence of resistant infection types. Genetic analyses of these lines to rust isolate avirulent to both lines indicated that each of them contained a different major resistance gene. Moreover, the crown rust resistance in *Pc 50-2* was shown to be influenced by a group of minor genes controlling the infection severity and the frequency of infection types when infected with rust isolate 7-77 P (ŠEBESTA 1983).

Inheritance of Non-Specific Resistance

Non-specific resistance of plants to diseases, characterized by a reduced rate of multiplication of the pathogen (*slow diseasing*) has been found to occur in oats to crown rust (HARDER *et al.* 1984; KOCHMAN & BOWN 1975; LUKE *et al.* 1972, 1975, 1981; SIMONS 1975, 1985).

Characterization and determination components of partial resistance

BRAKE and IRWIN (1992) characterized several oat cultivars to determine components of ***partial resistance***. Panfive was the only cultivar to exhibit partial resistance; it gave a moderately susceptible reaction but the infection efficiency was lower, the pathogen developed fewer and smaller uredia and produced fewer spores than on the susceptiblle cvs Algerian and Sual. In the field, cv. Panfive has consistently shown partial resistance. Cv. Garry, which has been reported to be slow rusting in the glasshouse, but highly susceptible in the field in Queensland, Australia, expressed a significantly lower infection efficiency and uredium density than cvs Algerian and Sual on the first leaf. Older leaves of cv. Garry, however, did not show a significant reduction in the components of partial resistance when compared with Algerian and Sual. The results suggested that uredium density gave the best indication of partial resistance across the three leaf ages tested for cv. Panfive. A significant difference was present between uredium densities on upper and lower surfaces of the fourth leaf only for the susceptible and partially resistant cultivars. In general, the resistance of all cvs tested tended to increase with an increase in leaf age.

Slow rusting is recognized by the reduced development of rust during an epidemic and determined by comparing disease progress curves (SKOVMAND *et al.* 1978) or rust severity during the logarithmic stage of development of an epidemic (WILCOXSON 1981). The rates of rust development are calculated with the logistic and Gompertz models. The latter transformation was found to be more consistent at detecting slow rusting (LUKE & BERGER 1982). But, the *area under disease progress curve* (AUDPC) was found to be better suited for primary data processing of powdery mildew in wheat (FORMANOVÁ & ŠEBESTA 1994).

BRIERE *et al.* (1994) carried out a screening for partial resistance to an isolate of crown rust in oat cultivars and breeding lines. Crown rust severity was visually estimated as percentage leaf area diseased at weekly intervals commencing at approx. Zadoks growth stage 37, on the flag leaf and on the four preceding leaves on each of four randomly selected plants/plot. The parameters used in the data analysis were the cumulative proportion of leaf area diseased (CPLAD), averaged for all five leaves sampled, and the proportion of flag leaf area diseased (PFLAD). The CPLAD and PFLAD values were submitted to both principal component and cluster analysis to obtain specific groups of oat cultivars and breeding lines with similar levels of partial resistance to one isolate. The results obtained from the cluster analysis of the CPLAD values gave a group of six oat lines that displayed high levels of partial resistance in both years; OA 712-17, Woodstock, Fidler, Sylva, Ultima and Glen. The cluster analysis of the PFLAD grouped five and eight oat accessions displaying high levels of partial resistance in 1989 and 1990, respectively. The oat accessions common in cluster groupings of both years based on PFLAD were similar to those obtained based on the CPLAD parameter, suggesting that the former could be used in mass screening for partial resistance to crown rust (BRIERE, KUSHALAPPA 1995).

Slow rusting is a heritable trait (WILCOXSON 1981). However, so far there is only a limited amount of information available about heritability of race non-specific resistance.

PARKER (1920) in segregating progenies from a cross between a resistant and a susceptible oat cultivar found that multiple factors were responsible for the resistance. Red Rustproof is typical of a group of oat cultivars having so called ***race non-specific (general), slow rusting resistance***. LUKE *et al.* (1975) found that the resistance of Red Rust-Proof was controlled by a small number of genes showing slight partial dominance for susceptibility. Heritability was high, with a broad sense value of 87% making selection for this resistance practical.

SIMONS (1975) found heritability values of the slow rusting resistance of four unadapted oat strains to range from 46 to 86%, when measured in terms of yield reduction attributable to *P. coronata*, and 65–92% in terms of reduction in seed weight. The relationship of yield to resistance in the absence of rust was generally negative, and none of the lines combined maximum yield with maximum resistance. Thus manipulation of slow rusting resistance in disease resistance breeding programmes will require large populations in spite of the heritability values.

KIEHN *et al.* (1976) showed that the resistance of two strains of *A. sterilis* was controlled by a number of minor recessive genes having additive effects, a type of inheritance commonly associated with slow-rusting resistance.

Use of Molecular Markers in Oat Disease Resistance Breeding and Mapping of Resistance Genes

Use of molecular markers associated with desirable phenotypic traits has great potential for higher efficiency in plant breeding. For example, this would allow efficient combination (pyramiding) of disease resistance genes in multigenic cultivars when pathotypes of the pathogen with the appropriate virulence combinations are not available to differentiate resistance genotypes (MCDANIEL 1992; CHONG *et al.* 1994).

It was shown that molecular markers, based on DNA (PENNER *et al.* 1993a, b) or endosperm protein polymorphisms (HOWES *et al.* 1992), can be used to identify crown rust (*Pc 68* major gene) and stem rust (*Pg 3* and *Pg 13* major genes) resistance genes (CHONG *et al.* 1994).

PENNER *et al.* (1993a) investigated the feasibility of using bulk segregant analysis to identify molecular markers for disease resistance genes, utilizing random primers in conjunction with polymerase chain reaction technology.

Random primers were screened for the amplification of polymorphic DNA fragments on two pools of genomic DNA isolated from plants that were homozygous for the presence and absence of the crown rust resistance gene *Pc 68*. Ten primers were identified that amplified polymorphic DNA fragments, of these, one was tightly linked, in repulsion, to the target gene, while the other nine were not linked to this trait.

The relatively low cost of polymerase chain reaction technology, coupled with rapid leaf disc genomic DNA extraction techniques should result in the effective use of this linked marker in oat breeding selection programmes.

Some studies have been undertaken to determine the map distance between different genes, especially for crown rust and stem rust resistance. CHONG *et al.* (1994) in the cv. Dumont demonstrated the linkage of gene *Pg 13* for oat stem rust resistance with a 56.6-kDa polypeptide locus (map distance of 11.47 ± 2.70 cm) using sodium dodecylsulfate-polyacrylamide gel electrophoresis (SDS-PAGE). A 27.9-kDa polypeptide was shown to be associated with the linked *Pcx/Pg 9* loci by SDS-PAGE but appeared to be more reliably separated as an avenin band, designated B4, using acid-PAGE. Another avenin band, designated B2, also was shown to be associated with the *PcX/Pg 9* loci using acid-PAGE. The loci conditioning the B2 and B4 bands appear to tightly linked or allelic and are separated from the linked *PcX/Pg 9* loci by a map distance of 1.03 ± 0.36 cm.

The association of *Pg13* with a 56.6-kDa polypeptide and the tight linkage between *PcX/Pg 9* and the B2 (in coupling) and B4 (in repulsion) avenin loci found recently (CHONG *et al.* 1994) offer a useful tool to breeders to detect the presence of these genes in oat disease resistance breeding.

In the study of BUSH and WISE (1996), hexaploid oat lines *A. byzantina*, cv. Kanota and *A. sativa*, cv. Ogle were found to differ in their responses to the crown rust isolates, PC 54 and PC 59. Seventy recombinant inbred lines derived from a cross between these two lines were examined. Analysis of rust infection type data indicated that resistance to both of these isolates was due to multiple loci, with major effects exerted by two loci located on linkage groups 4 and 13 of the molecular map developed from this cross. The resistance to the two isolates could not be separated unequivocally and so resistance to both isolates is apparently influenced by each of the two loci identified. These results corroborate the growing evidence that the two homoelogous groups containing linkage groups 4 and 13 contain many disease resistance genes.

BUSH *et al.* (1994) examined near-isogenic lines of hexaploid oat to identify markers linked to genes for resistance to crown rust. RFLPs and resistance loci were mapped using Bcl and F_2 populations. The results showed that three markers were linked to a locus for resistance to crown rust race 203, the closest at 1.9 cm in line D494 and 3.8 cm in line X466-1. In lines D526 and Y345 a marker was placed 1.0 and 1.9 cm, respectively, from the locus conferring resistance to crown rust race 345, and in D486 and X434-II a marker mapped at 8.0 and 10.2 cm from the locus for resistance to rust race 264B.

YU-GONGXIN *et al.* (1996) determined the colinearity of markers linked with resistance loci on linkage group A of diploid oat (from the cross *A. strigosa [A. nuda]* × *A. wiestii*), on the homoeologous groups in hexaploid oat (*A. sativa*). Most of the heterologous probes detected polymorphisms that mapped to linkage group A of diploid oat and two linkage groups of hexaploid oat. Many of these DNA markers appeared to have conserved linkage relationships with resistance and prolamin loci in *Avena*. These resistance loci included the *Pca* crown rust resistance cluster in diploid oat and the R 203 crown rust resistance locus in hexaploid oat. Prolamin encoding loci included Avn in diploid oat. A high degree of colinearity was revealed among the common RFLP markers on the small chromosome fragments among these homoeologous groups.

WISE *et al.* (1996) indicated that the recombination within a 5-centimorgan region in diploid *Avena* reveals multiple specificities conferring resistance to *P. coronata*. A set of 100 recombinant inbred lines (RILs) was produced from a cross of diploid ($n = 7$) *A. strigosa* (*A. nuda*) with *A. wiestii*, resistant and susceptible, respectively, to crown rust. The RILs were inoculated with 11 isolates of *P. coronata*. All isolates detected resistance specificities that mapped to the *Pca* region in *A. strigosa* for resistance to crown rust. *Pca* was positioned between the oat restriction fragment length polymorphism marker Xisu2 192 and a new randomly amplified polymorphic DNA marker, XisuC18, near the end of linkage group A. Five unique specificities within the *Pca* region were differentiated by recombination.

RAYAPATI *et al.* (1994) prepared a linkage map of diploid *Avena* based on RFLP loci and included a locus conferring resistance to nine isolates of *P. coronata* var. *avenae* which was localized.

A F_2 population was produced by crossing the diploid ($n = 7$) species *A. strigosa (A. nuda)* (CI3815) with *A. wiestii* (CI 1994), resistant and susceptible respectively, to 40 isolates of *P. coronata*. Eighty-eight F_2 individuals were used to construct an RFLP linkage map representing the A genome of cultivated hexaploid oat. Some 208 RFLP loci were placed into 10 linkage groups. This map covers 2416 cm, with an average of 12 cm between RFLP loci. A further 88 F_3 lines, derived from the F_2 individuals, were screened for resistance to 9 isolates of *P. coronata*. One locus, *Pca*, was found to confer dominant resistance to isolates 203, 258, 263, 264B, 290, 298, 325A and 345. *Pca* also conferred resistance to isolate 276, although in this case an unlinked second gene may also be involved.

ROONEY *et al.* (1994) identified RFLP markers linked to crown rust resistance genes *Pc91* and *Pc92*. Crown rust resistance from two unadapted *Avena* germplasms was characterized with a view to identifying RFLP markers linked to the respective Pc genes. The resistance genes *Pc91* and *Pc92* were identified from the hexaploid germplasms Amagalon and Obee/Midsouth, respectively. At each locus, crown rust resistance was conditioned by the presence of a single dominant allele. In backcross-derived lines, RFLP markers putatively linked to *Pc91* and *Pc92* were identified, and map distances were calculated using segregating F_2 populations for each gene. RFLP probe UMN145 identified a sequence 4.5 cm from *Pc91* and RFLP probe Og176 identified a sequence 13.6 cm from *Pc92*. Using aneuploid stocks to map the location of RFLP markers, *Pc91* was localized to chromosome 18, while *Pc92* could not be localized to a chromosome, probably because of the lack of the corresponding aneuploid stock. The RFLP markers for these genes may prove useful in efforts to increase the durability of resistance.

LIN-KUOCHIH *et al.* (1996) isolated four cDNA clones (corresponding to *tlp-1*, *-2*, *-3* and *-4* genes) encoding thaumatin-like (TL) pathogenesis-related proteins from oat infected by an incompatible isolate Pg a-1H of *P. g.* f. sp. *avenae*. All cDNA clones contained an open reading frame predicted to encode a 169 amino acid polypeptide with a signal peptide of 21 amino acids. They concluded that the expression especially tlp-1 in oat is associated with resistance reaction in response to infection by incompatible and inappropriate isolates of *P. g.* f . sp. *avenae*.

WOLPERT *et al.* (1994) carried out the identification of the 100-kD victorin binding protein from oats. The fungus *Cochliobolus victoriae*, the causal agent of victoria blight of oat, produces the host-specific toxin victorin. Sensitivity of oats to victorin, and thus susceptibility to the fungus, is controlled by a single dominant gene, which is believed to also confer resistance to the crown rust. In the case of victoria blight, the gene has been hypothetized to condition susceptibility by encoding a toxin receptor. A 100-kDa victorin binding protein (VBP) was identified (GenBank accession number U11693) which may function as a toxin receptor; it binds radiolabelled victorin derivatives in a ligand-specific manner and in a genotype-specific manner *in vivo*.

In vitro translation coupled with indirect immunoprecipitation was used to identify the VBP mRNA, and fractionated mRNAs were used to prepare cDNA libraries enriched in the relative abundance of VBP cDNA. A 3.4-kb cDNA clone was isolated that, when subjected to a 400-bp 5'deletion, was capable of directing the synthesis of a protein in *Escherichia coli* (GenBank accession number L20872), which reacted to an antibody specific for the 100-kDa VBP.

Peptide mapping, by limited proteolysis, indicated that the protein directed by the cDNA is the 100-kDa VBP. Nucleotide

Table 1. Some oat genes of importance for crown rust resistance

Resistance gene	Source of resistance	Remarks	References
Pc-2	cv. Victoria	pleiotropic or closely linked with *Hv-1*	SIMONS *et al.* (1978)
Pc-3	cv. Bond	complementary gene with *Pc-4*	HAYES *et al.* (1939), SIMONS *et al.* (1978)
Pc-5	cv. Landhafer	dominant gene	LITZENBERGER (1949), SIMONS *et al.* (1978)
Pc-6	cv. Santa Fe	dominant gene	LITZENBERGER (1949), SIMONS *et al.* (1978)
Pc-11	cv. Victoria	dominant gene	WELSH & JOHNSON (1954), SIMONS *et al.* (1978)
Pc-22	cv. Ceirch, dubach	Incompletely dominant gene	MCKENZIE (1961), SIMONS *et al.* (1978)
Pc-38	*A. sterilis*, CW491-4	dominant gene	FLEISCHMANN & MCKENZIE (1968)
Pc-39	*A. sterilis*, F366	dominant gene, allelic or closely linked to *Pc-55*	FLEISCHMANN & MCKENZIE (1968); KIEHN *et al.* (1976)
Pc-48	*A. sterilis*, F-158	dominant gene	FLEISCHMANN *et al.* (1971a, b)
Pc-50	*A. sterilis*, CW486	dominant gene, allelic or closely linked to *Pc-46*	FLEISCHMANN *et al.* (1971a, b), SIMONS *et al.* (1978)
Pc-54	*A. sterilis*, CAV1830, 1832	incompletely dom.gene, allelic or closely linked to *Pc-35*	SIMONS *et al.* (1978)
Pc-55	*A. sterilis*, CAV4963	incompletely dominant gene, allelic or closely linked to *Pc-39*	KIEHN *et al.* (1976)
Pc-58	*A. sterilis*, P.I.295919, TAM-0-301	dominant gene	SIMONS *et al.* (1978)
Pc-59	*A. sterilis*, P.I.296244	dominant gene	SIMONS *et al.* (1978)
Pc-60	*A. sterilis*, P.I.287211, cv. Coker 227	dominant gene	SIMONS *et al.* (1978)
Pc-61	*A. sterilis*, P.I.287211	dominant gene	SIMONS *et al.* (1978)
Pc-68	*A. sterilis*, *CAV4904*	dominant gene allelic or tightly linked to *Pg-9* and *Pc 46*	CHONG *et al.* (1994), WONG *et al.* (1983)

sequence analysis of the cDNA revealed extensive homology to previously cloned cDNA for the P protein component of the nuclear-encoded mitochondrial multienzyme complex glycine decarboxylase (glycine dehydrogenase [decarboxylating]). Protein gel blot analysis indicated that the 100-kDa VBP co-purifies with mitochondria. Based on these results, it is concluded that the 100-kDa VBP is the P protein component of glycine decarboxylase.

Genetic Control of Fungal Diseases of Oat

Crown rust

Resistance to crown rust (*P. c.* f. sp. *avenae*) has historically been relatively short-lived, but the discovery of a large pool of major resistance genes in *A. sterilis* has provided the basis for effective control (MARTENS & DYCK 1989).

The breeding of multigenic cultivars, consisting of several resistance genes, as an alternative to multiline cultivars, was proposed and has been practised with success in Canada at the Cereal Research Centre in Winnipeg for a number of years (MCKENZIE *et al.* 1971a, b) and also in Europe (ŠEBESTA & ČERVENKA 1978; ŠEBESTA *et al.* 1983, 1984, 1985, 1989, 1997; ŠEBESTA & HARDER 1983; ŠEBESTA & ZWATZ 1995). The cultivation of multigenic cultivars, especially if connected with spatial (regional) deployment of genes, can considerably prolong the usefulness of the resistance genes (MCKENZIE *et al.* 1971a, b; ŠEBESTA 1988).

A synopsis of studies of inheritance of resistance in terms of *Pc*-genes described has been compiled by SIMONS *et al.* (1978). This catalogue lists 61 genes for crown rust resistance. Most of those reported since the early 1960's have been carried by *A. sterilis* (Table 1). Most of the genes listed have some special characteristic that makes them of value in breeding resistant oat cultivars. At present, the number of *Pc*-genes known is significantly higher.

In a recent European study there were considerable differences in the disease resistance index (DRI) to crown rust among oat lines, ranging from 13 to over 180 (ŠEBESTA *et al.* 1995a, b, c, d, 1997). The highest index values were found mainly in those lines that had highly effective resistance in the seedling stage. The most resistant lines were Pc 68, Pc 58, Rodney ABDH, Rodney E, Pc 50-2, Pc 59 and Pc 39, all of which had a DRI over 170 or higher. Virulence phenotypes of *P. c.* f. sp. *avenae* with different combinations of virulence on resistant lines, were identified in twelve European countries and Israel. The resistance genes *Pc39, Pc55, Pc58* and *Pc68* were effective against all pathotypes. Genes such as *Pc48, Pc50-2, Pc50-4, Pc54-1* and *Pc 59*, however, are of importance for the European crown rust resistance breeding of oat as well CHONG and BROWN (1996) studied the inheritance of resistance to oat crown rust in two heterogeneous resistant accessions, MG 85039 and MG 85181, identified in preliminary screening of 45 oat accessions obtained from the National Research Council Germplasm Institute, Italy. Segregation of F_3 families of the Makuru/MG 85039 cross for resistance to crown rust suggested that this accession had two independent resistance genes, designated *PcA* and *PcB*. Segregation for resistance of F_3 families of the Makuru/MG 85181 cross suggested that this accession had a single gene, designated *PcC*. Resistance to 19, 4 and 10 of 22 crown rust isolates in seedling tests was conditioned by genes *PcA, PcB* and *PcC*, respectively. Gene *PcA* was the most useful source of resistance to crown rust in the Canadian prairie region. It conditioned a high level of resistance to > 97% of all isolates obtained from the annual crown rust surveys in Manitoba between 1991 and 1994. It is linked to gene *Pc 35* and is independent of genes *Pc38, Pc39, Pc45, Pc48, Pc55, Pc62, Pc63, Pc64* and *Pc68*. It is thought that *PcA* has not been previously described and it is assigned the gene symbol *Pc96*. It is suggested that this gene could be a valuable source of resistance for the diversification of resistance gene combinations used in the development of new oat cultivars.

Recently, FOX *et al.* (1997) studied the inheritance of crown rust resistance in four *A. sterilis* accessions (IB1487, IB 2402, IB2465 and IB3432). Each accession had a single incompletely dominant or dominant gene conferring resistance, and genes were designated as Gene *A* in IB1487, Gene *B* in IB2402, Gene *C* in IB2465, and Gene D in IB3432. Gene *A* is allelic or closely linked to *Pc56*. Genes *B* and *C* are allelic or closely linked to *Pc68*. Gene *D* was considered to be a useful addition to existing resistant germplasm.

WISE *et al.* (1993) studied the crown rust resistance in diploid and hexaploid *Avena* spp. The minimum number of *Pc* genes for resistance to crown rust was determined from infection-type data based on the interaction of several oat lines and 47 isolates of crown rust from the Iowa State University collection. Infection-type data from seedlings of seven diploid oat accessions, a hexaploid chromosome addition line (X117), and its recurrent parent (C649), were evaluated in reference to 33 standard differentials to determine low or high infection type. At least 17 unique crown rust resistance genes were detected in the hexaploid differentials with the Iowa State University crown rust collection, and four additional resistance genes were detected among the diploid accessions.

Among the diploid oat accessions, CI2630, CI3815 and CI6954 (all *A. strigosa* [*A. nuda*]) were resistant to most isolates of crown rust, while CI1994 (*A. wiestii*), CI9009 (*A. nuda*), CI3214 (*A. wiestii*), and CI4748 (*A. strigosa*) were susceptible to most isolates. An evaluation of parents from two diploid mapping populations revealed 37 differential reactions that can be detected and mapped in the progeny from a cross between CI2630 and CI9009, and 40 from a cross between CI3815 and CI1994. The two crosses have 32 differential reactions in common.

GREGORY and WISE (1994) studied linkage of genes conferring specific resistance to crown rust in diploid *Avena* species. The inheritance of resistance to an isolate of *Puccinia coronata* f. sp. *avenae*, race 276 was investigated in four diploid oat lines. F_3 families derived from *A. strigosa* (*A. nuda*) CI2630 × *A. nuda* CI9009, *A. wiestii* CI1994 x *A. strigosa* CI3815 and CI2630 were inoculated at the seedling stage and evaluated 9–12 days later. Segregation of the F_3 families indicated that CI2630 and CI3815 each possess one dominant gene that conditions resistance to race 276. The alleles conferring resistance occur at two different loci, separated by 17 cm on the same chromosome, and appear to be complementary. CI3815 may have an additional recessive resistance allele at a different locus on the same chromosome, 19 cm from its other resistance gene locus.

MUNDT and BROPHY (1988) studied the influence of number of host genotype units on the effectiveness of host mixtures for crown rust control and they demonstrated that the number of host genotype units in a population is a more important determinant of the effectiveness of mixtures for disease control than is host genotype unit area (the ground area occupied by a host genotype unit). Their results suggested that intraspecific or interspecific mixtures of large plants and the culture of alternating rows, swaths or fields of different host genotypes may provide greater disease control than previously anticipated.

BRIERE and KUSHALAPPA (1995) evaluated thirty-two breeding lines and oat cultivars for components of resistance to isolate CR 13 of *P. c.* f. sp. *avenae* under controlled environment conditions. A completely randomized design with four replicates was used and the experiment was repeated once. The number of pustules on each first leaf were counted daily after the first appeared. The length and width of five randomly selected pustules in each leaf were measured 14 days after inoculation. From the above data the mean latent period, number of pustules per leaf and pustule area were determined. Multivariate analysis was used to classify breeding lines and cultivars into groups with similar values of components of resistance. In both trials, the first three clusters of the dendrogram was composed of lines with relatively high levels of resistance, based on the components measured. Lines OA 712-17, OA 712-33 and cvs. Woodstock, Sylva and Ultima were consistently found to be in the first three clusters in both trials. The lines and cultivars (Fidler, Tibor and QO 576.27) that were grouped in the fourth cluster in the first trial were grouped in the first clusters in the second trial. analysis, Seven lines and cultivars were selected for further sporulation studies. Pustules were counted daily and spores harvested every third day. From this data, the mean latent period, the number of pustules per leaf, the number of spores per pustule and per leaf were determined. A correlation analysis showed high correlations (0.57) between the resistance components.

Stem rust

As with crown rust, the breeding of multigenic cultivars to stem rust is prospective (MCKENZIE 1964; MCKENZIE *et al.* 1971a, b; ŠEBESTA 1976, 1977, 1980a, b; ŠEBESTA & ZWATZ 1977, ŠEBESTA et al. 1996a, b; HARDER 1994; HARDER & HABER 1992). At present, ten major resistance genes for stem rust of oat (*Pg*-genes) are available to plant breeders (Table 2) (MARTENS 1985; HARDER 1994; ŠEBESTA 1975a, b; ŠEBESTA *et al.* 1998). The dominant (thermostable) *Pg-1* gene was extensively used in the USA for many years (STEWART & ROBERTS 1970) but not in Canada. The average effectiveness of the *Pg-1* in Europe in 1989–1996 was 34% (ŠEBESTA *et al.* 1998).

The dominant (thermostable) *Pg-2* gene has been incorporated into about 130 cultivars in North America, thus indicating the importance of this gene in the past (MARTENS 1985). The average effectiveness of *Pg-2* in Europe in 1989–1996 was about 47%

The dominant (thermolabile) *Pg-3* gene (MARTENS *et al.* 1967) is of limited importance in both North America and Europe (ŠEBESTA *et al.* 1991, 1998). In Europe in 1989–1996 the average effectiveness of *Pg-3* was about 30%. The *Pg-3* is either closely linked to a gene for crown rust resistance or itself confers resistance to both rusts (MCKENZIE *et al.* 1968). The virulence on this type of resistance was found to be inherited extrachromosomally (GREEN & MCKENZIE 1967).

Gene *Pg-4*, dominant and themolabile, has been used widely and together with *Pg-1* and *Pg-2* has in the past been the basis for stem rust resistance breeding, both in North America and Europe (MARTENS 1985; ŠEBESTA & ZWATZ 1980; ŠEBESTA *et al.* 1991, 1998). Its effectiveness in Europe in 1989–1996 was 73%.

The dominant genes *Pg-6* and *Pg-7*, conditioning resistance to a wide range of races, identified in the diploid species *A. strigosa* Schreb., C.D.3820, have not yet been transferred into hexaploid species. These genes may be the same (DYCK 1966).

The recessive and thermolabile gene *Pg-8* has not been used in breeding programmes in both North America and Europe.

Table 2. Genes of oat resistance to *Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn.

Resistance gene	Source of resistance	Remarks	References
Pg-1 ('D')	cv. White Russian, C.I.9318, R.L.899	dominant gene	GARBER (1921),, MARTENS *et al.* (1979), SIMONS *et al.* (1978)
Pg-2 ('A')	cv. Green Russian	dominant gene, allelic or closely linked with *Pg-1*	DIETZ (1928), MARTENS *et al.* (1979), SIMONS *et al.* (1978)
Pg-3 ('E')	cv. Joanette, C.I.9320, R.L.902	dominant gene, allelic or closely linked with *Pg-9*	WATERHOUSE (1930), MARTENS *et al.* (1979), SIMONS *et al.* (1978)
Pg-4 ('B')	cv. Hajira, C.I.6661, R.L.2123	dominant gene, allelic or closely linked with *Pg-13*	WELSH & JOHNSON (1954), SIMONS *et al.* (1978)
Pg-8 ('F')	cv. Hajira, C.I.9321, R.L.903	recessive genes, it may be allelic or closely linked with *Pg-1* and *Pg-2*	BROWING & FREY (1959), SIMONS *et al.* (1978)
Pg-9 ('H')	C.I.6792, C.I.9322, R.L.879	recessive genes, allelic or closely linked with *Pg-3*	MCKENZIE & GREEN (1965), SIMONS *et al.* (1978)
Pg-11	C.I.3034	incompletely recessive genes, conferring APR, independent of the *Pg-2*, *Pg-4* and *Pg-9*	MCKENZIE & MARTENS (1968), SIMONS *et al.* (1978)
Pg-12	cv. Kyto, C.I.8250	recessive genes, independent of the *Pg-2*, *Pg-4* and *Pg-9*	MARTENS *et al.* (1968), SIMONS *et al.* (1978)
Pg-13	*A. sterilis*, CAV2647, C.I.9212, R.L.618	recessive genes, allelic or tightly linked with *Pg-4*	MCKENZIE *et al.* (1970), SIMONS *et al.* (1978)
Pg-15	*A. sterilis*, CAV1830, C.I.9351, R.L.997	partially dominant gene	MARTENS *et al.* (1979)
Pg-16	*A. barbata* D203, C.I.9352, R.L.822	dominant gene	MARTENS *et al.* (1979)
Pg-17	*A. sterilis*, IB3056	dominant gene for APR	HARDER *et al.* (1989)
Pg-a	C.I.9139	3 recessive genes	MARTENS *et al.* (1979), ERPELDING & MCMULLEN (pers.comm.)

APR = adult plant resistance

However, it was effective against races from eastern Australian, eastern America and South America (COELHO 1976) and partially in Russia (SUZDALSKAYA *et al.* 1978). In Europe, in the 1989–96 period, *Pg-8* had an effectiveness of 35%.

The recessive and thermolabile gene *Pg-9* has not been used in breeding programmes until recently (MCKENZIE *et al.* 1976). Like the gene *Pg-3*, the *Pg-9* gene is closely associated with a gene for resistance to *P. coronata* (MCKENZIE & GREEN 1965; MCKENZIE *et al.* 1965). Tests of segregating populations of cv. Dumont indicated, in addition to *Pg-2* and *Pg-13*, the presence of the *Pg-9* gene tightly linked in coupling to a gene *PcX* for crown rust resistance (CHONG *et al.* 1994). The gene *Pg-9* was effective against the most common and virulent races of the Great Plain region in North America. In Europe, in 1989–96, the average effectiveness of *Pg-9* was 57%.

The incompletely recessive gene *Pg-11* has conferred adult plant resistance (APR) to all races of stem rust that have been tested (MCKENZIE & MARTENS 1968). There appears to be an association between this resistance and yellow plant colour, weak straw and reduced yield. A gene affecting chlorophyll levels may be tightly linked with *Pg-11*. Gene *Pg-11* may not be a rust resistance gene in the conventional sense but rather a progressively effective, sublethal, pigment deficiency gene that incidentally causes stem rust resistance.

The recessive gene *Pg-13* is one of the most effective stem rust resistance genes available to plant breeders (ROELFS *et al.* 1982; MARTENS 1981, 1985). Cvs. Fidler and Dumont developed by MCKENZIE *et al.* (1981, 1984) combine *Pg-13* with some other stem rust and crown rust resistance genes. The effectiveness of the *Pg-13* gene in Europe during 1989–96 period was 97%; with only one virulent pathotype identified in Estonia (ŠEBESTA *et al.* 1998).

Gene *Pg-14* is a partially dominant gene isolated by MACKEY and MATTSSON (1972) from Milford, C.I.5039, Winter Turf, C.I.1570 and some other lines. It is difficult to identify the origin for this gene.

Gene *Pg-15* is partially dominant and isolated from *A. sterilis* collected east of Uskudar on the Black Sea near Istanbul, Turkey (MARTENS *et al.* 1980; MARTENS 1985). Races avirulent on *Pg-9* were also avirulent on *Pg-15* in the Great Plains region of the North America (ROELFS *et al.* 1980; MARTENS 1981, 1985). This gene has not yet been used in commercial cultivars in either North America or Europe. The average effectiveness of this gene in Europe, in 1989–1996, was 72% (ŠEBESTA *et al.* 1998).

Gene *Pg-16* (MARTENS *et al.* 1979) is a highly effective gene from tetraploid *A. barbata* that may be successfully used in stem rust resistance breeding. Virulence to *Pg-16* was found in the USA in 1987, 1988, 1991, 1992 and 1993 by ROELFS *et al.* (1989, 1990, 1995). In Europe, in 1989–1996, the average effectiveness of *Pg-16* was 76%.

A dominant gene for stem rust resistance, designated *Pg-17*, was isolated by Harder et al. in 1990 from a Spanish *A. sterilis* accession IB3056, effective only at the adult plant stage.

The recessive gene *Pg-12* was isolated from Kyto, a cultivar introduced from the former Yugoslavia via Finland by the USDA in 1939 (MARTENS *et al.* 1968). The cv. Osmo expressed a rust reaction similar to that of Kyto (GREEN & MCKENZIE 1967). In Europe the effectiveness of *Pg-12* was 53%.

The *Pg-a* complex (MARTENS *et al.* 1981) was appears to consist of a gene *Pg-12* and interacting genes. ERPELDING and MCMULLEN (pers. comm.) identified 3 recessive genes in the *Pg-a* complex, and was found to be very effective in the USA, Canada and Europe (ROELFS *et al.* 1989, 1990, 1995). In Europe the *Pg-a* genes were effective of 87% (ŠEBESTA *et al.* 1998).

HARDER *et al.* (1995) studied an interesting stem and crown rust resistance in the Wisconsin oat selection Xl588-2 and found that it reacted either with mesothetic (moderately resistant) or 0; 1 (highly resistant) infection types to races of *P. g.* f. sp. *avenae*. This selection is also resistant to some pathotypes of *P. coronata* f. sp. *avenae*. Tests conducted at Winnipeg with 45 races of stem rust showed that X1588-2 was highly resistant to races that were avirulent to *Pg3* and/or *Pg4* and was moderately resistant to all other races. These results and the pedigree of X1588-2 indicated the presence of stem rust resistance genes *Pg3, Pg4* and *Pgl0* in X1588-2. The presence of these genes was confirmed in tests of seedlings of segregating Bcl, F_1 and F_2 populations from a cross of X1588-2 with the susceptible cv. Makuru.

Resistance to crown rust in X1588-2 was conferred by a gene designated as *Pc95*. The resistance conferred by *Pc95*, however, was not effective against a sufficient range of pathotypes of *P. coronata* to be useful source of resistance. Genes *Pg3* and *Pg4* are ineffective against current Norther American populations of stem rust. Gene *Pg10*, however, appears to confer a moderate but very board range of resistance and may be a useful source of resistance in oat breeding.

Powdery mildew

Like in rust control, the use of host resistance offers the most economic and environmentally benign method of the disease control (RODERICK et al. 1995; ŠEBESTA *et al.* 1997; JONES *et al.* 1987; JONES & RODERICK 1986a, b; NAQUI 1990). Owing to high variability of the pathogen and deficiency of major resistance genes the combination of race specific and adult plant resistance (APR) is very desirable (Table 3).

Until recently, four major resistance genes were described (SIMONS *et al.* 1978). Resistance genes *Eg-1* and *Eg-2* were found in Cc4146 and *A. strigosa,* respectively by JONES and GRIFFITHS (1952). Gene *Eg-3* was tranferred into the cv. Mostyn (Cc4347) by HAYES and JONES (1966) and gene *Eg-4* was transferred by THOMAS *et al.* (1975) from *A. barbata,* Cc4897. More recently, ŠEBESTA *et al.* (1987a, b) described monogenic mildew resistance in *A. sterilis,* CAV 2648, effective against groups OMV 1 and OMV 2.

The adult plant resistance (APR) of a number of oat genotypes has been shown to be inherited quantitatively. These include the cvs. Maldwyn, Maelor and Roxton (JONES 1974, 1977, 1978). Transgressive segregation for increased levels of APR has been demonstrated (JONES 1983; JONES & RODERICK 1986a, b). The lines APR 122 and APR 166 (HOPE 1991; HOPE & KUMMER 1991), derivatives of *A. eriantha,* CAV 0128, appear to be highly effective on the European Continent and also on the British Isles (ŠEBESTA 1995, 1996, 1997, 1998).

In addition to crown rust resistance gene *Pc 54* and stem rust resistance gene *Pg 15* the lines Cc7422 (UK) and *Pc 54-2* (CZ), reselected from the line Pc 54, carry mildew resistance conditioned by a single incompletely dominant gene with additional factors modifying adult plant resistance. There was no evidence of linkage between the mildew and crown rust resistance genes. The expression of both stem rust and mildew resistance was modifyied by, or linked to plant height (ŠEBESTA *et al.* 1993).

Mildew development of APR genotypes is characterized by a comparatively long latent period, low infection frequency and a low sporulation capacity (JONES 1978). The APR of the cv. Maldwyn has remained effective since the 1940's. JONES (1986)

Table 3. Sources of resistance to powdery mildew (*Erysiphe graminis* f.sp. *avenae*) in *Avena* species (Sebesta et al. 1997)

Accession/Cv	Species	OMR group**	Remarks	References
CAV 6773, CAV 6794	*A. atlantica* (2*x* = 14)			SEBESTA (1990a, b), HERMMANN & RODERICK (1996)
Cc 3678	*A. hirtula* (2*x*)		gene *Eg-2*	JONES & GRIFFITHS (1952), SIMONS *et al.* (1978)
CAV 5264	*A. macrostachya* (2*x*)			HOPPE & POHLER (1991)
Qu 8	*A. longiglumis* (2*x*)			HERMMANN & RODERICK (1996)
Cc 4852	*A. ventricosa* (2*x*)			JONES *et al.* (1982)
Cc 6557	*A. prostrata* (2*x*)			JONES *et al.* (1982)
AVE 128, 264, 488	*A. strigosa* (2*x*)			HERMMANN & RODERICK (1996)
S.171	*A. strigosa* × *A. brevis* (2*x*)			JONES & RODERICK (1986a, b)
Cc 4093	*A. strigosa* s.sp *glabrota* (2*x*)			JONES *et al.* (1982)
Cc 6558	*A. murphyi* (4*x* = 28)			JONES *et al.* (1982)
Cc 4146	*A. sativa/A. ludoviciana* (6*x*)	OMR 2	gene *Eg-1*	JONES & GRIFFITHS (1952), SIMONS *et al.* (1978)
Cc 4761	*A. byzantina* (6*x*)		selection from Creme	JONES (1975)
APR 122/166	*A. sativa* (6*x*)		derived from *A.eriantha*	HOPPE (1991), HOPPE & KUMMER (1991)
			CAV 0128	THOMAS *et al.* (1975, 1980)
AV 1860, Cc 6490	*A. sativa*	OMR 4	translocation lines, resistance from *A. barbata* Cc 4897; gene *Eg-4*	SIMONS *et al.* (1978)
Pc 54	*A. sativa*		an incomplete dominant gene	ŠEBESTA *et al.* (1993)
Pc 39	*A. sativa*			HOPPE (1984)
07718Cn	*A. sativa*	OMR 2	resistance from Cc4146	JONES (1982)
CAV 2648	*A. sterilis*	OMR 1+2		ŠEBESTA *et al.* (1987)
Cc 4346, Cc 4347	*A. sterilis* var. *ludoviciana* (6*x*)	OMR 3	incorporated into 9065Cn; gene *Eg-3*	LAWES & HAYES (1965), HAYES & JONES (1966), SIMONS *et al.* (1978)
CAV 2107	*A. byzantina* (6*x*)			ŠEBESTA (1990a, b)
CAV 3891, CAV 3889	*A. occidentalis* (6*x*)			ŠEBESTA *et al.* (1987a, b) HERMMANN & RODERICK (1996)
Mostyn	*A. sativa*	OMR 3	APR*	JONES (1983), LAWES & HAYES (1965);
Manod	*A. sativa*	OMR 1		GRIFFITHS (1962), THOMAS *et al.* (1975)
Maelor	*A. sativa*	OMR 0	APR	GRIFFITHS (1962), ALI (1985), RODERICK & CLIFFORD (1995)
Roxton	*A. sativa*	OMR 0	APR	JONES (1974), JONES & RODERICK (1986a, b), RODERICK & CLIFFORD (1995)
Maldwyn	*A. sativa*	OMR 0	APR	HAYES & JONES (1966), JONES (1978,1983)
Dal	*A. sativa*			HITE *et al.* (1977)
Solva	*A. sativa* (winter)			JONES (1994)
Av 2557	*A. sativa* substitution line (6*x* = 42)		derived from *A. prostrata* addition line	THOMAS & GRIFFITHS (1985), NAQUI (1990)
93-2-4	*A. sativa*		high level of APR	JONES & RODERICK (1986a, b)
Bage sel Klein	*A. byzantina* (6x)		high level of APR	JONES & RODERICK (1986a, b)
Rouge d'Algerie	*A. byzantina*		high level of APR	ALI (1985)
OM 1711	*A. sativa*	OMR 3	bred for high APR	RODERICK & JONES (1991), RODERICK & CLIFFORD (1995)
OM 1621, OM 1387	*A. sativa*	OMR 3	bred for high APR	JONES (1983), RODERICK & JONES (1991)

*Adult plant resistant type; **JONES & JONES (1979), RODERICK *et al.* (1995).

demonstrated that this resistance was governed by up to seven additive genetic factors and also showed that such factors could be accumulated even from now susceptible cultivars such as the cv. Mostyn, to produce transgressive lines (JONES 1983). Several other sources of APR have been identified and characterized (JONES 1978; JONES & RODERICK 1986a, b; RODERICK & CLIFFORD 1995; ŠEBESTA *et al.* 1987a, b).

In a genetic study of the components of APR in a range of cultivars, ALI (1985) found that with race 2 (OMV 1) latent period was under additive control, but could not form a clear

conclusion from the results with race 5 (OMV 1 + 2 + 3). Infection frequency was controlled by both additive and dominant factors for both races.

The major gene sources of mildew resistance exploited so far have not been durable and in most cases have shortened the commercial life of the cultivar. JONES (1982, 1983) found evidence for a residual effect of a 'defeated' major gene, which suggests a possible association between major gene resistance and APR. An alternative breeding objective could be to combine effective major gene resistance with APR so that if the major gene resistance became ineffective the APR would give added protection.

ŠEBESTA *et al.* (1993) recommended that this could be carried out with the *Pc54* resistance since it appears to be mainly under single gene control. A method by which major gene and APR could be combined was outlined by JONES (1982). This method relies on identifying segregating family lines and repeatedly quantifying the amount of mildew on the homozygous susceptible plants within these families from the F_3 generation onwards.

The smuts

There are two species of smut on oat: loose smut (*Ustilago avenae* [Pers.] Rostr.) and covered smut (*Ustilago kolleri* Wille, *U. avenae* [Pers.] Rostr. var. *levis* Kellerm. & Sw.).

Smut resistance has been found in cultivated oats (DRACEA *et al.* 1978; MYAGKOVA *et al.* 1982; NIELSEN 1977a, b) and also in wild oat accessions (NIELSEN 1978a, b; WILLIAMS & VERMA 1956). NIELSEN (1977a, b) found 447 of 5 485 entries of the USDA oat collection immune or highly resistant to a composite smut population. The diversity of resistance is not yet clear because it has not been possible to screen the resistance on a gene-for-gene basis (HARDER & HABER 1992). Anyway, more diverse sources of resistance have been found, and the success of breeding for resistance to changing virulence in smut populations has been demonstrated (MCKENZIE *et al.* 1981, 1984 – cit. HARDER & HABER 1992).

Septoria leaf blight and black stem

The fungus *Septoria avenae* Frank, was described for the first time in Germany by FRANK (1895). Later the disease was isolated in Denmark, France, Norway and Poland. In the United States of America the pathogen was reported in 1922 by Weber who named its perfect state *Leptosphaeria avenaria* Weber (WEBER 1922). JOHNSON (1947) found that the population of the fungus infecting oat did not infect the other cereals. Therefore, the fungus on oats was designated as *L. avenaria* f. sp. *avenaria* and its conidial stage as *S. avenae* f. sp. *avenae* (SHAW 1957a, b – cit. HARDER & HABER 1992). Recently, the accepted name for this pathogen was *Stagonospora avenae* f. sp. *avenae* (syn *Septoria avenae*, teleomorph *Phaeosphaeria* [= *Leptospharia] avenaria*) (CUNFER 1994).

S. a. f. sp. *avenae (Phaeosphaeria avenaria* f. sp. *avenaria)* is a very harmful fungus on oat. *S. avenae* can attack any aboveground part of the oat plant. Stem infection results in severe lodging. Losses of between 34–43% were reported from Germany (MIELKE 1975; MULLER 1964), but its occurrence was also announced from other parts of Europe (NOBLE & MONTGOMERIE 1956; ŠEBESTA 1985 – cit. HARDER & HABER 1992).

CLARK and ZILLINSKY (1960 a, b) found resistance in accessions of *A. strigosa.* Furthermore, differences between oat lines were found in terms of leaf area affected (DERICK 1954; HUFFMAN 1955a, b), size of lesions and stem or seed infection (HOOKER, 1955, 1957a, b; LUND & SHANDS 1956 – cit. HARDER & HABER 1992) or when measured as seed yield reduction (CLARK & JOHNSTON 1973). Stem infections showed clear differences in resistance expression (CLARK 1980; HOOKER 1957a, b; LUND & SHANDS 1956).

Variation in the disease resistance index (DRI) (ŠEBESTA *et al.* 1995) of *S. avenae* on the 52 oat genotypes was observed in EODN trials in Austria, Germany, Italy and Poland between 1990–1993. A high index value is assumed to be associated with a high level of quantitative resistance. The accession *A. sterilis* CAV 2648 had the highest resistance index followed by Cc4761, Pc 55, Pc 67, Pc 50-2, Pc 60, Pc 50-4, Pc 54, IL 86-6404, Garland, Pc 58, Pc 48 and Cc6490.

However, some differences in aggressiveness of *S. avenae* populations presumably occur, e.g., *A. sterilis*, CAV 2628 with the highest frequency of resistant evaluations was susceptible in Poland, at the locality Wielopole in 1992. On the other hand, Rodney E, Roxton, Pc 59, OA 504-6, OA 504-5 and APR 166 were classified, ten, eight, six, five and four times, respectively, as moderately susceptible or susceptible, but are supposed to be susceptible (ZWATZ *et al.* 1994.). Recently, CORAZZA *et al.* (1990, 1992) found that some of the oat cultivars grown in Italy were moderately resistant to *S. avenae* (Argentina, Lidia, Manoire, Weibull), moderately susceptible (Angelica, Astra, Ava, Condor, Kalott, Nave, Ombrone, Perona, Vintero) or susceptible (Rogar 8, Sole II, Sonar).

No studies of genetics of resistance to *S. avenae* have been carried out so far.

Helminthosporium leaf and panicle blotch

The fungus *Helminthosporium avenae* Eidam *(Pyrenophora avenae* Ito & Kurib.) is a common and destructive pathogen of oat in humid and cold regions of Europe and North America (HARDER & HABER 1992; ŠEBESTA *et al.* 1995). It has also been reported from Asia and South Africa (WELSH *et al.* 1953). Before the application of mercury organic dressing the disease caused damage in the United Kingdom (DILLON *et al.* 1943). According to MULLER (1963), *P. avenae* was the most frequent pathogen inciting the characteristic spot symptom in the former German Democratic Republic. Later on, KIEWNICK (1974) reported that *Pyrenophora* leaf blotch was the most serious disease in Germany after loose smut and crown rust. The leaf blotch causal agent was noted to be common fungus in Sweden (OLOFSSON 1976) and Finland (REKOLA *et al.* 1970).

The varietal reaction of oat to *Pyrenophora* was extensively studied by EARHART and SHANDS (1952). The oat lines B 1-7-67 and Wisconsin hybrid X 279-1 expressed resistance which is probably applicable in resistance breeding to this disease (EARHART & SHANDS 1952).

MULLER (1963) showed that the oat line Bernburg St. 37664 was heavily attacked whereas cvs. Omiko and Flamingsweisse II were attacked by less than half of the former. American cultivars were much less attacked than the German ones. The cvs. Clinton and Bonham and the oat line 2411 were recommended for disease resistance breeding. Furthermore, some lines resistant to *H. avenae* were identified by PANDEY and MISRA (1973). GRACHEV (1962) and KUNOWSKI and BRESHKOV (1981) found resistance to *H. avenae* in Iowa 2052, Aigorudo, selections from Garry and in the accessions of *A. byzantina, A. strigosa* and *A. brevis.*

Recently, rate-limiting resistance on several oat breeding lines was reported by FRANK and CHRIST (1988 – cit. HARDER & HABER 1992). In a study by ŠEBESTA *et al.* (1994) the quantitative response of oats to *P. avenae* both on leaves and panicles

differed considerably between genotypes. The severity of infection varied with the twenty genotypes scoring between 0.10 to 1.00 included in the resistant group, eight genotypes (1.67 to 4.33) were classified as moderately resistant, nine genotypes (7.42–20.00) were moderately susceptible and one genotype, the cv. Marloo (30.00) was susceptible. Even within these categories there are significant differences between genotypes (ŠEBESTA *et al.* 1994). The level of panicle infection showed that 26 genotypes were resistant, six moderately resistant, four moderately susceptible and two susceptible (ŠEBESTA *et al.* 1994). The following cultivars and lines were resistant in both leaves and panicles; KR 89-18, KR 8122, KR 9046, Explorer, Zlatak, Adam, David, Ardo, Fuchs, KR 9478, Trafalgar, Pan, IL 86-1158, Hirondel, IL 86-6467, Arne and Nero. Moderately resistant in leaves and resistant in panicles were the cvs. Lars, IL 86-6404, Semu 3767, Flamingsnova and the line IL 86-4467. Moderately resistant both in leaves and panicles were cvs. Auron, Wiesel, Tomba, IL 86-4189 and the cv. Calibre. Moderately susceptible in leaves and resistant in panicles were the cvs. P 5137, IL 85-2069, IL 86-5698 and the cv. Saia. Moderately susceptible in leaves and moderately resistant in panicles was the cv. Ogle. Moderately susceptible in leaves and moderately susceptible in panicles were the cvs. Akiwase, Dolphin, Joycee and Walaroo. The cv. Marloo was susceptible both in leaves and panicles.

The high incidence of *P. avenae* in Central Bohemia in 1992 made it possible to distinguish between the quantitative responses of oat genotypes to this pathogen. However, the data should be considered as preliminary since it only relates to a single season of observations (ŠEBESTA *et al.* 1994). Pathogenic specialization of the fungus was reported by PANDEY and MISRA (1973) and by TVEIT (1956 – cit. HARDER & HABER 1992).

In the EODN trials in 1990–1993 high or moderate–high incidence of *P. avenae* was recorded in Austria, Finland, Italy, Poland and Russia. Moderate or a low-moderate incidence was recorded in the Czech Republic, Germany, Poland, Russia and Sweden, and a low incidence in Finland, Italy and Poland (ŠEBESTA *et al.* 1995). A high resistance index was found in the majority of Illinois lines and a number of other oats. The quality of resistance is given by the sum of resistant and moderately resistant scores within this period. There is probably pathogenic specialization of the fungus and differences in aggressiveness exist among its populations. Oat lines such as IL 86-1158, IL 86-6467, IL 86-4189, IL 85-2069, IL 86-6404 and IL 86-5698 and other ones included in the EODN can be assumed to possess some resistance to *P. avenae* (ŠEBESTA *et al.* 1995).

Up to now, our knowledge of the reaction of the oats to *P. avenae*, especially on panicles, has been poor. The reaction of panicles can differ significantly from that on leaves so that the reaction of leaves and panicles should be taken into consideration when selecting parental cultivars for resistance breeding programme.

It is promising that a number of the Czech cultivars and advanced lines, such as KR 89-18, KR 8122, KR 9046, Zlatak, Adam, David, Ardo and KR 9478 were resistant both on leaves and panicles. This might be due to selection since the pathogen is common in the country and causes significant yield loss. So that selection for the high yield has also presumably been the selection for a higher resistance to *P. avenae*.

Conclusions

Oat is subjected to a number of diseases, and the use of resistant cultivars is the most feasible and economic means of controlling these diseases.

Race specific and race non-specific resistance, and tolerance of oat genotypes to diseases have been characterized to some of these diseases.

The specific resistance of oat to fungal diseases is mostly conditioned by single dominant and independently inherited genes, along with some genes in interaction. Non-specific resistance (slow diseasing) is a heritable trait conditioned by recessive genes with additive effects.

The use of molecular markers associated with desirable phenotypic traits has great potential for higher efficiency in plant breeding. Markers based on DNA or endosperm protein polymorphisms have been used to identify crown rust and stem rust resistance genes. Studies have also been undertaken to determine the map distance between different crown rust and stem rust resistance genes.

The genetic control of individual fungal diseases is discussed. The breeding of multigenic cultivars in relation to crown rust and stem rust resistance has been proposed and is practised in Canada and Europe.

Owing to the deficiency of major resistance genes to powdery mildew, a combination of major gene resistance and adult plant resistance (APR) (under additive control) is recommended.

Diverse sources of resistance to smuts have been found and the success of breeding for resistance has been demonstrated.

Differences in oat resistance to Septoria leaf blight and black stem, and Helminthosporium leaf and panicle blotch have been found. However, genetics studies of resistance to these diseases have not been carried out so far.

References

ALI A. (1985): Expression of inheritance of adult plant resistance in oats to *Erysiphe graminis avenae*. [Unpublished Msc Thesis.] University College of Wales, Aberystwyth, UK.

BAKER E. P., UPADHYAYA Y. M. (1967): Studies on the inheritance of rust resistance in oats. IV. Expression of allelomorphism and gene interaction for crown rust resistance in crosses involving the oat cultivar Bond. In: Proc. Linn. Soc. NSW, **91**: 155–175.

BRAKE V. M., IRWIN J. A.-G. (1992): Partial resistance of oats to *P. coronata* f. sp. *avenae*. Austr. J. Agr. Res., **43**: 1217–227.

BRIERE S. C., KUSHALAPPA A. C. (1995): Evaluation of components of resistance in oat breeding lines and cultivars to crown rust (*Puccinia coronata* f. sp. *avenae*) under controlled environmental conditions. Can. J. Plant Pathol., **17**: 319–324.

BRIERE S. C. KUSHALAPPA A. C., MATHER D. E. (1994): Screening for partial resistance to an isolate of crown rust (*Puccinia coronata* f. sp. *avenae*) race 264 in oat cultivars and breeding lines. Can. J. Plant Pathol., **16**: 49–55.

BROWDER L. E. (1985): Parasite: Host environment specificity in the cereal rusts. Annu. Rev. Phytopathol., **23**: 201–222.

BROWNING J. A., FREY K. J. (1959): The inheritance of new sources of oat stem rust resistance. Plant Dis. Rep., **43**: 768–771.

BUSH A. L., WISE R. P. (1996): Crown rust resistance loci on linkage groups 4 and 13 in cultivated oat. J. Heredity, **87**: 427–432.

BUSH A. L., WISE R. P., RAYAPATI P. J., LEE M. (1994): Restriction fragment length polymorphosms linked to genes for resistance to crown rust (*Puccinia coronata*) in near isogenic lines of hexaploid oat (*Avena sativa*). Genome, **37**: 823–831.

CHONG J., BROWN P. D. (1996): Genetics of resistance to *Puccinia coronata* f. sp. *avenae* in two *Avena sativa* accessions. Can. J. Plant Pathol., **18**: 286–292.

CHONG J., HOWES N. K., BROWN P. D., HARDER D. E. (1994): Identification of the stem rust resistance gene *Pg9* and its association with crown rust resistance and endosperm protein in 'Dumont'oat. Genome, **37**: 440–447.

CLARK R. V. (1980): Evaluation of oats for resistance to *Septoria* leaf blotch and black stem. Can. J. Plant Pathol., **2**: 213–216. In: CLARK R. V., JOHNSTON H. W. (1973): Tolerance of oats to the *Septoria* diseases.Can. J. Plant Sci., **53**: 471–475.

CLARK R. V., JOHNSTON H. W. (1973): Tolerance of oats to the *Septoria* diseases. Can. J. Plant Sci., **53**: 471–475.

CLARK R. V., ZILLINSKY F. J. (1960a): Epidemiology studies on the *Septoria* disease of oats. Can. J. Bot., **38**: 93–102.

CLARK R. V., ZILLINSKY F. J. (1960b): Varietal reaction of oats to the *Septoria* disease. Can. J. Plant Sci., **53**: 471–475.

COELHO E. T. (1976): Distribution, prevalence and a new race of *Puccinia graminis avenae* in Rio Grande do Sul. Pesquis. Agropecu. Bras., **11**: 19–22.

CORAZZA L., BALMAS V., ARDUINI F., PALUMBO M. (1990): Comportamento varietale dell'avena nei confronti delle principali malattie fungine. L'Inform. Agrario, **46**: 39–41.

CORAZZA L., BALMAS V., PEZZALI M., PALUMBO M., TRABUCCO A. (1992): Le principali malattie fungine dell'avena rilevate nel 1992. L'Inform. Agrario, **48**: 55–57.

CUNFER B. M. (1994):. Biology and taxonomy of *Stagonospora nodorum* on barley. In: Proc. 4th Int. Workshop Septoria of Cereals. IHAR, Radzikow, Poland: 35–38.

DERICK Y. R. A. (1954): A survey of varietal resistance to black stem (*Septoria avenae*) in oats in eastern Can. Plant Dis. Rep., **38**: 85–87.

DIETZ S. M. (1928): Inheritance of resistance in oats to *Puccinia graminis avenae*. J. Agr. Res., **37**: 1–23.

DRACEA A. E., GOIAN M., POPESCU G. (1978): Response of some oat varieties to attack by *Ustilago avenae* (Pers.) Jens. Lucr. Stiint. Inst. Agron. Timisoara Ser. Agron., **15**: 165–168.

DYCK P. L. (1966): Inheritance of stem rust resistance and other characteristics in diploid oats, *Avena strigosa*. Can. J. Genet. Cytol., **8**: 444–450.

EARHART R. W., SHANDS H. L. (1952): Oat varietal responses to infection by *Helminthosporium avenae* Eid. Agron. J., **44**: 234–238.

FINKNER V. C. (1954): Genetic factors governing resistance and susceptibility of oats to *Puccinia coronata* Corda var. *avenae*, F. and L. race 57. Iowa Agric. Exp. Sta. Bull., **411**: 1040–1063.

FLEISCHMANN G, MCKENZIE R. I. H. (1968): Inhderitance of crown rust resistance in *Avena sterilis*. Crop Sci., **8**: 710–13.

FLEISCHMANN G, MCKENZIE R. I. H. SHIPTON W. A. (1971a): Inheritance of crown rust resistance in *Avena sterilis* L. from Israerl. Crop Sci., **11**: 451–453.

FLEISCHMANN G, MCKENZIE R. I. H. SHIPTON W. A. (1971b): Inheritance of crown rust resistance genes in *Avena sterilis* collections from Israel, Portugal, and Tunisia. Can. J. Genet. Cytol., **13**: 251–255.

FORMANOVÁ M., ŠEBESTA J. (1994):. Assessment of adult plant resistance of winter wheat to powdery mildew. Ochr. Rostl., **30**: 189–198.

FOX S. L., BROWN P. D., CHONG J. (1997): Inheritance of crown rust resistance in four accessions of *Avena sterilis* L. Crop Sci., **37**: 342–345.

FRANK B. (1895): Die neuen deutschen Getreidepilze. Ber. Dtsch. Bot. Ges., **13**: 61–65.

FRANK J. A., CHRIST B. J. (1988): Rate limiting resistance to *Pyrenophora* leaf blotch in spring oats. Phytopathology, **78**: 957–960.

FREY K. J. (1994): Remaking a crop gene pool: the case history of *Avena*. In: Proc. Nat. Sci. Council, Rep. China. Part B. Life Sci., **18**: 85–93.

FREY K. J., BROWNING J. A. (1971): Association between genetic factors for crown rust resistance and yield in oats. Crop Sci., **11**: 757–760.

FREY K. J., BROWNING J. A., SIMONS M. D., DAY P. R. (1977): Management systems for host genes to control disease loss. Ann. N. Y. Acad. Sci., **287**: 255–274.

GARBER R. J. (1921): A preliminary note on the inheritance of rust resistance in oats. J. Amer. Soc. Agron., **13**: 41–43.

GAUNT R. E. (1981): Disease tolerance – an indicator of thresholds? Phytopathology, **71**: 915–916.

GRACHEV A. F. (1962): The resistance of oats to crown rust and helminthosporiosis under the conditions of Primorski Krai. Ref. Zhur. Biol. No. 141G236 (Abstract).

GREEN G. J., MCKENZIE R. I. H. (1967): Mendelian and extrachromosomal inheritance of virulence in *Puccinia graminis* f. sp. *avenae*. Can. J. Genet. Cytol., **9**: 785–793.

GREGORY J. W., WISE R. P. (1994): Linkage of genes conferring specific resistance to oat crown rust in diploid *Avena*. Genome, **37**: 92–96.

GRIFFITHS D. J. (1962): The Aberystwyth Varieties of Oats. Welsh Plant Breed. Sta., Aberystwyth.

HARDER D. E. (1994): Virulence dynamics of *Puccinia graminis* f. sp. *avenae* in Canada, 1921–1993. Phytopathology, **84**: 739–746.

HARDER D. E., HABER S. (1992): Oat diseases and pathologic techniques. Oat Sci. Technol. – Agronomy Monogr. No. 33: 307–425.

HARDER D. E., MARTENS J. W., MCKENZIE R. I. H. (1971): Changes in chlorophyll and carotinoid content in oats associated with the expression of adult plant resistance to stem rust conferred by gene *Pg11*. Can. J. Bot., **49**: 1783–1785.

HARDER D. E., MCKENZIE R. I. H., MARTENS J. W. (1984): Inheritance of adult plant resistance to crown rust in an accession of *Avena sterilis*. Phytopathology, **73**: 352–353.

HARDER D. E., CHONG J., BROWN P. D., MARTENS J. W. (1989): Inheritance of resistance to *Puccinia coronata avenae* and *P. graminis avenae* in an accession of *Avena sterilis* from Spain. Genome, **33**: 198–202.

HARDER D. E., CHONG J., BROWN P. D. (1995): Stem and crown rust resistance in the Wisconsin oat selection X1588-2. Crop Sci., **35**: 1011–1015.

HAYES H. K., MOORE M. B., STAKMAN E. C. (1939): Study of inheritance in crosses between Bond, *Avena byzantina*, and varieties of *A. sativa*. Minnesota Agric. Exp. Sta., Techn. Bull. 137.

HAYES J. D., JONES I. T. (1966): Variation in the pathogenicity of *Erysiphe graminis* D.C. f. sp. *avenae*, and its relation to the development of mildew-resistant oat cultivars. Euphytica, **15**: 80–86.

HERRMANN M., RODERICK H. W. (1996): Characterisation of new oat germplasm for resistance to powdery mildew. Euphytica, **89**: 405–410.

HITE R. E., SHERWOOD R. T., MARSHALL H. G. (1977): Adult plant resistance to powdery mildew in 'Dal'oats. Plant Dis. Rep., **61**: 273–277.

HOOKER A. L. (1955): *Septoria* reactions of entries in the North Central states uniform oat yield nursery and other strains in 1955. Plant Dis. Rep., **39**: 963–966.

HOOKER A. L. (1957a): Varietal reaction correlation studies in the *Septoria* disease of oats. Agron. J., **40**: 600–609.

HOOKER A. L. (1957b): *Septoria* reactions of oat varieties and selections in 1956. Plant Dis. Rep., **41**: 385–388.

HOPPE H.-D. (1984): Untersuchungen zur Feldresistenz von Hafer (*Avena sativa* L.) gegen echten Mehltau (*Erysiphe graminis* D. C. f. sp. *avenae* March.). Arch. Zucht.-Fors., **14**: 353–358.

HOPPE H.-D. (1991): Moglichkeiten zur Introgression von Wildartinformation aus Avena-Arten niederer Ploidiestufen und deren zuchterische Nutzung in hexapliden Kulturhafer. [Dissertation.] Halle-Wittenberg, Landwirt. Fak. Martin-Luther-Univ.

HOPPE H.-D., KUMMER M. (1991): New hexaploid derivatives after introgression from *A. pilosa*.Vortr. Pfl.-Zucht., **20**: 56–61.

HOPPE H.-D., POHLER W. (1991): Successful hybridisation between *Avena prostrata* and *A. macrostachya*. Cereal Res. Commun., **16**: 231–235.

HOWES N. K., CHONG J., BROWN P. D. (1992): Oat endosperm proteins associated with resistance to stem rust of oats. Genome, **35**: 120–125.

HUFFMAN M. D. (1955a): Testing for resistance to the *Septoria* disease of oats. Plant Dis. Rep., **39**: 25–28.

HUFFMAN M. D. (1955b): Disease cycle of *Septoria* disease of oats. Phytopathology, **45**: 278–280.

HUMPHREYS D. G., MATHER D. E. (1996): Heritability of beta-glucan, groat percentage, and crown rust resistance in two oat crosses. Euphytica, **91**: 359–364.

JOHNSON T. (1947): A form of *Leptosphaeria avenaria* on wheat in Canada. Can. J. Res. (C), **25**: 259–270.

JONES D. G., CLIFFORD B. C. (1978): Cereal diseases, their pathology and control. BASF United Kingdom Ltd.

JONES E. (1994): Spring Oats. AFRC Institute of Grassland and Environmental Research, Aberystwyth: 23.

JONES E. T., GRIFFITHS D. J. (1952): Varietal resistance and susceptibility of oats to powdery mildew (*Erysiphe graminis* f. sp. *avenae*) on spring oats. Trans. Brit. Mycol. Soc., **35**: 71–80.

JONES I. T. (1974): The expression of adult plant resistance to oat mildew (*Erysiphe graminis* f. sp. *avenae*). A. Rep. 1973, Welsh Plant Breed. Sta., Aberystwyth: 64–66.

JONES I. T (1975): The preconditioning effect of day-length and ligh intensity on adult plant resistance to powdery mildew in oats. Ann. Appl. Biol., **80**: 301–309.

JONES I. T (1977): The effect on grain yield of adult plant resistance to mildew in oats. Ann. Appl. Biol., **86**: 267–277.

JONES I. T (1978): Components of adult plant resistance to powdery mildew (*Erysiphe graminis* f. sp. *avenae*) in oats. Ann. Appl. Biol., **90**: 233–239.

JONES I. T (1982): A study of the host-pathogen relationship and inheritance of resistance to *Erysiphe graminis avenae*. [Unpublished PhD Thesis.] Univ. Wales, UK.

JONES I. T (1983): Transgressive segregation for enhanced level of adult plant resistance to mildew in the oat cross Mostyn × Maldwyn. Euphytica, **32**: 499–503.

JONES I. T (1986): Inheritance of adult plant resistance to mildew in oats. Ann. Appl. Biol., **109**: 187–192.

JONES I. T., JONES E. R. L. (1979): Mildew of oats. A. Rep. 1978, UK Cereal Pathogen Virulence Survey: 59–63.

JONES I. T., RODERICK H. W. (1986a): Sources of resistance in oats to mildew. A. Rep. 1985, Welsh Plant Breed. Sta., Aberystwyth: 113–114.

JONES I. T., RODERICK H. W. (1986b): Transgressive segregation for increased levels of adult plant resistance to mildew in oats. In: Proc. 2nd Int. Oats Conf., Univ. Coll. Wales, WPBS, Aberystwyth, UK, July 15–18, 1985: 83–87.

JONES I. T., O'REILLY A., DAVIES I. J. E. R. (1982): Oat mildew – *Avena barbata* resistance. A. Rep. 1981, Welsh Plant Breed. Sta., Aberystwyth: 89–91.

JONES I. T., RODERICK H. W., CLIFFORD B. C. (1987): The integration of host resistance with fungicides in the control of oat powdery mildew. Ann. Appl. Biol., **110**: 591–602.

KIEHN F. A., MCKENZIE R. I. H., HARDER D. E. (1976): Inheritance of resistance to *Puccinia coronata avenae* and its association with seed characteristics in four accessions of *Avena sterilis*. Can. J. Genet. Cytol., **12**: 230–236.

KOCHMAN J. K., BROWN J. F. (1975): Development of the stem rust and crown rust on leraves, sheaths, and peduncles of oats. Phytopathology, **65**: 1404–1408.

KUNOWSKI Z., BRESHKOV T. (1981): Field resistance of oat varieties from the world collection to *Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn. and *Pyrenophora avenae* Ito. Rasten. Nauki, **18**: 118–123.

LAWES D. A., HAYES J. D. (1965): The effect of mildew (*Erysiphe graminis* f. sp. *avenae*) on spring oats. Plant Pathol., **14**: 125–128.

LIN-KUOCHIH, BUSHNELL W. R., SZABO L. J., SMITH A. G., LIN K. C. (1996): Isolation and expression of a host response gene family encoding thaumatin-like proteins in incompatible oat–stem rust fungus interactions. Mol. Plant Microbe Interaction, **9**: 511–522.

LITZENBERGER S. C. (1949): Inheritance of resistance to specific races of crown and stem rust, to Helminthosporium blight, and of certain agronomic characters of oats. Iowa Agric. Exp. Sta., Bull. 370: 453–496.

LUKE H. H., BERGER R. D. (1982): Slow rusting in oats compared with the logistic and Gompertz models. Phytopathology, **72**: 400–402.

LUKE H. H., CHAPMAN W. H., BARNETT R. D. (1972): Horizontal resistance of Red Rustproof oats to crown rust. Phytopathology, **62**: 414–417.

LUKE H. H., BARNETT R. D., PFAHLER P. L. (1975): Inheritance of horizontal resistance to crown rust in oats. Phytopathology, **65**:631–632.

LUKE H. H., PFAHLER P. L., BARNETT R. D. (1981): Influence of disease severity and environmental conditions on low receptivity of oats to crown rust. Plant Dis., **65**: 125–127.

LUND S., SHANDS H. L. (1956): The response of oat strains to *Septoria avenae*. Phytopathology **46**: 48–50.

MAC KEY J. (1992): Use of near-isogenic lines in analysing race-specific pathogen interrelations. Norwegian J. Agric. Sci., Suppl., **7**: 63–76.

MAC KEY J., MATTSSON B. (1972): Breeding for race specific resistance against Scandinavian oat stem rust. Sver. Utsaedesfoeren. Tidskr., **82**: 186–203.

MARTENS J. W. (1981): Incidence and virulence of *Puccinia graminis* f. sp. *avenae* in Canada in 1980. J. Plant Pathol., **3**: 231–234.

MARTENS J. W. (1985): Oat stem rust. In: ROELFS A. P., BUSHNELL W. R (Ed.): The Cereal Rusts. Vol. II. New York, Acad. Press: 103–129.

MARTENS J. W., DYCK P. L. (1989): Genetics of resistance to rust in cereals from a Canadian perspective. Can. J. Plant Pathol., **11**: 78–85.

MARTENS J. W., MCKENZIE R. I. H., GREEN G. J. (1967): Thermal stability of stem rust resistance in oat seedlings. Can. J. Bot., **45**: 451–458.

MARTENS J. W., MCKENZIE R. I. H., FLEISCHMANN G. (1968): The inheritance of resistance to stem and crown rust in Kyto oats. Can. J. Genet. Cytol., **10**: 808–812.

MARTENS J. W., ROELFS A. P., MCKENZIE R. I. H., ROTHMAN P. G., STUTHMAN D. D., BROWN P. D. (1979): System of nomenclature for races of *Puccinia graminis* f. sp. *avenae*. Phytopathology, **69**: 293–294.

MARTENS J. W., MCKENZIE R. I. H., HARDER D. E. (1980): Resistance to *Puccinia graminis avenae* and *P. coronata avenae* in the wild and cultivated *Avena* populations of Iran, Iraq, and Turkey. Can J. Genet. Cytol., **22**: 641–649.

MARTENS J. W., ROTHMAN P. G., MCKENZIE R. I. H., BROWN P. D. (1981): Evidence for complementary gene action conferring resistance to *Puccinia graminis avenae* in *Avena sativa*. Can. J. Genet. Cytol., **23**: 591–595.

MCDANIEL M. E. (1992): Playing to win against oat rusts. In: Proc. 4th Int. Oat Conf., Adelaide, South Australia, October 20th–23rd, Vol. III: 26–32.

MCKENZIE R. I. H. (1961): Inheritance in oats of reaction to race 264 of oat crown rust. Canad. J. Genet. Cytol., **3**: 308–311.

MCKENZIE R. I. H. (1964): Dangerous new races of oat stem rust and sources of resistance to them. Can. J. Plant Sci., **44**: 418–426.

MCKENZIE R. I. H., GREEN G. J. (1965): Stem rust resistance in oats. I. The inheritance of resistance to race 6AF in six varieties of oats. Can. J. Genet. Cytol., **7**: 268–274.

MCKENZIE R. I. H., MARTENS J. W. (1968): Inheritance in the oat strain C.I. 3034 of adult plant resistance to race C 10 of stem rust. Crop Sci., **8**: 625–627.

MCKENZIE R. I. H., FLEISCHMANN G., GREEN G. J. (1965): A close association of stem rust and crown rust resistance in Ukraine and Rosen's Mutant oats. Crop Sci., **5**: 551–552.

MCKENZIE R. I. H., MARTENS J. W., FLEISCHMANN G., SAMBORSKI D. J. (1968): An association of stem rust and crown rust resistance in Jostrain oats. Can. J. Genet. Cytol., **10**: 190–195.

MCKENZIE R. I. H., MARTENS J. W., MALLOUGH E. D., FLEISCHMANN G. (1976): Registration of Hudson oats. Crop Sci., **16**: 740–741.

MCKENZIE R. I. H., MARTENS J. W., RAJHATHY T. (1970): Inheritance of oat stem rust resistance in a Tunisian strain of *Avena sterilis*. Can. J. Genet. Cytol., **12**: 501–505.

MCKENZIE R. I. H., FLEISCHMANN G., MARTENS J. W. (1971a): Oat rust resistance through gene management. In: The way ahead in plant breeding. In: Proc. 6th Congr. Eucarpia, Cambridge, UK: 127–128.

MCKENZIE R. I. H., MARTENS J. W., GREEN G. J. (1971b): The oat stem rust problem. In: Proc. Mutation Dis. Resist., Vienna, Austria, IAEa et FAO: 151–157.

MCKENZIE R. I. H., MARTENS J. W., BROWN P. D., HARDER D. E., NIELSEN J., BOUGHTON G. R. (1981): Registration of Fidler oats. Crop Sci., **21**: 632–633.

MCKENZIE R. I. H., BROWN P. D., MARTENS J. W., HARDER D. E., NIELSEN J., GILL C. C., BOUGHTON G. R. (1984): Registration of Dumont oats. Crop Sci., **24**: 207.

MIELKE H. (1975): Über die Blattfleckenkrankheit (*Septoria avenae*) des Hafers. Mitt. Biol. Bundesanst. Land-Forstwirtsch. Berlin-Dahlem, **163**: 41–47.

MYAGKOVA D. V. ZHUKOVA A. E., RODIONOVA N. A., SOLDATOV V. N. (1982): Sources of resistance to fungus diseases in oats. Tr. Prikl. Bot., Genet. Selek., **71**: 58–62.

MULLER H. J. (1963): Untersuchungen uber Blattfleckenkrankheiten des Hafers. II. Pilzliche Blattfleckenerreger des Hafers. Phytopath. Z., **49**: 266–290.

MULLER H. J. (1964): Untersuchungen uber Blattfleckenkrankheiten des Hafers. II. Pilzliche Blattfleckenerreger des Hafers: *Helminthosporium avenae* Eidam, *Septoria avenae* Frank, *Helminthosporium sativum* P., K. et B., *Fusarium* spec. und *Heterosporium avenae* Oud. Phytopath. Z., **49**: 266–290.

MUNDT C. C., BROPHY L. S. (1988): Influence of number of host genotype units on the effectiveness of host mixturers for diseases control: a modelling approach. Phytopathology, **78**: 1087–1094.

NAQUI Z. U.-H. (1990): Genetic studies of *Avena sativa* using aneuploids. Unpublished PhD Thesis. University College of Wales, Aberystwyth.

NIELSEN J. (1977a): Occurrence in western Canada of races of covered smut (*Ustilago kolleri*) virulent on cultivars of oats with resistance from Victoria. Can. J. Plant Sci., **57**: 611–612.

NIELSEN J. (1977b): A collection of cultivars of oats immune or highly resistant to smut. Can. J. Plant Sci., **57**: 199–212.

NIELSEN J. (1978a): Two grasses as common hosts for all species of *Ustilago* parasitic on wheat, barley, and oats. Can. J. Bot., **56**: 2712–2716.

NIELSEN J. (1978b): Frequency and geographical distribution of resistance to *Ustilago* in six wild species of *Avena*. Can J. Plant Sci., **58**: 1099–1101.

NOBLE M., MONTGOMERIE I. G. (1956): *Griphosphaeria nivalis* (Schaffnit) Muller et von Arx and *Leptosphaeria avenaria* Weber on oats. Trans. Br. Mycol. Soc., **39**: 449–459.

OLOFSSON B. (1976): Undersokningar rorande Drechslera-arte has korn och havre. Meddel. Stat. Vaxtskyd., **16**: 323–425.

OSLER R. D., HAYES H. K. (1953): Inheritance studies in oats with particular refernce to the Santa Fé type of crown rust resistance. Agron. J., **45**: 49–53.

PANDEY S. C., MISRA A. P. (1973): Varietal reaction of oats to *Helminthosporium avenae*. Ind. J. Mycol. Pl. Pathol., **3**: 214–216.

PARKER J. H. (1920): A preliminary study of the inheritance of rust resistance in oats. Agron. J., **12**: 23–38.

PENNER G. A., CHONG J., WIGHT C. P., MOLNAR S. J., FEDAK G. (1993a): Identification of an RAPD marker for the crown rust resistance gene *Pc 68* in oats. Genome, **36**: 818–820.

PENNER G. A., CHONG J., LÉVESQUE-LEMAY M., MOLNAR S. J., FEDAK G. (1993b): Identification of a RAPD marker linked to the oat stem rust gene *Pg 3*. Theor. Appl. Genet., **85**: 702–705.

POLITOWSKI K., BROWNING J. A. (1978): Tolerance and resistance to plant disease: an epidemiological study. Phytopathology, **68**: 1177–1185.

RAYAPATI P. J., GREGORY J. W., LEE M., WISE R. P. (1994): A linkage map of diploid *Avena* based on RFLP loci and a locus conferring resistance to nine isolates of *Puccinia coronata* var. *avenae*. Theor. Appl. Genet., **89**: 831–837.

REKOLA O., RUOKOLA A. L., KURTTO J. (1970): Damage caused by *Helminthosporium avenae Eidam.* on the crop yield of oats in Finland. Acta Agr. Scand., **20**: 225–229.

RODERICK H. W., CLIFFORD B. C. (1995): Variation in adult plant resistance to powdery mildew in spring oats under field and laboratory conditions. Plant Pathol., **44**: 366–373.

RODERICK H. W., JONES I. T. (1991): The evaluation of adult plant resistance to powdery mildew (*Erysiphe graminis* f. sp. *avenae*) in transgressive lines of oats. Euphytica, **53**: 143–149.

RODERICK H. W., JONES E. R. L., CLOTHIER R. B. (1995): Breeding oats for resistance to powdery mildew in the UK. In: 1st Europ. Oat Dis. Nursery Workshop, Prague, 1993, Petria **5**: 76–77.

ROELFS A. P., LONG D. L., CASPER D. H. (1980): Races of *Puccinia graminis* f. sp. *avenae* in the United States during 1979. Plant Dis., **64**: 947–949.

ROELFS A. P., LONG D. L., CASPER D. H. (1982): Races of *Puccinia graminis* f. sp. *avenae* in the United States and Mexico during 1980. Plant Dis., **66**: 208–209.

ROELFS A. P., CASPER D. H., LONG D. L., ROBERTS J. J. (1989): Races of *Puccinia graminis* in the United States and Mexico during 1987. Plant Dis., **73**: 385–388.

ROELFS A. P., CASPER D. H., LONG D. L., ROBERTS J. J. (1990): Races of *Puccinia graminis* in the United States in 1988. Plant Dis., **74**: 555–557.

ROELFS, A. P., LONG, D. L., ROBERTS, J. J. (1995): Races of *Puccinia graminis* in the United States during 1993. Plant Dis., **79**: 967–972.

ROONEY W. L., RINES H. W., PHILLIPS R. L. (1994): Identification of RFLP markers linked to crown rust resistance genes *Pc91* and *Pc92* in oat. Crop Sci., **34**: 940–944.

SCHAFER J. F. (1971): Tolerance to plant disease. Ann. Rev. Phytopathol., **9**: 235–252.

ŠEBESTA J. (1975a): Fragen zur Elternwahl bei der Getreidezuchtung auf Resistenz gegen Rostkrankheiten. Bericht Arbeitstagung 1975, Gumpenstein (Austria), 25–27 Nov.: 215–227.

ŠEBESTA J. (1975b): Moderní metody boje proti obilním rzím. ÚVTI, Stud. Inform., Ochr. Rostl., č. 1.

ŠEBESTA J. (1976): Dědičnost vertikální rezistence ovsů k *Puccinia graminis* Pers. f. sp. Erikss. et Henn. *a P. coronata* Cda *var. avenae* Fraser et Led. In: Sbor. věd. Pr. VI. československ. Konf. Ochr. Rostl., České Budějovice, IIB: 207-210.

ŠEBESTA J. (1977): Dědicnost vertikalni rezistence ovsa odrůd Dodge a Garland ke rzi travní (*Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn.) ve střední Evropě. Genet. a Šlecht., **13**: 239–244.

ŠEBESTA J. (1978): Complete or incomplete dominance of resistance as a function of crown rust culture. In: 3rd Int. Congr. Plant Pathol., Munchen (FRG), 16–23 August 1978.

ŠEBESTA J. (1979a): Complete or incomplete dominance of resistance pf the oat cultivar Delphin as a function of crown rust culture. Euphytica, **28**: 807–809.

ŠEBESTA J. (1979b): Untersuchungen über die Virulenz der Getreideroste aus dem Gesichtspunkt der Zuchtung auf die multigene Resistenz. Bericht Arbeitstagung 1979, Gumpenstein (Austria), 27–29 November: 199–222.

ŠEBESTA J. (1980a): Zur Problematik der chemischen Bekampfung des Schwarzrostes an Weizen. Bericht Arbeitstagung 1980, Gumpenstein (Austria), 25–27 November: 163–177.

ŠEBESTA J. (1980b): Probleme Monogen-resistenter Getreidesorten gegenüber Rostkrankheiten. Bericht Arbeitstagung 1980, Gumpenstein (Austria), 25–27 November: 49–161.

ŠEBESTA J. (1983): Race specific expression of oat crown rust resistance conditioned by major and minor genes. Euphytica, **32**: 857–861.

ŠEBESTA J. (1985): European oat disease nursery, survey report 1978–1984. Res. Inst. Crop Product. Prague, Czechoslovakia.

ŠEBESTA J. (1987): Vliv rezistence a tolerance ke rzím a padlí travnímu na výnos zrna ovsa. Ochr. Rostl., **23**: 23–34.

ŠEBESTA J. (1988): Resistance of oats to rust diseases. In: 3rd Int. Oat Conf., Lund, Sweden, July 4–8: 265–278.

ŠEBESTA J. (1990a): Oat resistance to rusts and powdery mildew in central Europe. A. Rep., Res. Inst. Crop Product., Divis. Plant Protect. Praha-Ruzyně: 12–13.

ŠEBESTA J. (1990b): European Oat Disease Nursery Annual Report 1990. FAO Eur. Syst. Cooperative Res. Networks Agric., Prague.

ŠEBESTA J. (1991): Hodnocení chorob polních plodin z hlediska šlechtění na odolnost. Vědeckotechnicky rozvoj v zemědělství. Rostl. Výr., 10.

ŠEBESTA J. (1991): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1992): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1993): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1994): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1995): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1996): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1997): European Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J. (1998): European and Mediterranean Oat Disease Nursery. A. Rep. FAO Eur. Syst. Coop. Res. Networks Agric., Prague.

ŠEBESTA J., ČERVENKA J. (1978): Šlechtení obilnin na multigenní rezistenci ke rzím. In: Sbor. věd. prací VII. českosl. konf. ochrane rostlin, Nitra, 6.–8. 9: 123.

ŠEBESTA J., HARDER D. E. (1983): Occurrence and distribution of virulence in *Puccinia coronata var. avenae* in Europe, 1977–1980. Plant Dis., **67**: 56–59.

ŠEBESTA J., SÝKORA, J. (1974): Škodlivost rzí na ovsu jako rasová a odrůdová zvlástnost. Ochr. Rostl., **10**: 265–269.

ŠEBESTA J., ZWATZ B. (1977): A new virulence gene in Central European populations of *P. graminis* f. sp. *avenae* Erikss. et Henn. Cereal Rusts Bull., **5**: 11–12.

ŠEBESTA J., ZWATZ B. (1980): Virulenz der mitteleuropaischen Rassenpopulationen der Schwarzrostes des Hafers (*Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn.) unter besonderer Berucksichtigung der Wirkamsamkeit der Resistenzgene. Pfl.-Schutz Berichte, XLXI, 1/5.

ŠEBESTA J., ZWATZ B. (1995): Resistenzzuchtung gegen Krankheiten des Hafers im Hinblick auf das Auftreten neuer Krankheiten (FAOPROJEKT). 46. Tagung der Vereinigung osterr. Pflanzenzuchter, 21–23 November: 117–122.

ŠEBESTA J., HARDER D. E., JONES I. T., CLIFFORD B. C., ZWATZ B. (1983): Resistance of oats to rusts and powdery mildew in Europe. In: Abstr. 4th Int. Congr. Plant Pathol., Melbourne, Australia, August 17–24: 207.

ŠEBESTA J., HARDER D. E., ZWATZ B. (1984): Resistance of oats to crown rust in central Europe. Cereal Rusts Bull., **12**: 7–17.

ŠEBESTA J., HARDER D. E., JONES I. T., KUMMER M., CLIFFORD B. C., ZWATZ B. (1985): Pathogenicity of crown rust, stem rust and powdery mildew on oats in Europe and sources of resistance. In: Proc. 2nd Int. Oat Conf., Aberystwyth (UK), July 15–18: 67–71.

ŠEBESTA J., HARDER D. E., ZWATZ B. (1987a): K otázce výskytu a patogenity evropských populací rzi ovesné (*Puccinia coronata Cda.* var. *avenae* Fraser et Led.) a donory rezistence. Česká Mykol., **41**: 97–106.

ŠEBESTA J., JONES I. T., KUMMER M., ZWATZ B. (1987b): The virulence of powdery mildew on oats in Europe, the effectiveness of resistance donors and the strategy of resistance breeding. Ochr. Rostl., **23**: 103–116.

ŠEBESTA J., HARDER D. E., JONES I. T., KUMMER M., ZWATZ B. (1989): Possibilities of breeding oats for rusts and powdery mildew resistance in Europe. In: XIIth EUCARPIA Congr. February 27–March 4, 1989, Gottingen, Germany, I: 4.

ŠEBESTA J., ZWATZ B., STOJANOVIĆ S., KUMMER M., HOPPE H. D., RODERICK H. W. (1991): Rust and mildew resistance of oats in central and south eastern Europe in 1983–1988. Cereal Rusts Powdery Mildew Bull., **19**: 9–16.

ŠEBESTA J., RODERICK H. W., CHONG J., HARDER D. E. (1993): The oat line *Pc*54 as a source of resistance to crown rust, stem rust and powdery mildew in Europe. Euphytica, **71**: 91–97.

ŠEBESTA J., FORMANOVÁ M., ŠÍP V., VACKE J., ŠKORPÍK M. (1994): Occurrence of *Pyrenophora avenae* Ito et Kurib. in the Czech Republic in 1992 and the varietal reaction of oat to it. Genet. a Šlecht., **30**: 1–8.

ŠEBESTA J., ČERVENKA J., SWIERCZEWSKI A., MULLER K. (1995a): Breeding oats for disease resistance in central Europe. Petria, **5**: 79–80.

ŠEBESTA J., ZWATZ B., CORAZZA L., RODERICK H. W. (1995b): Disease resistance index – a mult-site indicator of the effectiveness of plant genotypes against diseases. Ochr. Rostl., **31**: 167–176.

ŠEBESTA J., ZWATZ B., CORAZZA L., STOJANOVIĆ S. (1995c): Oat diseases in Europe and the European Oat Disease Nursery. In: 1st EODN Workshop, Prague, July 12–13. Petria, **5**: 35–49.

ŠEBESTA J., ZWATZ B., CORAZZA B. (1995d): Incidence of *Pyrenophora avenae* Ito et Kurib. in Europe and the varietal reaction of oat to it. Arch. Phytopath. Pfl.-Schutz., **29**: 485–490.

ŠEBESTA J., ZWATZ B., HARDER D. E., CORAZZA L., RODERICK H. W., STOJANOVIĆ S. (1996a): Incidence and resistance of oats to fungus diseases in Europe in 1988–1994. Ochr. Rostl., **32**: 103–113.

ŠEBESTA J., ZWATZ B., HARDER D.E., CORAZZA L., RODERICK H. W., STOJANOVIĆ S. (1996b): Resistance of oat to fungal diseases in Europe. In: Proc. Vth Int. Oat Conf. and the VIIth Int. Barley Genetics Symp., Canada, Saskatoon, **2**: 771–773.

ŠEBESTA J., ZWATZ B., HARDER D. E., CORAZZA L., STOJANOVIĆ S. (1997a): Incidence of crown rust and virulence of *Puccinia coronata* on oat and the effectivenes as of *Pc*-genes for resistance in Europe during 1990–1994. Arch. Phytopath. Pfl.-Schutz, **30**: 507–518.

ŠEBESTA J., RODERICK H. W., JONES I. T., JONES E. R. L., HERRMANN M., ZWATZ B., CORAZZA L. (1997b): The occurrence of oat powdery mildew in Europe and effectiveness of sources of resistance. Petria, **7**: 121–140.

ŠEBESTA J., ZWATZ B., RODERICK H. W., HARDER D. E., CORAZZA L., STOJANOVIĆ S. (1998): Incidence of oat stem rust and virulence of *Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn. on oat and the effectiveness of resistance in Europe during 1988 to 1996. Arch. Phytopath. Pfl.-Schutz., **31**: 393–413.

SHAW D. E. (1957a): Studies on *Leptosphaeria avenaria* f. sp. *avenaria*. Can. J. Bot., **35**: 97–112.

SHAW D. E. (1957b): Studies on *Leptosphaeria avenaria* f. sp. *triticea* on cereals and grasses. Can. J. Bot., **35**: 113–118.

SIMONS M. D. (1975): Heritability of field resistance to the oat crown rust fungus. Phytopathology, **65**: 324–328.

SIMONS M. D. (1979): Influence of genes for resistance to *Puccinia coronata* from *Avena sterilis* on yield and rust reaction of cultivated oats. Phytopathology, **69**: 450–452.

SIMONS M. D. (1985): Crown rust. The cereal rusts. Vol. II: 131–172.

SIMONS M. D., MARTENS J. W., MCKENZIE R. I. H., NISHIYAMA I., SADANAGA K., ŠEBESTA J., THOMAS H. (1978): Oats: A standardized system of nomenclature for genes and chromosomes and catalog of genes governing characters. U.S. Dep. Agric., Agric. Handbook 509: 1–40.

SKOVMAND B., ROELFS A. P., WILCOXSON R. D. (1978): The relationship between slow rusting and some genes specific for stem rust resistance in wheat. Phytopathology, **68**: 491–499.

STEWART D. M., ROBERTS B. J. (1970): Identifying races of *Puccinia graminis* f.s p. *avenae:* A modified international system. U.S. Dept. Agric., Techn. Bull., **1416**: 1–23.

SUZDALSKAYA M. V., BUKANOVA V. K., GORBUNOVA Y. U., KOROLEVA L. A. (1978): Virulence gene populations of the oat stem rust pathogen, (*Puccinia graminis* Pers. f. sp. *avenae*

Eriks. and E. Henn.) on the European territory of the USSR and in the Transcaucasus. Mikol. Fitopatol., **12**: 415–418.

THOMAS H., GRIFFITHS N. (1985): Alien chromosome substitution line. A. Rep. 1984, Welsh Plant Breed. Sta., Aberystwyth:. 102–103.

THOMAS H., LEGGETT J. M., JONES I. T. (1975): The addition of a pair of chromosomes of the wild oat *Avena barbata* (2*n* = 28) to the cultivated oat *A. sativa L.* (2*n* = 42). Euphytica, **24**: 717–724.

THOMAS H., POWELL W., AUNG T. (1980): Interfering with regular meoitic behaviour in *Avena sativa* as a method of incorporating the gene for mildew resistance from *Avena barbata*. Euphytica, **29**: 635–340.

TVEIT M. (1956): Pathogenicity of species of *Helminthosporium* from Brazilian oats. Phytopathology, **46**: 45–48.

UPADHYAYA Y. M., BAKER E.P. (1965): Studies on the inheritance of rust resistance in oats. III. Genetic diversity in the varieties Landhafer, Santa Fe, Mutica Ukraine, Trispernia, and Victoria for crown rust resistance. In: Proc. Linn. Soc. N.S.W., **90**: 129–151.

VANDERPLANK J. E. (1968): Disease Resistance in Plants. New York and London, Academic Press.

WATERHOUSE W. L. (1930): Initial results of breeding for rust resistance. Linnean Soc. New South Wales Proc., **55**: 596–636.

WEBER G. F. (1922): Septoria diseases of cereals. I. Speckled blotch of oats caused by *Leptospharia*. Phytopathology, **12**: 449–470.

WELSH J. N., JOHNSON T. (1951): The sopurce of resistance and the inheritance of reaction to 12 physiologic races of stem rust, *Puccinia graminis avenae* Erikss. and Henn.). Can. J. Bot., **29**: 189–205.

WELSH J. N., JOHNSON T. (1954): Inheritance of reaction to race 7A and other races of oat stem rust *Puccinia graminis avenae*. Can. J. Bot., **32**: 347–357.

WELSH J. N., PETURSON B., MACHACEK J. E. (1954): Associated inheritance of reaction to races of crown rust, *Puccinia coronata avenae* Erikss, and to Victoria blight, *H. victoriae* M. and M., in oatsa. Can. J. Bot., **32**: 55–68.

WILCOXSON R. D. (1981): Genetics of slow rusting in cereals. Phytopathology, **71**: 989–993.

WILLIAMS W., VERMA U. N. (1956): Investigations on resistance to disease among species of the genus *Avena*. I. Resistance to physiologic races of *Ustilago avenae* and *U. kolleri*. Ann. Appl. Biol., **41**: 405–416.

WISE R. P., GOBELMAN-WERNER K. S. (1993): Resistance to oat crown rust in diploid and hexaploid *Avena*. Plant Dis., **77**: 355–358.

WISE R. P., LEE M., RAYAPATI P. J. (1996): Recombination within a 5-centimorgan region in diploid *Avena* reveals multiple spe-cificities conferring resistance to *Puccinia coronata*. Phytopathology, **86**: 340–346.

WOLPERT T. J., NAVARRE D. A., MOORE D. L., MACKO V. (1994): Identification of the 100-kD victorin binding protein from oats. Plant Cell., **6**: 1145–1155.

WONG L. S. L., MCKENZIE R. I. H., HARDER D. E., MARTENS J. W. (1983): The inheritance of resistance to *Puccinia coronata* and of floret characters in *Avena sterilis*. Can. J. Genet. Cytol., **25**: 329–335.

YU-GONGXIN, BUSH A. L., WISE R. P., YU G. X. (1996): Comparative mapping of homoeologous group 1 regions and genes for resistance to obligate biotrophs in *Avena, Hordeum,* and *Zea mays*. Genome, **39**: 155–164.

ZWATZ B., ŠEBESTA J., HERRMANN M., KROLIKOWSKI J., CORAZZA L. (1994): Incidence of *Septoria avenae* in Europe in (1990–1993) and the varietal reaction of oats to it. Pflanzenschutzberichte, **54** : 129–135.

Souhrn

ŠEBESTA J., RODERICK H. W., STOJANOVIĆ S., ZWATZ B., HARDER D.E., CORAZZA L. (2000): **Genetický základ rezistence ovsa k houbovým chorobám**. Plant Protect. Sci., **36**: 23–38.

Jsou charakterizovány rasově specifická a rasově nespecifická rezistence a tolerance genotypů ovsa k houbovým chorobám. Specifická rezistence k houbovým chorobám je podmíněna dominantními nebo neúplně dominantními, méně recesivními nebo neúplně recesivními nezávislými geny, ale také geny v interakci (komplementární geny). Nespecifická rezistence (slow diseasing) a tolerance jsou většinou podmíněny recesivními geny s aditivními účinky. Předpokládá se, že molekulární markéry umožní významně zvýšit efektivnost šlechtění na odolnost. Markery založené na DNA nebo endospermových proteinových polymorfismech byly užity k identifikaci genů rezistence ke rzi ovesné a rzi travní. Je rozebrána problematika genetické ochrany ovsa k chorobám. Šlechtění ovsa na rezistenci ke rzi ovesné a rzi travní se v Kanadě a v Evropě orientuje na tvorbu multigenních odrůd, kdežto ve šlechtění na odolnost k padlí travnímu se doporučuje kombinace major genové rezistence s rezistencí v dospělosti, řízenou aditivními geny.

Klíčová slova: oves; houbové choroby; rezistence; tolerance; molekulární markery; *Puccinia coronata* f. sp. *avenae*; *P. graminis* f. sp. *avenae*; *Erysiphe graminis* f. sp. *avenae*; *Phaeosphaeria avenaria* f. sp. *avenaria*; *Pyrenophora avenae*

Corresponding author:

Doc. Ing. JOSEF ŠEBESTA, DrSc., Výzkumný ústav rostlinné výroby, odbor rostlinolékařství, 161 06 Praha 6-Ruzyně, Česká republika, tel.: + 420 2 33 02 24 68, fax: + 420 2 33 31 06 36, e-mail: sebesta@hb.vurv.cz