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EUROPEAN OAT DISEASE NURSERY BIOLOGICAL (GENETIC) CONTROL OF FUNGAL DISEASES OF OAT IN EUROPE

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INTRODUCTION

Characterization of the genetic control of plants

There are different concepts of biological control of diseases and pests. In the recent past the biological control meant only the use of "third-party" biological agents (COOK and VESETH, 1991).

In this book the concept of biological control is enlarged and includes also the breeding and growing resistant varieties. Resistance genes incorporated into the host plant control of pathogen (COOK, 1985).

Importance of oat crop in Europe and in the world

The hectarage of oat in the world has decreased by half over the last 50 years owing to reduction of oat acreage in the United States, Canada and Europe, oat having been replaced by other crops such as maize and barley. Oat is mainly cultivated for grain production, for feeding and food industries. However, the crop is of some importance also as a green fodder and for silage and haylage. The proportion for human consumption has been increasing due to recognition of good quality of oat protein and oil and the soluble fibre (MATTSSON, 1985).

Despite a considerable decline of oat acreage in the last decades, oat is constantly considered to be important cereal crop in the world. In Europe, it remains a major cereal in relation to wheat and barley in Finland and Sweden, of intermediate importance in Russia, Belarus and Poland, and of lesser importance in Germany and Ukraine (VALENTINE, 1996).

The nutritional value of oat grain and its effect on the health of humans and animals are well known since a long time. As reviewed by MCDONALD, SHINICK and INK (1992), recent research has identified several active components in oat kernel associated with specific health benefits such as lowering the level of blood lipids, regulating blood glucose metabolism and protecting against colon tumors.

The analyses of oat areas harvested in 1983 and grain yield in 1953 and 1983 (FORSBERG, 1985) indicated that the former USSR harvested by far the largest area (12,516,000 ha) followed by the United States, Australia, Canada and Poland. Average grain yields in the former USSR, USA, Australia, Canada, Poland, Germany FR, China, Spain, Finland, France and Sweden were 1,425.0 and 2,219.9 kg/ha in 1953 and 1983, respectively. Therefore, the grain production increase in this period was 59.8% (FORSBERG, 1985).

At present, Europe produces approximately a quarter of the world's oat, more than that produced in North America (VALENTINE, 1996). The oat area (in 1000 ha) in Europe and North America (Canada, Mexico, USA) in 1995, 1996 and 1997 was 3109, 3186 and 3233 and 2422, 2825 and 2731, respectively (FAO quaterly Statistics, 1998). Top ten European countries in oat production are as follow: Russian Federation, Germany, Ukraine, Poland, Finland, Sweden, Belarus, France, United Kingdom and Romania.

The breeding of oat crop is concentrated on categories such as grain quality, disease resistance (crown rust, stem rust, powdery mildew, Barley yellow dwarf), morphological characters and actual grain yield (FORSBERG, 1985).

Importance of the fungal diseases of oat

As the other cereals, oat is subjected to a large number of diseases (HARDER and HABER, 1992). Many of them can cause severe damage (Figs. 1–5) (ŠEBESTA, 1971, 1974, 1987, ŠEBESTA ET AL., 1972, ŠEBESTA and SYKORA, 1974). It can be assumed that the diseases are a major limiting factor in oat production (Figs. 6 and 7) (ŠEBESTA, 1971, 1987).

In addition, the quality of oat grain is considerably lowered by the physiological and morphological consequences of diseases (Figs. 8–10) (ŠEBESTA ET AL., 1972, ŠEBESTA and SYKORA, 1974, KRYZANEK and ŠEBESTA, 1974).

The grain of the attacked plants could even become unsuitable for their utilization by the food and feed industries (ŠEBESTA, 1974).

Among the better known fungal diseases of economic importance in oat are crown rust (Puccinia coronata Cda. f. sp. avenae Eriks.) (Fig. 1), stem rust (P. graminis Pers. f. sp. avenae Eriksss. et Henn.) (Fig. 2), powdery mildew (Erysiphe graminis D. C. f. sp. avenae Em. Marschal) (Fig. 3), loose and covered smuts incited by Ustilago avenae (Pers.) Rostr. and Ustilago hordei (Pers.) Lagerh. f. sp. avenae Boerema et Verhoeven, respectively (ŠEBESTA ET AL., 1995).

Among the less known oat fungal diseases, at least from the point of view of their genetic interaction with the host, might be included Septoria leaf blight and black stem (Stagonospora avenae/Frank./Besset, perfect state Phaeosphaeria avenaria (G. F. WEBER, O. ERIKSSON) (Fig. 4), Helminthosporium leaf blotch (Drechslera avenae Eidam Scharif, perfect state Pyrenophora avenae Ito et Kuribay) (Fig. 5), Fusarium blight (F. graminearum Schwabe, F. avenaceum (Fr.) Sacc.) Fusarium common root rot (Fusarium culmorum (W. G. SMITH) Sacc.)

International cooperation in oat disease resistance breeding

After finalizing a number of fundamental studies on disease resistance breeding in cereals, especially in oat in 1969, the senior author of the publication realized very early that a key to success in this work is in a close international cooperation. A good cooperation was opened with plant pathologists and plant breeders from Austria, Germany (East) and Poland.

The first, joint project was called "Oat Rust Nursery" and covered the most effective donors of resistance to stem rust and crown rust found by the senior author in the foregoing studies.

As the number of diseases in target and the number of countries involved was soon enlarged, the project was re-named on "European Oat Disease Nursery" (EODN). In 1976 the EODN trials were established at 31 locations in 11 countries of the Continent.

In 1990, 21 years after the establishment of the first international trials, the EODN project was included into the European System of Cooperative Research Networks in Agriculture (ESCORENA) of Food and Agriculture Organization (FAO), 31 national cooperators in 18 European countries participated in this project.

In 1997 the trials of the EODN were established in 19 European countries (Austria, Belarus, Bulgaria, Czech Republic, Estonia, Finland, Germany, Great Britain, Greece, Holland, Hungary, Italy, Norway, Poland, Russia, Slovakia, Spain, Sweden and Yugoslavia) and in Israel and Morocco (Table 11). In 1998, owing to participation of Israel and Morocco, the project was re-named on European and Mediterranean Oat Disease Nursery (EMODN).

To monitor disease incidence and to clarify the effectiveness of resistance sources to diseases is of primary importance, for oat resistances included in the project are immediately available and they can be appropriately used in breeding programmes. (ŠEBESTA ET AL., 1997).

The disease resistance breeding programmes in oat achieved during this period (1969–1997) remarkable success in a number of countries involved.

Oat Fungal Diseases in Europe and their Genetic Control

Disease resistance index – A multi-site indicator of the effectiveness of plant genotypes against diseases

Evaluating new germplasm to identify potential donors of disease resistance is of prime importance in crop improvement. Obtaining information on host reactions to pathogenic fungi and viruses from different regions where particular diseases are prevalent is especially valuable. The more information we have regarding the effectiveness and durability of resistant donors the better we can make decisions regarding the deployment of these resistances.

Up to now we have lacked an index which uses data on the effectiveness of different resistant genotypes between sites in different regions where the crop is grown. The proposed disease resistance index is calculated on the basis of resistant and moderately resistant quantitative reactions to diseases.

The disease resistance index was tested using data collected from the European Oat Disease Nursery in 1990–1994, for *Puccinia coronata* f. sp. *avenae, Phaeosphaeria avenaria* f. sp. *avenaria* (ZWATZ ET AL., 1994) and *Pyrenophora avenae Ito et Kurib* (ŠEBESTA ET AL., 1995). The European Oat Disease Nursery is a collection of oat genotypes with resistance to different fungal and virus diseases and is grown annually in field nurseries at research or plant breeding centers throughout Europe (ŠEBESTA, 1990–1997).

Several assessment scales were used in different countries to record disease severity, these were:

I/James assessment key (JAMES, 1971) (J)

II/a 1–9 'western' scale (where 1 = no disease, up to 9 severe infection, > 70%) (W)

III/a 1–9 'eastern' scale (where 9 = no disease, to 1 severe infection, > 70%) (E) Therefore, to unify the assessments the following transformations of each method were carried out:

Resistance score Assessment scales		
Resistant (R)	4	1– 4 (J), 1–2 (W), 9–8 (E)
Moderately resistant (MR)	3	5–24 (J), 3–4 (W), 7–6 (E)
Moderately susceptible (MS	2	25–49 (J), 5–6 (W), 5–4 (E)
Susceptible (S)	1	50 + (J), 7–9 (W), 3–1 (E)

The level of the diseases at individual localities and years was evaluated on the occurrence on susceptible cultivars as weak (1–3 W, 9–7 E), moderate (4–6 W, 6–4 E) and high (7–9 W, 3–1 E). Just the data recorded at the moderate and high levels of occurrence of the pathogens were used for processing (ŠEBESTA ET AL., 1995).

For the purpose of calculating a disease resistance index (DRI) only the resistant and moderately resistant evaluations are used, so that the DRI for each genotype is the sum of number of locations with an R category score multiplied by the resistance score and number of locations with MR category score multiplied the resistance score, e. g. in Avena sterilis L. CAV 2648 a DRI of 48 for Ph. avenaria f. sp. avenaria was calculated thus (9 x 4) + (4 x 3). The adjustment of the calculated DRI to the same number is then carried out in individual genotypes. The genotypes are ranked according to their resistance index.

The combined disease resistance index (CDRI) could only be calculated as the average of all indices. But it should only be used for comparisons if the numbers of sites is constant (ŠEBESTA ET AL., 1995).

Effect of resistance and tolerance to rusts and powdery mildew on the grain yield of oat

Twenty-four cultivars and release of oat were analyzed for their qualitative and quantitative reactions to crown rust, stem rust and powdery mildew (ŠEBESTA, 1987).

On the basis of these analyses, these genotypes could be evaluated as to the stability of their 1000-grain weight (TGW), which in central Europe is one of the most important indicators of the harmful effect of rusts and powdery mildew on oat.

Oat crown rust caused a comparatively low reduction of the TGW in the resistant cultivars and new advanced lines including Roxton, Szegedi 30, KR 106/31 and KR 2209, but also in the susceptible cvs. Petkus 7573 and Maelor.

Stem rust caused a relatively small damage to the cv. Solidor, KR 106/31 and Szegedi 30.

Powdery mildew with crown rust did not damage much the cv. Roxton, KR 106/31 and Szegedi 30.

The stability of the 1000-grain weight is indicated as a function of genotype of the host plant.

The effectiveness of the qualitative (specific) and quantitative resistance and the problems of the genotype tolerance and its detection were analyzed (Tables 8, 9, 10).

Crown rust

Characterization of the pathogen

Crown rust (*Puccinia coronata* Cda. f. sp. *avenae* Eriks.) belongs to the most widespread and damaging diseases of oat. It affects leaves, sheaths and panicles (Fig. 1). The uredial pustules (uredinia) are oblong and yellow-orange. The telia of P. coronata are dark and form rings around uredinia. The inoculum responsible for epidemics of crown rust in Europe comes either from wild oat species, other host grasses and volunteer oat plants or from alternate host, *Rhamnus catharticus.*

The transport of urediospores like in other cereal rusts is supposed to be realized by wind from a long distance, from south-eastern Europe (ZADOKS, 1965, ŠEBESTA and BARTOS, 1969).

Effects of crown rust on quantity and quality of oat yield

In exact experiments, under artificial inoculatin of the spreader rows, rust decreased grain yield of oat by 15–18%, 1.000-grain weight by 8–20%. Percentage of hulls was increased by l2–24% (Tables 1,2) (ŠEBESTA, 1971).

Crude protein and bound amino acids content crown rust decreased by 18%. Crown rust like stem rust decreased most the content of basic amino acid histidine, less the content of arginine and lysine. The content of phenylalanine was sharply decreased. Crown rust, unlike stem rust especially decreased methionine content. Basically, results suggested that both crown rust and stem rust affect with some exceptions, the same amino acids according to their harmfulness (Tables 3, 4a, 4b) (ŠEBESTA ET AL., 1972).

The content and proportions of free amino acids and oligosaccharides was examined in oat kernels affected by oat stem rust and oat crown rust. The results showed a considerable decrease of the content of aspartic acid, glutamic acid, the serine-glycine fraction and the asparagine and glutamine fraction. The content of oligosaccharides was decreased only slightly (Table 4b; Figures 8, 9, 10).

In other exact experiments it was indicated that as to the harmfulness there are distinct and varietal differences in crown rust and stem rust as well. (Tables 6, 7).

In crown rust, race 239, widely distributed in central Europe, was much more harmful than the race CS 1, isolated for the first time. In the grain the races affected especially the content of histidine, phenylalanine, tyrosine, aspartic acid, serine and valine. The reduction of crude protein corresponded especially to reduction of histidine. Race 239 lowered it about 42 p. c. and the race CSl by 28 p.c. (ŠEBESTA and SÝKORA, 1974). Other experiments confirmed expressive effects of both crown rust and stem rust on the composition of kernels and straw as well. The content of nitrogene substance and the amount of starch units was significantly decreased in the kernels but the fibre content was increased. On the other hand, the content of nitrogene substances and the amount of starch units was significantly increased in straw. The content of ash was decreased by both rusts whereas the fibre content only by stem rust in the straw (Tables 5 and 6) (ŠEBESTA, 1974).

Incidence of crown rust in Europe as recorded in the EODN trials in 1990–1997

The occurrence of oat crown rust in the EODN locations is shown in Table 12. The disease was wide-spread in Europe during 1990–1997 In the EODN trials high or moderate natural levels of incidence of the disease were recorded in Austria (8 times) Bulgaria (3 times), in the Czech Republic (6 times) (partially inoculated), Estonia (3 times), Finland (once), France (3 times), Great Britain (7 times) (inoculated), Israel (twice), Italy (5 times), Morocco (once), Poland (7 times), Russia (5 times), Slovakia (twice) and Yugoslavia (4 times).

From the incidence of oat crown rust recorded in Europe during 1990–1997 (ŠEBESTA, 1990–1997) it was concluded that the disease reached damaging levels in a number of localities of Europe during this period as it had in previous years (Table 12) (ŠEBESTA 1971, 1972, 1973, 1987, ŠEBESTA ET AL., 1972).

Virulence of *Puccinia coronata* Cda. f. sp. *avenae* Eriks. in Europe

Analyzed uredinial samples of the rust were obtained from oat lines and cultivars in the EODN trials (ŠEBESTA and ZWATZ, 1980, ŠEBESTA, 1990–1997), commercial oat fields, and wild oat *(Avena fatua* L.*)* in the 12 European countries and Israel. Rust fungal isolates were differentiated according to avirulence/virulence combinations on sets of single Pc-gene lines according to the method of Green (GREEN, 1965, ŠEBESTA and HARDER, 1983).

Detailed studies of the virulence combinations of P. coronata f. sp. avenae in Europe were carried out in 1992 and 1993. The spectra of avirulence/virulence combinations in twelve European countries and Israel are shown in Table 13.

In Austria in 1992 and 1993 nine virulence combinations were identified. The pathotypes ranged from virulence to one (/Pc 56, /63, /67), two (Pc-50-4, 56), three (/Pc 54-2, 56, 63), four (/Pc 38, 50-4, 62, 64, /Pc 50-2, 50-4, 62, 64, /Pc 54-2, 60, 61, 64) to five (/Pc 38, 50-2, 50-4, 62, 64) resistance genes. The pathotype with virulence to Pc 56 was also isolated from the Polish population, that with virulence to Pc 67 from the Czech, Italian, Slovak and Swedish populations. The pathotype with virulence to Pc 54-2, Pc 60, Pc 61 and Pc 64 was isolated in Austria twice and also was identified in Yugoslavia. The average number of virulences in Austria was 2.8.

From the Belgian *P coronata* f. sp. *avenae* population in 1993, only one pathotype that atacked Pc 64 was identified. The same pathotype also was identified in the Czech Republic, France and the United Kingdom.

In the Czech Republic in 1992 and 1993, twenty pathotypes were isolated that were virulent to: none (two isolates), one (Pc 38, Pc 54-2, Pc 64, Pc 67), two (Pc 38, 63, Pc 38, 64, Pc 56, 67, Pc 38, 67), three (Pc 50-4, 63, 67, Pc 56, 62, 64, Pc 38, 48, 63, Pc 38, 56, 63), four (Pc 54-2, 60, 61, 67, Pc 38, 48, 63, 67, Pc 38, 56, 64, 67), and five (Pc 38, 48, 50-4, 54-2, 56, Pc 56, 62, 63, 64, 67) resistance genes.

The pathotypes that were virulent to none, two (Pc 38, 63, Pc 38, 64) and four resistance genes were isolated, respectively, twice, and there were three isolates virulent to Pc 38, 48, 63. The average number of virulences in the Czech Republic was 2.6.

In the Estonian *P. coronata* f. sp. *avenae* population there were no virulences to any of the genes that were used in the Pc set.

In the French crown rust populations seven pathotypes were identified that were virulent to one (Pc 64), two (Pc 64, 67), three (/Pc 38, 63, 67, /Pc 54-2, 60, 67, /Pc 56, 64, 67), and six (/Pc 38, 56, 62, 63, 64, 67) Pc genes. Pathotype /Pc-64 , 67, which was isolated twice in France, was also indentified three times in the United Kingdom. The average number of virulences in France was 2.9.

From the United Kingdom in 1992 and 1993, six pathotypes were identified possessing no virulence (three times), virulences to one (/Pc-64) (three times), two (/Pc 64, 67, /Pc 48, 54-2) and three (/Pc-50-4, 62, 64, /Pc-54-2, 60, 61), Pc-genes. The pathotype/Pc-64, 67 also was isolated three times. Interesting is that the latter was also isolated in France (twice) and the pathotype /Pc-48, 54-2 also was isolated in Spain. The average number of virulences in the United Kingdom was 1.8.

Italian crown rust populations contained pathotypes possessing virulences to one (/Pc-67), five (/Pc-54-2, 60, 61, 62, 64, /Pc-54-2, 60, 61, 64, 67) and six (/Pc-54-1, 54-2, 60, 61, 63, 67)

Pc-genes. The average number of virulence genes was in Italy 4.3, the highest of the all checked regions.

From the two Polish crown rust populations only two pathotypes were isolated, possessing virulences, respectively, to one (/Pc-56), and five (/Pc-38, 56, 62, 63, 64) Pc-genes.

In Slovakia, mainly in 1992, ten avirulence combinations were identified possessing virulences to none, one (/Pc-38, /Pc-67), two (/Pc-54-2, 64), three (/Pc-38, 48, 63, /Pc-38, 63, 64), four (/Pc-38, 50-4, 56, 63, /Pc-38, 56, 64, 67), five (/Pc-38, 48, 50-2, 50-4, 63) and six /Pc-38, 56, 62, 63, 64, 67) Pc-genes. The first pathotype with no virulence was isolated three times and those possessing virulences to Pc-38 and Pc 67 were isolated twice. The pathotypes /Pc-67, /Pc-38, /Pc-38, 48, 63 and /Pc-38, 64, 67 were also isolated in the Czech Republic, and the pathotype /Pc-38, 56, 62, 63, 64, 67 also occurred in France. The average number of virulences in the Slovak crown rust populations was 2.9.

From the Spanish crown rust population in 1992, two avirulence /virulence combinations were isolated possessing virulences to two (/Pc-48, 54-2) and five (/Pc-54-1, 54-2, 60, 61, 67) Pc-genes, with the former one also isolated in the United Kingdom.

In the Swedish crown rust population only pathotype possessing virulence of Pc-67 was identified.

From the Yugoslav crown rust populations five pathotypes were isolated that possessed virulence to none, two (/Pc-38, 67), three (/Pc-38, 56, 67), four (/Pc-54-2, 60, 61, 64) and six (/Pc-54-1, 54-2, 59, 60, 64, 67) Pc-genes. The average number of virulences of the Yugoslav pathotypes of oat crown rust was 3.0.

From the three Israeli crown rust populations in 1993, three pathotypes were isolated with virulences to three (/Pc-54-2, 60, 61), five (/Pc-50-2, 50-4, 60, 61, 67) and six (/Pc-38, 39, 48, 54-1, 54-2, 55) Pc-genes. The latter pathotype is important because of its virulence to genes Pc-39 and Pc-55.

Effectiveness of crown rust resistance genes transferred from the wild oat *Avena sterilis*

The effectiveness of various Pc-genes to the European *P. coronata* f. sp. *avenae* populations are shown in Table 14 and demonstrated in Figures 13–20. The resistance genes Pc-39, Pc-55, Pc-58 and Pc-68 were completely effective against all pathotypes of *P. coronata* f. sp. *avenae* isolated in Europe in 1992 and 1993 (Table 14).

In addition, genes such as Pc-48, Pc-50-2, Pc-50-4, Pc-54-1 and Pc 59 also appear to be useful resistance sources for breeding disease resistant oat in Europe.

Gene Pc-48 was effective against all ten crown rust isolates from Austria and against all of the Belgian population. Of the twenty-six crown rust samples from the Czech Republic, gene Pc-48 was overcome by five of the samples. Against the Estonian population, gene Pc-48 and all other resistance genes tested were effective. All of the French crown rust samples were avirulent to Pc-48. Of the twelve crown rust samples from the United Kingdom, however, one possessed virulence to Pc-48. It is remarkable that all five Italian and the two Polish samples of crown rust were avirulent to Pc-48. Of the fourteen Slovak crown rust samples, two possessed virulence on Pc-48, as did one of two Spanish samples and one Swedish sample. Similar to the Italian samples, all of the Yugoslav samples were avirulent to Pc-48. Of three Israeli crown rust samples, two were avirulent and one was virulent to Pc-48.

Gene Pc-50-2 showed somewhat higher effectiveness against the European crown rust samples as compared to Pc-48. Of the ten Austrian samples, eight were avirulent to Pc-50-2. All of the twenty-six Czech samples were avirulent to Pc-50-2. Gene Pc-50-2 also was effective against the Belgian, Estonian, French, British, Italian, Polish, Spanish, Swedish and Yugoslav

samples. Gene Pc-50-2, however, was overcome, respectively, by one Slovak and one Israeli sample.

The Pc-50-4 line was attacked by four of the 10 Austrian samples but only by one of the 26 Czech samples. This line was resistant to all Belgian, Estonian, French, Italian, Polish, Spanish, Swedish and Yugoslav samples, but was attacked by one sample obtained from each of Britain, Slovakia and Israel.

The same percentage of isolates were virulent to the Pc-54-1 line as to the Pc-50-2 line. The Pc-54-1 line was resistant to all Austrian, Belgian, Czech, Estonian, French, British, Polish, Slovak and Swedish samples, and was susceptible to one sample each from Italy, Spain, Yugoslavia and Israel.

The line with gene Pc-59 was susceptible only to one Yugoslav sample of all European samples analyzed.

The line with gene Pc-61 was susceptible to two samples each from Austria and Israel, to one sample from each to the Czech Republic, Britain, Spain, and Yugoslavia, and to four from Italy.

The line with gene Pc-62 was susceptible to three samples from Austria, two from Italy and one from each of Britain, France, Poland and Slovakia.

Disease resistance index (DRI) to crown rust in lines and cultivars of oat

The data in Table 15 show considerable differences in the disease resistance indexes (DRI) to crown rust among the 71 oats evaluated during 1990–1994. The adjustment of the calculated DRI values to the same number of sites enables a more precise comparison of resistance expressed by the various lines. No error is supposed among the oat lines tested at the identical number of localities. A larger inaccuracy in the calculated DRI values is expected if the difference in the number of checked sites between lines is higher.

The values of the DRI ranged from 13 to over 180. The highest resistance indexes occurred in those lines that had proven to have highly effective resistance in the seedling stage.

Among 21 of the most resistant lines (Tab. 15), the lines with Pc-68, Pc-58, Pc-50-2, Pc-59, and Pc-39, and Rodney E had DRI of 170 or higher, followed by Garland, Pc-50, Pc-63, IL 85-2069, Pc-55, Pen^{2}xCAV 1376, Rodney H, Pc-62 and the A. sterilis accession CAV 2648. Oat lines such as IL 86-5698, OA 504-5, Pc-56, KR 288/73L/569, OA 503-1, Cc4761, IL 86-1158, Pc-16, KR 3813/73, IL 86-6404, Pc-50-4, Rodney F and Roxton, however, may also have some importance in crown rust resistance breeding. The other group of lines beginning with the line Rodney D (DRI 139) and ending with the line Pc-67 (DRI 104) was of more variable effectiveness, perhaps with useful resistance in some region, but are not effective at all locations. Presumably, a number of the remaining lines also may have minor levels of crown rust resistance, at least in adult stage, but are not considered important for oat breeding.

Resistance of oat cultivars to crown rust

Oat cultivars tested in the seedling stage to races 228, 231, 239, 240 and CS 1 of oat crown rust were divided according to their reaction into five groups:

I. Cultivars highly resistant or immune to physiologic races 228, 231, 239 and mostly moderately resistant to physiologic race CS 1 (Bondvic, C. I. 7009, Landhafer, C. I. 7005, Santa Fe, C. I. 7006 and Trispernia, C. I. 7008 and cultivars or lines possessing a resistance derived from several of these cultivars.

II. Cultivars highly resistant or immune to physiologic races 228, 231, 239, and 240 and

susceptible to race CS 1 (Appler, C. I. 7003, Bond, C. I. 7004 and Victoria, C. I. 7002 resistance types).

III. Cultivars with combination more or less similar to Anthony, C. I. 7001 resistance type.

IV. Resistance combination of Ukraine, C. I. 7007 differential variety.

V. Cultivars with reaction combination more or less different from the preceding types (Abegweit, C. I. 4970, Ajax, C. I. 4157, Cody II, Curt, C. I. 7424, Palestine, C. I. 2696, and Tonka, C. I. 7192) (Table 16) (ŠEBESTA, 1970).

Of 130 cultivars and lines of oat tested in the seedling tests most genotypes were resistant to race 229, somewhat less to race 201 and essentially a lower number of resistant cultivars was found to races 203/216 and 214. The least number of oats possess the resistance to highly pathogenic race 265.

The resistance to all of these crown rust races was found in cultivars Dodge, Garland and Minnesota Oat Selection 643114.

A genetic basis for crown rust resistance of Garry and Rodney cultivars was identical. The cv. Rodney was resistant to races 201 and 229 and susceptible to races 203/216, 214 and 265, similarly as Bage Sel. 364 Klein, Buck 152 and other cultivars, whereas Garry, C. I. 6662 cultivar was only resistant to race 229. From the other derivatives of the cv. Victoria cvs. Burnett, Branch, Mo 0-205 and Vicar, CAN 827 were only resistant to race 229 (Table 17) (ŠEBESTA, 1970, 1972).

Resistance of 102 oat cultivars in the adult plant stage to the crown rust races 231, 239, 240, 282 and CS 1 was tested. Fourty-three cultivars were resistant, the same number indicated mesothetic reaction and the remaining cultivars were susceptible. Resistant and weakly attacked were e. g. cultivars Ag 313, Bentland, Bondvic, Cleo, Clintafe, Dodge, Floriland, Garland, Jefferson, Landhafer, Minland, Nora, Ora, Putnam 61, Santa Fé, Sunland, Trispernia and many others. From those of mesothetic reaction weakly acceptive were Bonda, Clinton x Arkansas, Jackson, La Prévision 13, Minton, Newton and others. For the most part, resistant cultivars were resistant in the seedling stage as well (Table 18) (ŠEBESTA, 1970).

Inheritance of crown rust resistance in oat

The crown rust resistance of cvs. Dodge and Garland to *P. coronata* f. sp. *avenae,* races 201, 228, 229, 230, 232, 238, 239 and 240 is conditioned by two independent dominant and probably by one recessive gene. The resistance to races 216, 265 and CS 1 is conditioned by one gene. The gene conferring resistance to race 265 is probably derived from the cv. Victoria while the gene effective against races 216 and CS 1 comes from the cv. Landhafer. It was proved that at least a part of the cvs. Dodge and Garland is identical.

The genes for crown rust resistance of the cv. Dodge and probably the cv. Garland as well are non-allelic with the gene Pc-39 so that these can be accumulated in one genotype. The question of breeding a multigenic cultivar by the use of these crown rust resistance sources is shown by ŠEBESTA (1977) (Tables 19–23).

Peculiarities in inheritance of crown rust resistance

The crown rust resistance of the cv. Delphin to ten *P. coronata* f. sp. *avenae* races was found to be conditioned by two complementary genes. These genes express a complete dominance in relation to the eight races, the F_2's segregating in a 9:7 ratio. On the other hand, the same genes behaved differently in relation to two other races, the F_2's segregating in 5:11 ratio. The double heterozygotes scored resistant with the former, and susceptible with the latter race group. Special experiments showed that the same genes were responsible for the resistant reaction to all avirulent races (ŠEBESTA, 1979).

An incomplete dominance of complementary genes observed by Baker and Upadhyyaya (1967) in cv. Bond at high temperatures and by us in the cv. Delphin to two races might be explained by assuming a heterozygotic constitution for pathogenicity in these races of the pathogen. (Tables 23, 26).

In the selected oat lines Pc 50-2 and Pc 50-4 (ŠEBESTA, 1983), identical in immune reaction to many crown rust races, an unusual difference in reaction to the Polish crown rust isolate 7-77 P was found. Whereas the Pc 50-4 was highly susceptible and severely attacked, the Pc 50-2 showed a reproducibly very low occurrence of resistant infection types. Genetic analyses of these lines to a rust isolate, avirulent to both lines, indicated that each of them contains a different major resistance gene. Furthermore, the crown rust resistance in Pc 50-2 was indicated to be influenced by a group of minor genes controlling the infection severity and the frequency of infection types when attacked with rust isolate 7-77 P (Figures 32 and 33) (Tables 27, 32) (ŠEBESTA, 1983).

Avena fatua L. subsp. *fatua v. glabrata* Peterm. *subv. pseudo-basifixa* Thell. as a source of crown rust resistance genes

Avena fatua L. is a common weed species in central Europe. As a hexaploid it can be crossed readily with commercially grown oats *(A. sativa* L.*)*. As pointed out by Briggle & Youngs (1975) characters of potential utility in *A. fatua* include disease resistance, early maturity, rapid growth rate, seed dormancy, high seed protein and high groat percentage. The resistance to crown rust, race 203, was noted by Briggle (1974) and Briggle (Youngs (1975).

An accession of *Avena fatua v. glabrata Peterm. subv. pseudo-basifixa Thell.* (*A. fatua* L. CS Sel. No. 1), collected in the former Czechoslovakia in 1971, was found to be resistant to a wide range of crown rust races (Fig. 34). Analyses of crosses of this oat with cvs. Weikuss, Leanda, Mona, Rodney A, Rodney B, Rodney M, Dodge and K 316 indicated that the resistance of *A. fatua* CS Sel. No. 1 is conditioned by one recessive gene which is in interaction with one partially dominant gene with additive effect. The expression of rust reaction was affected by temperature. The crown rust resistance genes of *A. fatua* L. CS Sel. No. 1 were non-allelic with stem rust resistance genes Pg-2 (A) and Pg-4 (B). (Tables 33–38) (ŠEBESTA and KUHN, 1990).

Induction to resistance to crown rust in oat

The phenomenon of induced resistance, also referred to as 'acquired immunity' or 'cross protection' in the rust diseases was first described by YARWOOD (1956) for the reaction of bean to *Uromyces phaseoli* and *Antirrhinum majus* to *Puccinia antirrhini.* LITTLEFIELD (1969) demonstrated its effects in flax to *Melampsora lini.* ELISTON ET AL. (1971) and KUC (1982) have reported on the cross protection resulting in bean from prior inoculation with avirulent isolates of several fungal pathogens. SKIPP and DEVERALL (1973) have reported a similar phenomenon in anthracnose on bean. OUCHI ET AL. (1976) have exhaustively discussed resistance reactions occurring in a number of pathogen-host plant associations.

In cereals, JOHNSON (1978) and JOHNSON and HUFFMAN (1968) reported the occurrence of a 'local antagonism' between cereal rust fungi. Induced resistance in wheat to *Puccinia graminis* was demonstrated by CHEUNG and BARBER (1972) and to *P. striiformis* by JOHNSON and ALLEN (1975) and JOHNSON and TAYLOR (1976). Induced resistance to *Erisyphe graminis* in barley has been reported by OUCHI ET AL. (1976). Cross-protection was demonstrated in oat seedling inoculated previously with *P. graminis tritici* or *P. recondita tritici* and challenged with virulent races of *P. coronata* or *P. graminis* by KOCHMAN and BROWN (1975). Strategies for the use of resistance genes for the control of diseases of small grains have been discussed by BROWNING and FREY (1981) and FREY ET AL. (1973).

The objective of this research was to determine if there are different levels of induced resistance resulting from several-challenger combinations of the pathogen inoculated on seedlings of three different host genotypes and to evaluate the potential of this induced resistance in controlling development of the pathogen.

Induction of resistance to crown rust, caused by *Puccinia coronata* Cda. f. sp. *avenae* Eriks., occurred in seedlings of three genetic lines of oats (*Avena sativa* L.) inoculated sequentially with one of three inducer-challenger combinations of the pathogen. There was significant reduction in both the number of pustules per leaf and the weight of urediospores harvested in all three host genotypes tested. There were highly significant differences between individual spore harvests correlated with age of the pustules.

Development of telia by the challenger was found to occur considerably later, and was less intense, when the challenger was inoculated alone in contrast to treatments in which the challenger followed pre-inoculation with the inducer. There was no correlation between the numbers of resistant and susceptible lesions within the range of inoculum density used in these experiments (ŠEBESTA ET AL., 1996).

Strategy of genetic control of oat crown rust

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., 1971 – cit. MARTENS, 1985) and also in Europe (Figures 45–47) (ŠEBESTA, 1979 b, ŠEBESTA ET AL., 1997). It is supposed that the cultivation of multigenic cultivars and, especially if connected with spatial (regional) deployment of genes, can significantly prolong the usefulness of the resistance genes (ŠEBESTA, 1979, 1988).

A synopsis of studies of inheritance of resistance in terms of Pc-genes described was compiled by SIMONS ET AL. (1978). This catalog lists 61 genes for crown rust resistance. Most of those reported since the early 1960's have been carried by *Avena sterilis*. 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 supposed higher than 90 (CHONG and BROWN, 1996).

In Europe, in the recent study (ŠEBESTA ET AL., 1997) there were considerable differences in the disease resistance index (ŠEBESTA ET AL., 1995) to crown rust among oat lines, ranging from 13 to over 180. The highest resistance index (DRI) was found mainly in those lines that had highly effective resistance in the seedling stage. The most resistant lines were Pc 68, Pc 58, Rodney E, Pc 50-2, Pc 59 and Pc 39, all of which had the disease resistance index (DRI) over 170 or higher. Virulence phenotypes of *P. coronata* f. sp. *avenae* with different combinations of virulence on resistance lines, were identified in twelve European countries and Israel. The resistance genes Pc 39, Pc 55, Pc 58 and Pc 68 were effective against all pathotypes. Genes such as Pc 48, Pc 50-2, Pc 50-4, Pc 54-1 and Pc 59, however, are of importance for the European crown rust resistance breeding of oat as well.

Stem rust

Characterization of the pathogen

Stem rust (*Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn.) is in oat potentially still more damaging disease than crown rust (ŠEBESTA, 1971). It affects all above-ground parts of oat plant, leaves, sheaths, stems and panicles (Figures 2. 11 and 12). The pustules of stem rust are dark redbrown in colour and elongate. The urediospores of stem rust are elliptical, about 20 x 30 µm in size (HARDER and HABER, 1992). Epidemics of stem rust in Europe are incited by *P. graminis* f. sp. *avenae* inoculum that comes either from wild oat species, other host grasses and volunteers, especially in the south or from *Berberis vulgaris* bushes. Like in other cereal rust, the transport of urediospores is supposed from a long distance, south-eastern areas of the Continent (ZADOKS, 1965, ŠEBESTA and BARTOS, 1969).

Effects of stem rust on quantity and quality of oat yield

As mentioned, the stem rust is more destructive pathogen than crown rust (Tables 1–9). Significant differences in harmfulness were found also between different races (ŠEBESTA, 1971, 1974, ŠEBESTA and SÝKORA, 1974, ŠEBESTA ET AL., 1972). The reduction of grain yield on susceptible cultivar resulting from the effect of stem rust varied between 27–29%. The 1000-grain weight was decreased by 25–29%. Percentage of hulls was increased by stem rust by 30–39%.

Crude protein and bound amino acids content is expressively reduced in oat kernels by both rusts, especially by stem rust. Stem rust decreased protein content by 25%, crown rust by 18%. Stem rust like crown rust decreased most the content of basic amino acids histidine, less the content of arginine and lysine. The content of phenylalanine was sharply decreased. The amount of aspartic acid, cystine, tyrosine and isoleucine was considerably decreased especially by stem rust. Stem rust race 6F was demonstrated to be more harmful than the race 1 (ŠEBESTA and SYKORA, 1974). To the lowered content of crude proteins corresponds especially the reduction of histidine. Race 6F of stem rust lowered it by 56% and race 1 by 40%.

As demonstrated, both stem rust and crown rust influence expressively the composition of kernels and straw of oat as well. The content of nitrogen substances and the amount of starch units was significantly decreased in the kernels. The fibre content is increased (ŠEBESTA and SYKORA, 1974).

Incidence of oat stem rust in Europe between 1988 and 1996 as recorded at some localities of the EODN

The incidence of oat stem rust in the EODN trial sites between 1988 and 1996 is shown in Table 39.

In Austria, stem rust was recorded at high or moderate levels at Vienna in all years between 1988 and 1991 and, similarly, at Drauhofen and St. Donat in 1992, 1993 and 1996. At Sadovo, Bulgaria, the pathogen was recorded at a moderate level in 1992 and 1994 and a low level in 1995 and at a moderate level at Rousse in 1996.

In the Czech Republic, stem rust occurred at a high level every third or fourth year. Inoculum has been shown to be transported from south-east Europe along the valley of the Danube by suitable winds (ŠEBESTA, 1973 a, 1993). At Bystrice n. P. a moderate level of stem rust was recorded in 1991, at Kromeriz weak and moderate levels in 1995 and 1996, respectively, and at Krukanice after artificial inoculations high or moderate levels occurred between 1990 and 1994, and in 1996 (Table 39).

At Jógeva, Estonia, high or moderate levels of stem rust were recorded in 1995 and 1996 respectively. Stem rust is probably more frequent in Italy. Moderate levels were recorded in

1990 at Badia-Polesine and at high or moderate levels in 1992 and between 1994 and 1996 at Rome.

At Wielopole, Poland, the disease was at a high level in 1988 and 1990 and similarly at St. Petersburg, Russia, in 1995 and 1996.

Moderate or high levels were also recorded at Pstrusa, Slovakia, in 1989 and 1994, respectively, and similarly in Spain (Madrid locality) in 1988 and 1992. At Kragujevac, Yugoslavia, stem rust was recorded nearly annually at a high or moderate level, especially under artificial inoculation.

Surprisingly a severe infection of oat stem rust occurred, late in the season, at Aberystwyth (Great Britain) in 1992 where it was previously only seen in trace quantities (RODERICK ET AL., 1994).

Disease resistance index (DRI) of oat genotypes to stem rust in the EODN trials, 1988–1996

The DRI values range from 139 for the resistant cv. Rodney M to 4 for the susceptible cv. Pan. A high DRI value is mainly an expression of the major gene resistance. The line Rodney M (DRI = 139) carries the major resistance gene Pg 13, cv. Garland (DRI = 131) carries the genes Pg 2 and Pg 4 (ŠEBESTA, 1977 b) and line Pg a has 3 recessive resistance genes (ERPELDING and MCMULLEN, 1987 pers. comm.). Resistance gene(s) were also effective in other oats with high DRI such as OA 504-5, KR 288/73L/569, Rodney ABDH (Pg 1, 2, 4, 9), KR 3813/73 and many other ones (Table 40).

Virulence gene spectrum of stem rust in Europe, 1989–1996

Uredial samples of *P. graminis* f. sp. *avenae* from Austria (A), Bulgaria (BG), the former Czechoslovakia (CS) (1989–1992), Czech Republic (CZ) (1993–1996), Estonia (EE), Finland (SF), Germany (D), Great Britain (GB), Italy (I), Poland (P), Russia (RU), Slovakia (SK), Spain (E), Sweden (S) and Yugoslavia (YU) were analysed for their virulence in relation to Pg lines and some other sources of stem rust resistance. In addition a small number of samples from Israel (IL) (Tables 41 and 42) were also included in the analysis.

Austria

Between 1992 and 1996, 52 uredial samples were isolated, 18 of which were obtained in 1992. There appeared to be 5 groups of virulence combinations and 3 single combinations in 1992 samples. The first group included the virulence combination based on Pg 1, 2, 3 & 8, which occurred 4 times. In addition, virulences to Pg 12, 15 & 16 occurred in pathotypes of this group. Virulences to Pg 1, 8 & 16 were common to those in the second group; in addition, two pathotypes belonging to this group differed from each other by virulence on Pg 15. Four isolates were in the third group based on virulences to Pg 2, 3 & 16. In addition, virulences on Pg 4, 8, 12 & 15 occurred in individual isolates of this group. The fourth group included two isolates virulent on Pg 2 & 4, in addition, one was virulent on Pg 9, 12 & 16, while the other to V 1. A further group, isolated four times, were all virulent on Pg 3 & 9. In addition, one isolate was virulent on Pg 12 & 15, one on Pg 12, 16 & V 1, and one on Pg 1, 8, & 16. Single isolates virulent on Pg 1, 2 & 4 and Pg 2, 4, 9 & 16 were also found.

In 1993, 15 isolates were identified. Six were virulent only on Pg 2, while three were virulent on Pg 2 & 3. Two isolates were virulent on Pg 1, 2, 8 & 16, two on Pg 1, 2, 4 & 8 (9, 12) and finally another two on Pg 4 & 9 (2, 3, 9, V 1).

In 1994, 17 isolates were identified. Nine were virulent on Pg 3 & 9, three of which were also virulent on Pg 15 and one on Pg 15 & 16. In addition, virulence combinations Pg 8, 9, 15,

16 & V 1; Pg 2, 4, 15 & 16 and Pg 2, 4 & V 1 were isolated. Two isolates were virulent on Pg 9 only while single isolates were virulent on Pg 2 and the combination Pg 9 & 12.

Two virulence combinations, namely Pg 1 & 2 and Pg 1, 2, 3 & 12 were identified in Austria in 1996.

Bulgaria

Only single samples were obtained from Bulgaria in 1990, 1993 and 1996. Three different virulence combinations were identified, namely Pg 1, 2, 3, 4 & 8; Pg 1, 2, 3 & 8 and Pg 3 & 15 respectively.

Czechoslovakia

Thirty-three isolates were obtained from the former Czechoslovakia between 1989 and 1992.

With six occurrences the virulence combination Pg 1, 2, 3 & 8 appeared to be more frequent in this period. Moreover, six other pathotypes with this combination in association with other virulences were identified. A pathotype with virulence on Pg 1, 2, 3, 8, 15 & V 2 was isolated twice. Furthermore, pathotypes Pg 1, 2, 3, 8 & V 2; Pg 1, 2, 3, 8 & 15; Pg 1, 2, 3, 8, 12 & 15 and Pg 1, 2, 3, 8, 12 & 16 and a were identified once. A pathotype with virulence on Pg 1 & 2 was identified three times.

The virulence combination Pg 1, 2, 3, 4, 8, 12 & V 1 was isolated twice, in 1990 and 1992. The combination Pg 1, 2, 3, 4 & 8 was also common to the following pathotypes Pg 1, 2, 3, 4, 8 & V 1; Pg 1, 2, 3, 4, 8, 12, 16, a, V 1 & V 2; Pg 1, 2, 3, 4, 8, 9, 12, 15, 16, a & V 1; Pg 1, 2, 3, 4, 8, 9, 12, 15, 16, a, V 1 & V 2; Pg 1, 2, 3, 4, 8, 12, 15, 16, a, V 1 & V 2 and Pg 1, 2, 3, 4, 8, 12, 15, 16 & V 2. Virulence to Pg a occurred for the first time in Czechoslovakia in 1991 in four populations (samples 38–91, 57–91, 68–91 and 69–91). The incidence of the virulence combination Pg 3, 9, 15 is interesting since it also occurred in Austria in 1994. Virulence to Pg 1 and 3 appeared three times (Pg 1, 3; Pg 1, 3, 8 & 16 and Pg 1, 3 & 8). The virulence combinations Pg 1, 2, 8, 15 & 16; Pg 2, 3 & 12; Pg 2, 3 & 12 and Pg 2, 3, 4, 9, 15, V 1 & V 2 each occurred once. The sample 86–92 was virulent only on Pg 1.

Czech Republic

Between 1993 and 1996 the virulence combination Pg 1, 2, 3 & 8 occurred either by itself (72–93) or in combination with others (Pg 12 & V 1 and Pg 9, 12, 15 & 16).

The virulence to Pg a occurred during this period in three pathotypes, with virulence on Pg 1, 2, 3, 4, 8, 9, 12, 15, a, V 1 & V 2; Pg 1, 2, 3, 12, 16 & a and Pg 1, 2, 3, 9, 12, 16 & a. Moreover, the virulence on Saia occurred for the first time in two pathotypes with the combinations Pg 1, 15, 16, Sa, V 1 & V 2 and Pg 1, 15, 16, Sa & V 2.

The other pathotypes, isolated in Czechia between 1993 and 1996, possessed two, three, four or five virulence combinations (viz. Pg 2 & 4; Pg 2 & 16; Pg 1 & 3; Pg 1, 2 & 8; Pg 2, 4 & 16; Pg 1, 2 & 12; Pg 9, 12 & 15; Pg 3, 9 & 12; Pg 1, 2, 4 & 8 and Pg 1, 2, 3, 12 & 15, respectively).

Estonia

In 1993 two pathotypes were isolated. One was virulent on Pg 1 & 8 and the other on Pg 1, 2, 3, 4, 8, 9, 13, 15, V1 & V2. The latter is of particular significance since it is the first report of virulence on Pg 13 in Europe.

Finland

Five virulence combinations were identified in 1995. Each possessed virulence to Pg a, four of these were also virulent on Pg 12.

Germany

Four virulence combinations were identified from samples collected in 1992. Their virulence combinations were as follows: Pg 1 & 3; Pg 1, 2, 3, 8, 12 & 16; Pg 1, 2, 3, 4, 8, 12 & 16 and Pg 1, 2, 3, 4, 8, 9, 12 & 16.

Great Britain

Three virulence combinations were identified in samples collected at Aberystwyth in 1992. All three had different virulence combinations (Pg 1 & 3; Pg 1, 2, 3, 8 & 12 and Pg 3, 9, 12 & 15.

Israel

Two virulence combinations were isolated from samples collected in 1993 and 1995 (Table 42), these were Pg 3, 9, 12 & 15 and Pg 1, 3, 15 & 16.

Italy

Twenty-three virulence combinations were identified between 1992 and 1996. These varied from zero (4–94/1), one (Pg 3 and Pg 9), two (Pg 3 & 9, Pg 9 & 12), three (Pg 3, 9 & 12; Pg 3, 9 & 15; Pg 1, 2 & 3), four (Pg 1, 3, 8 & 9; Pg 1, 3, 9 & 12; Pg 2, 3, 9 & 15; Pg 3, 9, 15 & V 1; Pg 3, 9, 12 & 15), five (Pg 3, 9, 12, 15 & 16; Pg 1, 2, 8, 9 & Sa; Pg 1, 2, 3, 12 & 16), six (Pg 3, 9, 12, 15, 19 & a; Pg 3, 4, 9, 12, 15 & 16), seven (Pg 1, 2, 3, 4, 8, 12 & a), eight (Pg 1, 2, 3, 4, 8, 9, 12 & V 1) to nine (Pg 1, 2, 3, 8, 9, 12, 15, 16 & a).

The virulence combination Pg 3, 9 & 15 appears to be more frequent. Virulence to Pg a (45–92/1, 45–92/2) and cv. Saia (24–96) are especially noteworthy.

Poland

The four samples comprised four different virulence combinations. The sample 76–93 was virulent on Pg 3 and Pg 9, the sample 77–93 was virulent on Pg 1, 2, 3, 4, 8, 9, 12 & V 1, the sample 115–96 overcame resistance genes Pg 1, 2, 3 & 9 and the sample 115–96/2 was virulent on Pg 1, 2, 3, 12 & 16.

Russia

Three virulence combinations were identified from the three samples received. The pathotype isolated in 1993 was virulent on Pg 1, 2, 3, 4, 8, 9 & V 1, and the two 1995 samples were virulent on Pg 1, 8, 9 & 12 and Pg 1, 2, 3, 4, 8, 9 & 12. As can be seen from the Table 3 each of the pathotypes was virulent on Pg 1, 8 & 9, with a close similarity between samples 130–93 and 90–95/1.

Slovakia

Six virulence combinations were identified, in 1993 and 1996, these being Pg 9; Pg 1 & 2; Pg 3 & 9; Pg 1, 2 & 12; Pg 1, 2, 3 & 12 and Pg 1, 2, 3, 8 & 16.

Spain

Eight samples were received in 1992. Virulence to Pg 1, 2, 3 & 8 was common to five samples. The occurrence of virulence to Pg a in samples 73–92, albeit with a reduced number of differentials, is noteworthy, and in sample 73–92/1. The sample 72–92 was virulent only on Pg 1 and sample 76–92 was not virulent on any of the differentials but again, unfortunately, on a reduced number of genotypes.

Sweden

Seven samples were received in 1993, 1995 and 1996. The sample 117–93 was virulent on Pg 1, 3, 4 & 8, and the 116–93 on Pg 1, 2, 3, 8, 12 & 16. A large spectrum of virulence was found in two isolates, namely 116–93/1 being virulent on Pg 1, 2, 4, 8, 9, 12, 15, a, V 1 & V 2

and 116–93/2 being virulent on Pg 1, 2, 3, 4, 8, 9, 12, 15, 16, a, V 1 & V 2. Both samples overcame Pg 4, Pg 9 and Pg a. The sample 89–95/1 – 1 was virulent on Pg 8, 9, 12 & 15. The samples 127–96 and 127–96/3 were virulent on V 1 & V 2 and Pg 3, 9, 12 & 15, respectively. Virulence to the resistance genes Pg 4, Pg 9 and Pg a are of importance since they are currently deployed in European cultivars and contained in advanced lines of oat.

Yugoslavia

Between 1992 and 1996 twenty-eight samples of oat stem rust were identified from Yugoslavia (now Serbia). The number of virulences varied from one (Pg 2, Pg 3 and Pg 9), two (Pg 3 & 9; Pg 3 & 12), three (Pg 1, 2 & 3; Pg 1, 16 & V 1; Pg 3, 9 & 15; Pg 3, 9 & V 2), four (Pg 3, 9, 12 & 15; Pg 3, 9, 12 & 16; Pg 8, 9, 12 & 16), five (Pg 1, 3, 9, 12 & 15; Pg 3, 9, 12, 15 & V 2; Pg 9, 12, 15, V 1 & V 2; Pg 3, 9, 15, 16 & V 2; Pg 2, 8, 9, 12 & 16; Pg 3, 8, 9, 12 & 15), six (Pg 3, 9, 12, 15, 16 & V 2; Pg 3, 4, 9, 12, 15 & V 2; Pg 1, 2, 3, 4, 9 & 12) up to seven (Pg 3, 4, 9, 12, 15, 16 & V 2). The pathotypes Pg 3 and Pg 9 occurred twice, while the combinations Pg 3, 9 & 15 and Pg 3, 9, 12 & 15 occurred three times.

It is interesting that the frequency of the virulence on Pg 9 is high in samples from Yugoslavia, occurring in 22 of the 28 samples. On the other hand, the virulence to Pg 4 was found only three times.

Effectiveness of stem rust resistance genes and some other resistance sources against *Puccinia graminis* f. sp. *avenae* in Europe in 1989–1996

Austria

Between 1992 and 1996 the highest effectiveness (100%) against oat stem rust populations was by Pg 13 (Rodney M) and Pg a. Those where over fifty percent of the samples were effective were resistance genes Pg 1 (Rodney D), Pg 4 (Rodney B), Pg 8 (Ea^2 x CAV 4023), Pg 9 (Rodney H), Pg 15 and Pg 16. The genes carried by cv. Saia and accessions of *A. sterilis* VIR 343–1 (V 1) and 343–2 (V 2) are of potential importance. Genes Pg 2 (Rodney A) and Pg 3 (Jostrain) seem to be less effective, especially in isolation. (Table 43).

Bulgaria

Resistance genes Pg 9, Pg 12, Pg 13, Pg 16 and Pg a were the most effective. The genes contained in the cv. Saia and the *A. sterilis* accessions VIR 343-1 (V 1) and VIR 343–2 (V 2) were highly effective as well. Furthermore, genes Pg 4 (Rodney B) and the Pg 15 were effective in over fifty percent of the samples. However, this data should be considered as tentative. More samples would give a better picture on effectiveness of individual genes in this country. (Table 43).

Czechoslovakia

In the former Czechoslovakia only Pg 13 and cv. Saia were very effective between 1989 and 1992. Pg 4 (Rodney B), Pg 9 (Rodney H), Pg 12 (Kyto), Pg 15, Pg 16 and Pg a were effective in over fifty percent of the samples. Potential sources of oat stem rust resistance are the *Avena sterilis* accessions VIR 343-1 (V 1) and the VIR 343-2 (V 2) (Table 43).

Czech Republic

In the newly formed the Czech Republic only Pg 13 (Rodney M) was highly effective against stem rust between 1993 and 1996. Pg 4 (Rodney B) (78%), Pg 9 (Rodney H) (72%), Pg 15 (67%), Pg 16 (61%), Pg a (83%) weer effective in over fifty percent of the samples. Virulence on the latter was the first record for the Czech Republic.

The *A. sterilis* accessions VIR 343-1 (V 1) and the VIR 343-2 (V 2) were very effective and might be considered as prospective sources of oat stem rust resistance genes in this country.

Estonia

Only two samples were obtained from this country, but Pg 12 (Kyto) (seedling resistance), Pg 16, Pg a and the cv. Saia were highly effective. As stated previously the breakdown of the Pg 13 (Rodney M) resistance was noteworthy.

Finland

The highest effectiveness in 1995 were on genes Pg 2, Pg 3, Pg 4, Pg 8, Pg 13, cv. Saia and the *A. sterilis* accessions VIR 343-1 and VIR 343-2. Also with relatively high effectiveness were Pg 1, Pg 9, Pg 15 and the gene Pg 16.

Germany

In the Bavarian region of Germany in 1992, Pg 13, Pg 15, Pg a and cv. Saia were highly effective, and to a lesser extent also the accessions of *A. sterilis*, VIR 343-1 and VIR 343-2. With 75% effectivenes Pg 9 and Pg 4 with 50%.

Great Britain

Stem rust is not considered an important disease in Great Britain so that there is no selection for resistance in oat breeding programmes. Most effective were Pg 4, Pg 13, Pg 16, Pg a and the gene in cv. Saia. The *A. sterilis* accessions VIR 343-1 and VIR 343-2 were also very effective. In half the samples Pg 2, Pg 9 and Pg 15 were effective.

Israel

Most effective were Pg 2, 4, 8, 13 and Pg a along with cv. Saia and the *A. sterilis* accessions VIR 343-1 (V 1) and VIR 343-2 (V 2).

Italy

Between 1992 and 1996 the gene Pg 13 was the most effective, and to a lesser extent Pg a and the gene in cv. Saia. Higher than 50% effectiveness was obtained by genes Pg 1, Pg 2, Pg 4, Pg 8, Pg 12 (seedling resistance), Pg 15 and Pg 16. As to *A. sterilis* accessions, the line VIR 343-1 was very effective and the line VIR 343-1 showed 96 percent effectiveness.

It appears that the spectrum of virulences in the oat stem rust populations of Italy and Yugoslavia are similar. (Table 43).

Poland

The resistances Pg 13, Pg a and the cv. Saia were very effective in 1993. Furthermore, Pg 4 and the gene Pg 16 were effective in over 50% of the samples. *A. sterilis*, VIR 343-2 (V 2) was highly effective, the line VIR 343-1 (V 1) showed 75% effectiveness. (Table 43).

Russia

Between 1993 and 1995 genes Pg 13, Pg 15, Pg 16, Pg a, cv. Saia and *A. sterilis*, VIR 343-2 (V 2) were very effective. The line VIR 343-1 (V 1) showed 67% effectiveness.

Slovakia

Between 1993 and 1996 genes Pg 4, Pg 13, Pg 15, Pg a and the cv. Saia were very effective. Pg 3 (Jostrain), Pg 9 (Rodney H), Pg 12 (Kyto) (seedling resistance) and Pg 16 were effective in 50% of cases. Both *A. sterilis* VIR 343-1 and VIR 343-2 were highly effective as well.

Spain

In 1992, Pg 9 and Pg 13 (Rodney M) and cv. Saia were very effective. Pg 4 (Rodney B), Pg 12 (Kyto) (seedling resistance), Pg 15 and Pg a were also moderately effective. Both VIR 343-1 and 343-2 were resistant to all isolates as well.

Sweden

In 1993, 1995 and 1996, only Pg 13 (Rodney M) and the cv. Saia were effective against all the isolates. Nevertheless Pg 4 (Rodney B), Pg 16 and Pg a were moderately effective. Surprisingly, VIR 343-1 (V 1) and VIR 343-2 (V 2) were only effective against 57 percent of the isolates. (Table 43).

Yugoslavia

Between 1992 and 1996, Pg 13, Pg a and the cv. Saia were the most effective resistances. Pg 1 (Rodney D), Pg 2 (Rodney A), Pg 4 (Rodney B), Pg 8 (Ea^2 x CAV 4023) and the gene Pg 16 were also relatively highly effective. The effectiveness of lines, VIR 343 1 (V 1) and VIR 343-2 (V 2) was 93 and 75%, respectively.

As mentioned previously there is some resemblance in the virulence gene spectrum in between Italy and Yugoslavia and, therefore, also in the effectiveness of the Pg-resistance genes. (Table 43).

Average effectiveness of Pg-resistance genes and the other sources of stem rust resistance in Europe in 1989–1996

The Rodney M line (Pg 13) was the most effective resistance (96.7%) and was overcome only once by an Estonian stem rust isolate. This was followed by Pg a (87.0%), Pg 16 (75.7%), Rodney B (Pg 4) (72.5%), Pg 15 (72.3%), Rodney H (Pg 9) (57.0%), Kyto (Pg 12) (53.4%), Rodney A (Pg 2) (46.9%), CAV 4023 (Pg 8) (34.7%), Rodney D (Pg 1) (33. 7%) and the cv. Jostrain (Pg 3) (33.7%). The effectiveness of the cv. Saia *(A. strigosa)* was 99.0% and 85.7% and 89.2%, respectively of the *A. sterilis* accessions WIR 343-1 and WIR 343-2 were effective (Figs. 21–25).

Comparisons with the situation in the foregoing periods and elsewhere in the world

Virulence spectrum of oat stem rust in Europe in the last decades

The virulence spectrum of oat stem rust populations and the effectiveness of resistance genes has changed considerably during the last decades, at least in central Europe. In the first studies on stem rust resistance, in 1965 and 1966 in the former Czechoslovakia (ŠEBESTA, 1969 a, b), a number of oat lines or cultivars were resistant to races 2, 3, 4 and 6, represented by e. g. Ag 313, C. I. 7145, Borne TT, Dodge, Garland, Garry, Putnam 61, Rodney (B), cv. Saia and many others. These oats also showed a high level of resistance to stem rust at the adult plant stage (ŠEBESTA, 1970 b).

In 1967 and 1968, races 2, 3, 4 and 6 were isolated again and races 1 and 8 were found for the first time in the former Czechoslovakia and race 6 was predominant. The gene Pg 4 remained effective against all races of *P. graminis* f. sp. *avenae*. It was shown that only some of the genes conferring stem rust resistance in cv. Minrus was transferred to the line Rodney D (Pg 1). Cv. Minrus was resistant to race 1, whereas the line Rodney D, like C. I. 3034 (seedling stage), was susceptible. Genetic analyses of the cross between cv. Minrus and Rodney D indicated that the former contains three additional complementary genes to which the gene Pg 1 is epistatic. F_2 seedling segregations for stem rust resistance were 45:19 and F_3 7:38 :19 (ŠEBESTA, 1980).

Studies also showed that the high virulence of some races of *P. graminis* f. sp. *avenae* might be associated with high aggressiveness and low virulence associated with low aggressiveness (ŠEBESTA, 1973 c).

The discovery of race 22 (8A) (ŠEBESTA and ZWATZ, 1977) caused a radical change in effectiveness of the resistance genes used in central European oat breeding programmes at the

time. A race similar or identical to race 22 was found for the first time in Europe in Sweden (LEIJERSTAM 1964). Race 8A was also found in Rumania (STEWART, RADULESCU and NEGULESCU (1967).

Between 1965 and 1975 twelve races were identified in Czechoslovakia, Austria and Romania (viz. Races 4, 8, 11, 22, 68, 70, 71, 72, 74, 75, 76, 77). The gene Pg 13 was effective against all these races. Whereas gene Pg 4 was overcome only by race 22 and likewise gene Pg 9 by races 71, 74, 75, 76 and 77 (ŠEBESTA and ZWATZ, 1980).

In the period between 1976 and 1979, in Austria, Czechoslovakia and Poland, fourteen races of oat stem rust were isolated (ŠEBESTA, 1984[a]) with cv. Saia showing the highest effectiveness against these races. The line Rodney M (Pg 13) was overcome by Polish race P 1 which, as found later, also possessed virulence to Pg 16 (ŠEBESTA ET AL., 1985).

European races E 3, E 4, E 5, E 6, E 7 and E 8 appear to correspond to the North American races NA 5, NA 50, NA 23, NA 21, NA 18 and NA 27, respectively (MARTENS ET AL., 1979).

The virulence of those *P. graminis* f. sp. *avenae* races isolated from Austrian, Czech, Slovak, Swiss, German and Polish populations in the period before 1988 showed clearly that the most effective resistance gene was Pg a, with genes Pg 4, 9, 13 and 16 making important contributions as well. The combination of genes Pg 4 + Pg 9 in the line Rodney ABDH (Pg 1 + Pg 2 + Pg 4 + Pg 9) was also very effective (ŠEBESTA ET AL., 1985).

In the period 1988 to 1996 the effectiveness of Pg resistances in Europe has changed dramatically (Table 5). The most resistant line was cv. Saia followed by the Pg 13, Pg a, Pg 16, Pg 4, Pg 15, Pg 9, Pg 12, Pg 2, Pg 8, Pg 1 and Pg 3. The mean effectiveness of the *A. sterilis* accessions VIR 343-1 and VIR 343-2 was 86 and 89%, respectively.

Oat stem rust virulence and the effectiveness of resistances in other parts of the world

In Canada the virulence pattern in *P. graminis* f. sp. *avenae* appears stable (Harder 1988, 1989, 1990, 1994). Common races of oat stem rust have tended to dominate populations for 25 years or longer. Virulence to genes Pg 9 and Pg 13, currently important resistance sources, was relatively common, then declined with the emergence and dominance of race 6AF/C10/NA27 in the 1960. The maintenance of Pg 2 resistance in contemporary Canadian cultivars should reduce the threat to Pg 13 resistance in the prairie region. The frequency of virulence of Pg 15 was very high across Canada but declined in the prairie region along with virulence to Pg 9 and Pg 13. Virulence to Pg 16 has only occurred in one year and virulence to gene Pg a was found once in the study.

Virulence to all of the Pg resistances has occurred at some time in the North American oat stem rust population, like in European populations, regardless of the exposure of the populations to these resistances (HARDER, 1994).

In the USA and Mexico the principal race of oat stem rust was NA 27, virulent on cultivars having resistance genes Pg 1, 2, 3, 4 and 8 (ROELFS, CASPER, LONG and ROBERTS, 1989, 1990, ROELFS, LONG and ROBERTS, 1993 a, b, 1995). This race has also been isolated in Europe as can be seen from the Table 41.

Resistance of Oat Varieties to Stem Rust

Of the 130 genotypes of the world assortment of oat a number of cultivars are resistant to all or to certain of races of stem rust isolated in central Europe (ŠEBESTA, 1969 a). Resistant to races 2, 3, 4 and 6 are the cultivars or lines Ag 313, C.I. 7145, Ag 354, Borne TT, Burnett, C. I. 6537, Canuck C. I. 4024, Dodge, Garland, Garry, C. I. 6662, Minn. II-47-12, Minn. II-47-17, Minn. Oat Sel. 643114, Nodoway, O. g. 313, O. g. 354, Putnam 61, C. I. 7531, Radar 2, Rod-

ney, C. I. 6661, Saia, C. I. 7010, Taggart, C. I. 4652, Torch, C. A. N. 812, Vicar, C. A. N. 827, and the unnamed lines C. I. 4023, C. I. 6666, C. I. 7438, C. I. 7921, C. I. 8040 and C. I. 8153. (Table 44).

The largest range of virulence was that of the race 6, followed by races 4, 3 and 2. Of the tested collection of oats 28 cultivars were resistant to races 6, 32 to race 3 and 69 cultivars were resistant to race 2.

Within the scope of our race spectrum of oat stem rust, resistance to race 2 occurred either separately conditioned by the D gene, or in combination with resistance to race 3 conditioned by the A (Pg 2) gene. In the samples Eagle^2 x C. I. 4023 and Eagle^2 x C. I. 7438 containing the F gene (Pg 8), resistance to race 2 was associated with resistance to race 4. Apart from the already mentioned combination a resistance to race 3 conditioned by the A gene (Pg-2) is further connected with resistance to race 4 in the Jostrain, C. I. 2660 cultivar, in which it is conditioned by the E gene.

Cultivars that were resistant to the race 6 were simultaneously resistant to all those three races. This resistance to all races of oat stem rust identifield in Czechoslovakia in the years 1965 and 1966 (ŠEBESTA, 1969 a) is conditioned by the B gene (Pg 4), which with regard to our race composition of oat stem rust, was the most valuable gene.

In the adult stage 112 oat genotypes were tested to the races 2, 3F, 4 and 6F. Resistance was found in 30 oats, 35 oats showed mesothetic reaction and the remaining cultivars were susceptible.

The average rate of severity varied from 5% (Saia, Sel. 4023) to 35% (Sel. C. I. 1921) in resistant cultivars. 10% severity was in resistant cultivars Ag 313, Minn. II-47-17, O. g. 313, O. g. 354, Sel. C. I. 4023 and C. I. 8153.

As to the mesothetic cultivars, the lowest severity was observed in the following cultivars: Winema (15%), Indio (20%) and Tonka (20%), the susceptible cultivars were attacked only in 20%, this value being found in the cultivars Buck 152, La Prévision, Cleo, Jefferson and Kanota.

In the resistant cultivars Saia, Minn. II-47-12 and Minn. II-47-17 the severity showed a very small range of fluctuation in the three year period. On the other hand, the cultivars Garry, C. I. 6662, Putnam 61, Rodney and some other resistant cultivars had infestation rates of considerably fluctuating values (Table 45).

Inheritance of stem rust resistance in oat

The resistance of the cv. Garry, C. I. 6662 to the central European races of *Puccinia graminis* f.sp. *avenae* like to the North American races, depends on two independent dominant genes Pg-2 (A) and Pg 4 (B). Czech new release 290-1-1 (Garry x Český zlatý) contains dominant gene Pg 4. The release 292-1-3 (HAG x Garry) was a heterogeneous population. Some plants contained both dominant genes, Pg-2 and Pg-4, other ones only dominant gene Pg 4. (Tables 46 and 47).

The Dodge and Garland cultivars were indicated to possess two dominant independent genes for resistance to the central European races of *P. graminis* f. sp. *avenae*. These are apparently the genes Pg-4 and Pg-2 described in these cultivars in previous papers (SHANDS ET AL., 1966 a, b). Neither of these two genes, Pg-4 and Pg-2 is allelic with the gene Pc 39 conferring the total resistance to the European race populations of *P. coronata* f. sp. *avenae* at that time. Therefore, they can be combined in one-line cultivar (Tables 48, 49 and 50).

The oat line Pc 54 contains a gene for crown rust resistance, tranferred from *A. sterilis* accessions CAV 1830 and CAV 1832 that come from Turkey (MCKENZIE and MARTENS, 1976) (cit. MERTENS, 1985). In our, as well as in Canadian experiments, the line Pc 54 was indicated

to possess also a resistance to stem rust. It was found that the gene Pg 15 is responsible for the stem rust resistance in Pc 54 (HARDER, 1985, pers. comm., ŠEBESTA ET AL., 1993). Some slight differences in stem rust reaction are supposed between the line Pg 15 and Pc 54 (Figures 21 and 22) ŠEBESTA ET AL., 1985).

In the accession *Avena sterilis* L., 343-1 was found a new type of oat stem rust resistance. The effectivenes of this stem rust resistance was indicated against many isolates in Europe (Table 51) ŠEBESTA ET AL., 1998).

Genetic analyses of the cross Flamingsonova x *A. sterilis* WYR 343-1 indicate that this resistance to stem rust is conditioned by one dominant gene conferring infection type 1 in some plants and in other plants by one dominant and one recessive gene conferring moderate resistance (infection type 2). (Figures 22–25) (Table 52) ŠEBESTA, 1986, HARDER, 1986, pers. comm.).

Peculiarities in inheritance of stem rust resistance

The cv. Minrus, C. I. 2144 belongs to the most old sources of oat stem rust resistance. The gene D (later designated Pg 1) (SIMONS ET AL., 1978) was described in the cv. White Russian by Garber (1921). Up to now, monogenic resistance was supposed in general (SIMONS ET AL., 1978).

In Canada, in sixties, a set of nearly isogenic lines was produced. The gene D (Pg 1) was transferred into the line Rodney D (GREEN, 1969, pers. comm., MURPHY and COFMAN, 1961). In the majority of tests the line Rodney D indicated identical reaction like the cv. Minrus.

However, some isolates of Austrian and Czechoslovak provenience indicated that the resistance of the cv. Minrus is more effective if compared with the Pg 1 line. If the stem rust isolate was avirulent on Pg 1 than both Minrus and Pg 1 reacted identically. But if an isolate of the pathogen overcomes Pg 1 then the remaining part of the resistance genotype of the cv. Minrus was effective (Figures 26, 28). In such cases the cv. Minrus was resistant and the Pg 1 line moderately resistant (ŠEBESTA, 1980).

Genetic analyses of the cross Minrus x Rodney D indicated that the cv. Minrus differs from the line Rodney D by other three genes to which Pg 1 (D) gene is epistatic (Figure 29). The resistance of the cv. Minrus in case when Pg 1 was overcome, was conditioned by one dominant gene which was complementary either with one or the other of the two other remaing genes. The experimental ratios in F_2 (45 R : 19 S) and F_3 (7 R : 38 Segr. : 19 S) confirmed this hypothesis (Figures 26–29) (Tables 53–55) (ŠEBESTA, 1980).

Approximative comparison of oat rust reaction of the cv. Jostrain and its derivative Rodney E

Comparison of the cv. Jostrain to its derivative, Rodney E line (Pg 3) in relation to six Austrian and two Polish races indicated that their genetic background to stem rust is not identical. There were cases when the cv. Jostrain was resistant and the line Rodney E highly susceptible (Figures 30 and 31) (ŠEBESTA and ZWATZ, 1980).

Strategy of genetic control of oat stem rust

Like in crown rust, the breeding of multigenic cultivars to stem is supposed to be prospective (Figures 45–47). (MCKENZIE ET AL., 1971 – cit. MARTENS, 1985, HARDER, 1994, HARDER and HABER, 1992).

At present, ten major resistance genes for stem rust of oat (Pg-genes) are to disposal to plant breeders (MARTENS, 1985, HARDER, 1994, ŠEBESTA ET AL., 1998).

The dominant thermostable Pg-l gene was extensively used in the USA for many years (STEWART and ROBERTS, 1970) but not in Canada. The average effectiveness of the Pg 1 in Europe in 1989–96 was 34% (ŠEBESTA ET AL., 1998).

The dominant thermostable Pg-2 gene was incorporated into about 13O cultivars in North America thus indicating how widespread and important was this gene in the past (Martens, 1985). The average effectiveness of Pg-2 in Europe in 1989–96 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 1989–96 the average effectiveness of the 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, ŠEBESTA, 1988). The virulence on this type of resistance was indicated to be inherited extrachromosomally (GREEN and MCKENZIE, 1967 – cit. MARTENS, 1985).

Gene Pg 4, dominant and thermolabile, has been used widely and together with Pg 1 and Pg 2 has in the past been the basis for stem rust resistansce breeding in both North America and Europe (MARTENS, 1985, 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 (DYCK, ZILLINSKY, 1962 – cit. MARTENS, 1985), were identified in the diploid species *A. strigosa* Schreb., C. D. 3820, have not yet been possible to transfer this resistance to the hexaploids. These genes may be the same (DYCK, 1966 – cit. MARTENS, 1985).

The recessive and thermolabile gene Pg 8 has not been used in breeding programmes in both North America and Europe. However, it was effective against eastern Australian races, in eastern America and South America (COELHO, 1976 – cit. MARTENS, 1985) and partially in Russia (SUZDALSKAYA ET AL., 1978 – cit. MARTENS, 1985). In Europe in 1989–96 the Pg 8 had effectiveness 35%.

The recessive and thermolabile gene Pg 9 has not been used in breeding programmes until recently (MCKENZIE ET AL., 1976 – cit. MARTENS, 1985). Like the gene Pg 3, the Pg 9 is closely associated with a gene for resistance to *P. coronata* (MCKENZIE ET AL., 1965 – cit. MARTENS, 1985). The tests of segregating populations of cv. Dumont indicated in this cultivar in addition to Pg 2 and Pg 13 the presence of the Pg 9 tightly linked in coupling to a gene Pc X 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 the Pg 9 was 57%.

Incompletely recessive gene Pg 11 conferred adult plant resistance to all races of oat stem rust that have been tested (MCKENZIE and MARTENS, 1968 – cit. MARTENS, 1985). It appears to be in association between the 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.

Recessive gene Pg 13 is one of the most effective stem rust resistance genes available to breeders (ROELFS ET AL., 1982, MARTENS, 1985). Cvs. Fidler and Dumont combining Pg 13 with some other stem rust resistance genes were developed by MCKENZIE ET AL. (1981, 1984 – cit. MARTENS, 1985).

The effectiveness of the Pg 13 in Europe during 1989–96 was 97%. Just a virulent pathotype on Pg 13 in Estonia occurred (ŠEBESTA ET AL., 1998).

Gene Pg 14 is a partially dominant gene isolated by McKEY and MATTSSON (1972 – cit. MARTENS 1985) from Milford, C. I. 5039, Winter Turf, C. I. 1570 and other lines. It is difficult to identify a putative origin for this gene.

Gene Pg 15 is a partially dominant gene isolated from *A. sterilis* (MARTENS, 1985) collected east of Uskudar on the Black Sea near Istanbul, Turkey. Races avirulent on Pg 9 were also avirulent on Pg 15 in the Great Plains region (MARTENS, 1985).This gene has not yet been used in commercial cultivars in North America and Europe. The average effectiveness of this gene in Europe was 72% (Figures 21 and 22) (ŠEBESTA ET AL., 1998).

Gene Pg 16 (MARTENS, 1985) is a highly effective gene from tetraploid *A. barbata* oat that may be successfully used in stem rust resistance breeding. No 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 the average effectiveness of the Pg 16 was 76% (ŠEBESTA ET AL., 1998).

A dominant gene for stem rust resistance, designated Pg 17, was isolated by HARDER ET AL. in 1990 from a Spanish *A. sterilis* IB 3056 accession wild oat. It is effective only in adult plant stage.

Pg 12 is a recessive gene that was isolated from Kyto, a cultivar introduced from Yugoslavia via Finland by the USDA in 1939 (MARTENS, 1985). The cv. Osmo expressed a rust reaction similar to that of Kyto (GREEN and McKENZIE, 1964 – cit. MARTENS, 1985). In Europe the effectiveness of Pg 12 in seedling stage was 53%.

The Pg a complex (MARTENS, ET AL. 1981 – cit. MARTENS, 1985) consists of a gene Pg 12 and interacting genes. ERPELDING and McMULLEN (pers. comm.) identified in Pg a complex 3 recessive genes. The Pg a complex was found to be very effective in the USA, Canada and Europe (ROELFS ET AL., 1989, l990, 1995).

In Europe the Pg a genes were effective of 87% (ŠEBESTA ET AL., 1998).

The early development of teliospore formation – a possible component of the genetic control of cereal rusts?

On cereals and other grasses two spore stages of rusts can occur, the uredial and the telial ones, both being dikaryotic. Urediospores are mainly responsible for epidemic development. Urediospore formation is maintained as long as green plants are available. At cereal ripening rusts cease to produce urediospores and begin to produce teliospores.

However, in glasshouse experiments cereal rusts populations are found that form teliospores even in the seedling stage. Such an early development of teliospores , in short EDT, observed also on adult plants, is interesting both from the epidemiological and genetic point of view. The role of the early teliospore formation, as a limiting factor in epidemic development, was often quoted by Professor STAKMAN (WAHL, pers. comm.).

Recently, an accession of *Avena sterilis* L. CAV 2648 was found from which teliospore formation in crown rust (*Puccinia coronata*) was transferred to various degrees simultaneously with specific resistance genes (ŠEBESTA ET AL., 1987). The EDT-syndrome seemed to be polygenically controlled in this host plant. Two lines with moderate resistance differing in teliospore formation in the seedling stage were isolated thus indicating possible accumulation or erosion of genes for early teliospore formation in this host plant. (Figures 33–40).

As indicated, the importance of host resistance in the EDT-phenomenon, reported earlier by PARKER (1918) (cit. ŠEBESTA, BAYER, 1992) and MURPHY (1935)(cit. ŠEBESTA, BAYER, 1992), was found in our experiments. In addition, our results also indicate that the EDT-phenomenon occurs and is effective on both resistant and susceptible cultivars and, furthermore, is detectable in both the seedling and adult plant stages. However,it seems that on susceptible cultivars teliospore formation is delayed in comparison with that on resistant cultivars.

Our results confirmed the role of race in EDT and are in accordance with those achieved by BAILEY (1925)(cit. ŠEBESTA, BAYER, 1992), JOHNSON (1931), FRENZEL (1930) (cit. ŠEBESTA, BAYER, 1992), ZIMMER and SCHAFER (1961) (cit. ŠEBESTA, BAYER, 1992) and by HASSEBRAUK (1965)(cit. ŠEBESTA, BAYER, 1992). EDT seems to be a result of the specific relationship between the cultivar and the isolate of the fungus and, moreover, is considerably affected by external factors. Furthermore, our observations of wheat stem rust and oat crown rust support the conclusion, drawn by ZIMMER and SCHAFER (1961) (cit. ŠEBESTA, BAYER, 1992), that in cereal rusts there occur populations that produce urediospores till the last stage of maturity of the host plant. According to ZIMMER and SCHAFER (1961) the rapidity of teliospore formation is independent of the reaction type of host cultivar but it does seem to be dependent on the rust race and host-parasite combination.

Early teliospore formation could be included in the genetic control of those cereal rust populations in which it functions. If present at a high level in donors of rust resistance, it could then be used in breeding programmes. Selection for this trait might be carried out in the field or even in seedling tests. However, more research on both pathogen – host relationships and the effect of external factors on the EDT-process is needed.

The mechanism(s) responsible for EDT are not yet known. If recognized and understood, the EDT-phenomenon might be induced in the host artificially and thus significantly contribute to the control of cereal rusts.

Powdery mildew

The characterization of the pathogen and its role in the history of oat cultivation

JONES & GRIFFITHS (1952) examined the susceptibility of a range of oat cultivars to powdery mildew and although some showed a degree of resistance, only Cc4146, a natural hybrid between *A. sativa* L. x *A. ludoviciana* Dur. (= *A. sterilis* L.) and the diploid species A. *hirtula* Lag. and *A. strigosa* Schreb. were highly resistant. HAYES & CATLING (1963) identified three races of *E. graminis* f. sp. *avenae* using five differential host genotypes and later HAYES & JONES (1966) identified another two races. The United Kingdom Cereal Pathogen Virulence Survey (UKCPVS) was initiated in 1967 (CHAMBERLAIN, CLOTHIER & WOLFE, 1972) using the same differential genotypes for characterizing oat mildew isolates. Oats are categorised according to their resistance characteristics (OMR groups, Table 57) and the corresponding virulences of the mildew isolates as OMV groups (JONES & JONES, 1979). At the same time *A. hirtula* (Cc3678) was substituted as a differential cultivar by the more relevant hexaploid translocation line Cc6490 of *A. barbata* Pott. ex Link (OMR 4).

The line 7718Cn (OMR 5), derived from Cc4146 (OMR 2), was added to the UK set of differentials because it gave a different reaction to Cc4146 with some isolates (JONES, 1982). This suggests that Cc4146 probably contains more than one resistance gene with only some transferred to 7718Cn.

The UK survey results have been published annually by the UKCPVS, summarised by RODERICK, JONES & CLOTHIER (1995) and presented up to 1994 in this chapter.

Mildew samples collected at some EODN nurseries were also tested at the Research Institute of Crop Production, Prague (ŠEBESTA ET AL., 1985, 1987 & 1991). Virulence to OMR 1 was present in the former Czechoslovakia in 1979 when surveys began, and to OMR 2 from 1982. The OMR 3 resistance, present in cv. Mostyn, remained effective until 1987 (ŠEBESTA ET AL., 1987). The OMR 4 resistance has remained effective in Europe, outside the UK. CORAZZA & BOZZINI (1995) found that in Italy cvs Manoire and Weibull were resistant to mildew but the source of this resistance was not identified.

The effect of powdery mildew on grain yield of oat

Over most of its range in Europe the oat crop occupies a relatively small area of land in comparison with wheat and barley and there is relatively less information available on the frequency of oat mildew and crop losses. LAWES, VALENTINE & JONES (1983) reported that the annual grain yield loss to powdery mildew in the United Kingdom was probably in the region of 5–10% making it the most important foliar disease of this crop. Mildew was more frequent and more severe on spring-sown than autumn-sown crops at trial sites in the UK between 1957 and 1976 and the frequency of oat mildew was significantly correlated with rainfall and temperature between November and January (PRIESTLEY & BAYLES, 1979).

The effect of mildew on grain yield has been reported by a number of workers. Losses of 39% were reported by LAWES & HAYES (1965) between resistant and susceptible near-isogenic lines. Similarly JONES (1977) reported a reduction of 20% in the susceptible cv. Sun II, whereas with the moderately resistant cv. Maldwyn there was only a 9% reduction. JONES ET AL., (1987) looked at different fungicide treatments on mildew control and grain yield over three years, thus with the susceptible cv. Selma grain yield losses varied between 9.8% and 32.4% respectively in years of low and high disease. The proportion of grain yield to total biomass (harvest index) was also reduced. More detailed analysis showed that the reductions in grain yield were mainly due to reductions in the number of fertile panicles and thousand grain weights (RODERICK & JONES, 1988). There was also a negative correlation between disease severity and percentage grain protein content and specific weight.

In the former Czechoslovakia Sebesta (1987) found that mildew in combination with late attack with crown rust (*Puccinia coronata* Cda f. sp. *avenae* Eriks.) reduced thousand grain weight (TGW) in the highly susceptible cvs Pan and Veles by 15 and 20% respectively, whereas in the breeding line KR 3975, which was highly resistant to mildew and moderately resistant to crown rust, the TGW was reduced by only 2%. When mildew was controlled the crown rust reduced the yield by 8 and 13% respectively for the former two cvs (Table 10).

Possible control ways

Cultural measures to reduce the risk of mildew such as earlier planting of spring cereals and earlier application of fertilisers of winter cereals were proposed by LAST (1954). Winter oats can act as a ‚green bridge' to infect spring crops, which will be dependant on such factors as the susceptibility of winter oat cultivars and winter temperatures. In regions of high mildew risk there are two other methods of disease control; firstly, the use of fungicides and secondly, the deployment of resistant cultivars. The use of foliar fungicides on oats in Europe appears to vary considerably, JENKINS & LESCAR (1980) estimated that 26% of the oat area in north-west Europe was treated with foliar applied fungicides during 1979, but this varied between 50% in both the UK and Ireland to less than 5% in Norway, Italy and Switzerland. Unfortunately these figures do not reveal what specific fungicides were being used and therefore it is not possible to say which diseases were being targeted.

Host resistance offers the most economic and environmentally benign method of disease control. Early mildew epidemics were shown to be more damaging in spring barley than late epidemics (CARVER & GRIFFITHS, 1981). Consequently the deployment of sources of resistance which are expressed at these later growth stages (adult plant resistance or APR) could leave these cultivars vulnerable at the earlier growth stages. Possible fungicide application strategies were investigated by JONES ET AL., (1987). They applied fungicide seed dressing along with foliar application to a range of cultivars which differed in their level of susceptibility to mildew and repeated the trial over three years. In years of high incidence of mildew seed treatment combined with APR gave adequate yield control but in low mildew incidence years APR alone was sufficient.

Another possible method of reducing disease levels which has received little attention in the control of oat mildew is the use of cultivar mixtures or multilines. JONES ET AL., (1980) found that the level of mildew infection on mixtures of cultivars with major gene resistances was generally about equal to the mean of their components, whereas the beneficial effect of APR cultivar mixtures was more marked.

The incidence of oat powdery mildew in the EODN, 1988–94

Powdery mildew was a prevelant disease on oats in Europe in the period 1988–94, presumably because of high levels of plant nutrients and the growth of susceptible cultivars. A high incidence of the disease occurred in all years at the Aberystwyth site in the United Kingdom (Table 59), where climatic conditions are favourable to mildew and the disease can over-winter on the autumn-sown crop. Moderate or high levels were recorded in three or more years at Krukanice, Czech Republic; Le Rheu, France; Quedlinberg, Germany; Thessaloniki, Greece and Kragujevac, Yugoslavia. High incidences were also recorded at three Austrian sites, the remaining three German sites, Ulrum, Netherlands and Badia Polesine, Italy.

Effectiveness of mildew resistant genotypes in the EODN

The calculated disease resistance indices for all genotypes in the EODN in the seven year period (1988–94) are shown in Table 60. New accessions or cultivars with potentially novel forms of disease resistance were added to the nursery during this period, consequently there

are some differences between the total number of evaluations per genotype. To overcome this the data was adjusted for a standard number of 50 evaluations.

High values were found in three genotypes with resistances recently introduced from wild oat species, these were APR 166, APR 122 and Cc6490. At Aberystwyth these lines were resistant to mildew at both the seedling and adult plant stage, under both glasshouse and field conditions (HOPPE & KUMMER, 1991; H. W. Roderick, personal observation). In recent tests of F_2 lines at IGER, Aberystwyth, segregation was observed at the seedling stage which suggests that both lines contain a specific resistance detectable at the seedling stage (H. W. Roderick, personal observation). Inheritance studies by the breeders of these lines also showed that this resistance was controlled by a dominant gene but that it was incompletely expressed at the seedling stage and more pronounced as the plant developed (HOPPE & KUMMER, 1991). Glasshouse tests at Groß Lüsewitz, Germany, also showed that the 2nd-formed leaves were moderately resistant but 5th-formed leaves highly resistant (HERRMANN & RODERICK, 1996). These observed differences in expression of resistance at different sites could be due to the conditions under which the plants were tested. The line Cc6490 is effective at all European sites but only in some years at Aberystwyth.

The line Cc4761, cvs. Maelor and Roxton have moderate to high levels of APR and this was expressed in their high indices. Similarly the transgressive lines OM 1621, which combines the major gene from cv. Mostyn with adult plant resistance from cv. Maldwyn, and OM 1387 along with cv Melys, although only recent additions to the nursery, were very effective at all sites. Other genotypes with high DRIs and with effective major genes at some sites were cvs. Mostyn and Orlando. The latter carries the OMR 2 resistance from Cc4146 but is moderately susceptible at Aberystwyth. Whereas the high index of Cc4146 not only reflects the effectiveness of the resistance gene in some regions but also its moderate level of APR. Cv. Manod (OMR 1) has been effective at only a small number of sites since 1991 (in Germany and Thessaloniki in 1991, Spain in 1992 and Yugoslavia in 1994). The relatively low DRI of Pc 54-1 indicates that this re-selection does not carry the mildew resistance present in the original population. Other re-selections Pc 54-2 and Cc7422 (ŠEBESTA ET AL., 1993) have recently been included in the nursery and have been very effective.

Other lines which have not previously been characterized for mildew resistance, such as Pc 39, Pc 58 and Pg 15, were included in the EODN as sources of crown or stem rust resistance but have also shown moderate levels of resistance to mildew.

In general sites where mildew is prevelant in most years the number of resistant genotypes is lower. e.g when comparing Aberystwyth with Madrid or the Austrian sites.

Virulence of oat powdery mildew in Europe

Mildew infected leaves from farm crops or trial sites in the United Kingdom have been collected annually since 1967 as part of the UK Cereal Pathogen Virulence Survey. The reaction of a set of differential genotypes (Table 57) to infection was recorded on second-formed leaves of seedling plants and isolates placed into virulence groups (OMV) based on host reaction types. Samples of mildew infected leaves were also obtained from EODN sites in the Czech Republic in some years and tested at the RICP, Prague, using a similar procedure.

The virulence combinations idenitfied so far in Europe are shown in Table 61. The UK survey results (Fig. 43) show that race 2 carrying virulence to OMR 1 in the spring oat cv. Manod and winter oat cv. Peniarth was the predominant race when the survey began in 1967 Within a few years of the introduction in 1968 of cv. Mostyn, carrying the OMR 3 resistance, the frequency of race 2 declined and race 4 (OMV 1+3) increased. Similarly, the release of cv. Maris Tabard (OMR 2) in 1973 resulted in an increase in race 3 (OMV 1+2). Virulence to the resistances in these two cultivars was detected before they had been released commercially.

The more complex race 5 (OMV 1+2+3), has, with the exception of 1991, had the highest frequency in the last ten years.

Virulence to OMR 4, derived from the *A. barbata,* accession Cc4897, was identified at Aberystwyth in 1978 (JONES & JONES, 1979) and this virulence was discovered in other isolates from other sites in the UK in 1980. Since 1989 the frequency of OMV 4 has increased and in 1991 Race 7 (OMV 1+2+3+4) had the highest frequency despite the absence in cultivation of cultivars with this resistance (JONES & CLIFFORD, 1992). The variable number of samples received each year, from a restricted number of sites, makes it difficult to analyse fully the variation in frequencies of virulences, but they do show that with the limited number of resistance sources deployed corresponding virulence frequencies follow classical gene-for-gene principles also noted by HARDER & HABER (1992). At present none of the cultivars recommended by the National Institute for Agricultural Botany in the UK possess any effective major gene resistances.

In the Czech Republic virulence to OMR 2, in Cc4146, was detected again in the EODN in 1988 and virulence to OMR 3, in cv. Mostyn, occurred at Krukanice in 1990 and at Salzmünde in eastern Germany. FELSENSTEIN ET AL., (1996) have recently shown that there were high levels of virulence to OMR 1, 2 and 3 over much of western and central Europe with some regional variation. For example a low frequency of virulence to OMR 2 was found in northern Germany, but high levels of virulence to OMR 1 and 3, whereas in south-east Germany there was a low frequency of virulence to OMR 3. Similarly there was a very low frequency of virulence to OMR 3 in southern France. The frequency of virulence to OMR 4 has recently been shown to be at a low level in some European countries (HSAM, 1996 pers. comm.).

Donors of race specific resistance and their effectiveness

Sources of race specific resistance are shown in Table 62. Cv. Mostyn, Cc4761 and Cc6490 still appear to be effective on the Continent, at least in some years, from EODN results. The monogenic resistance in *A. sterilis,* CAV2648 (ŠEBESTA ET AL., 1987) was effective only against OMV 1 (UK Race 2) and OMV 1+2 (UK Race 3). This resistance could be used in combination with another major gene or adult plant resistance (Figures 41 and 42). In areas where mildew is less prevelant it might be feasible to combine different resistance genes, but not, for instance in the UK, where the inoculum pressure is high and multi-virulence combinations have evolved rapidly in the past.

The feasibility of disease resistance breeding

Incorporating major gene sources of resistance into commercial cereal cultivars by conventional breeding methods is a relatively straightforward procedure and preferred by plant breeders. The ephemeral nature of these resistances and the length of time taken to evaluate advanced breeding lines results in many being overcome by new virulent strains before they are available to the grower. There are exceptions in other cereal crops; the recessive *ml-o* barley mildew resistant alleles have been effective throughout Europe since the early 1980s (Lyngkaer, 1995), but no similar type of resistance has been found in oats. Polygenically inherited resistances can be expected to be durable but more difficult to exploit fully. Further work is needed to identify the genes involved and understand the mechanisms that confer durability.

Problems associated with the production of resistant cultivars and their durability

Results from UK virulence surveys, as previously stated, show that the effectiveness of sources of resistance deployed over the last thirty years has been low. In central Europe, where mildew is less prevalent, some sources such as the OMR 3 resistance from cv. Mostyn have been more effective (ŠEBESTA ET AL., 1991). New sources of major gene resistance in hexa-

ploid oat species appear to be very scarce. The main source of resistance currently being deployed in European breeding programmes would appear to be that from the *A. sterilis*-derived line Pc 54 (ŠEBESTA ET AL., 1993). Resistances exist in Avena species at lower ploidy levels and have been successfully transferred into *A. sativa* notably from *A. barbata* (AUNG ET AL., 1977) and *A. eriantha* Dur. (HOPPE & KUMMER, 1991). However the former resistance has lost its effectiveness in the UK, demonstrating that transferring genes between species does not necessarily infer that these will be more effective than those from hexaploid backgrounds. Accessions of *A. atlantica* Baum. et *Fedak* sp. Nov., *A. longiglumis* Dur. and *A. strigosa*, all low ploidy species, have been identified with resistance to mildew isolates from Europe, including the UK, along with an accession of the hexaploid *A. occidentalis* Dur. (ŠEBESTA, ET AL., 1991; HERRMANN & RODERICK, 1996).

Donors of adult plant resistance

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, 1966, 1977 & 1978). Transgressive segregation for increased levels of APR has been demonstrated (JONES, 1983 and JONES & RODERICK, 1985). The lines APR 122 and APR 166 (HOPPE, 1991; HOPPE & KUMMER, 1991), derivatives of *A. eriantha* CAV 0128, appear to be highly effective at all EODN sites.

In field evaluations of a number of oat genotypes at Aberystwyth, several showed high levels of APR (Table 63) these were 93-2-4, Bage sel Klein, OM 1711 and Rouge d'Algerie along with the diploid S. 171.

Genetic analysis of the line *Pc 54* indicated that in addition to carrying the crown rust resistance gene *Pc 54* and *Pg 15* gene for stem rust resistance, it also possesses, in the re-selected lines Cc7422 and Pc 54-2, mildew resistance conditioned by a single incompletely dominant gene, along with additional factors modifying the expression of mildew resistance. There was no evidence of linkage between the mildew and crown rust resistance genes. Evaluation of selections from within the Pc 54 line showed that the expression of both stem rust and mildew resistance was modified by, or linked to, plant height (ŠEBESTA ET AL., 1993).

Identification of adult plant resistance and problems associated with its use

Mildew development on APR genotypes is characterised by a comparatively long latent period, low infection frequency and low sporulation capacity (JONES, 1978). The APR of cv. Maldwyn has remained effective since it was bred in the 1940s. JONES (1986) 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 cv. Mostyn to produce transgressive lines (JONES, 1983). Several other sources of APR have been identified and characterised (JONES, 1978; JONES & RODERICK, 1986; RODERICK & CLIFFORD, 1995, ŠEBESTA, ET AL., 1987). 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 (Figure 44).

The significant differences between high levels of APR under field conditions could not be fully reproduced in detached leaf tests under laboratory conditions (RODERICK & JONES, 1991; RODERICK & CLIFFORD, 1995). Some recent UK cultivars from IGER/WPBS, Aberystwyth, such as the spring oat cvs. Melys and Aberglen have a moderate level of APR, derived presumably from accumulating additive genes from parental sources, while the high level of mildew resistance of the winter oat cv. Solva is from an unknown source.

Possible linkage of mildew resistance genes with undesirable traits

Difficulties were encountered in combining the medium to high level of APR in cv. Roxton with high yielding potential, and also with shortness of straw, although after extensive testing a few lines were selected with satisfactory APR and length of straw. (LAWES ET AL., 1976). In a study of F_2 and F_3 generation plants of the cross of cv. Roxton with the susceptible cv. Selma there was no correlation found between the level of mildew and date of panicle emergence (JONES, 1974).

Combining high grain yield with the translocation *A. barbata* resistance in backcross lines also proved very difficult (JONES, ET AL., 1982), the translocated chromosome segment probably carrying deleterious yield genes.

Strategy of genetic control of powdery mildew

Major gene sources of mildew resistance so far exploited 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 was eroded the APR would give added protection. ŠEBESTA ET AL., (1993) recommended that this should be carried out with the resistance of Pc 54 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) and published here as Figure 44. 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.

Achievements of plant breeders in breeding mildew resistant cultivars

The mildew resistances identified in cv. Manod, Cc4146 and 9065Cn in the UK in the 1950s and 60s, were deployed in many cultivars grown from this period into the 1980s. So that the resistance from Cc4146 (OMR 2) was introduced firstly into cvs. Maris Tabard and Nelson and grown commercially from 1973, followed by cvs Maris Oberon, Trafalgar, Orlando, Cabana and Rollo. Similarly, the resistance from 9065Cn (OMR 3) was introduced into cv. Mostyn, grown commercially from 1968 and followed by cvs. Panema, Pinto, Avalanche and the naked spring oat cv. Rhiannon.

Since the sources of resistance exploited in the past did not remain effective for long periods most of the improvements in the level of mildew resistance in recent years have been through selecting for higher levels of APR. The results obtained by JONES (1983) demonstrated that adequate levels of resistance could be achieved by this breeding method. Some advanced breeding lines containing the mildew resistance from the line Pc 54 are showing promising results at IGER, Aberystwyth.

In the last decade in the Czech Republic a number of advanced oat lines with mildew resistance derived from cv. Mostyn (OMR 3) or its derivatives and line Pc39 were developed. Progress has also been made in incorporating these resistances into naked oats.

Septoria leaf blight and black stem

The characterization of the pathogen

The fungus *Septoria avenae* Frank was described for the first time by FRANK in Germany in 1895. Later the disease was found 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.

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 form *Septoria avenae* f. sp. *avenae* (SHAW, 1957a, b) (cit. HARDER and HABER, 1992). According to latest nomenclature the accepted name for this pathogen is the following: *Stagonospora* f. sp. *avenae* (syn. *Septoria avenae,* teleomorph: *Phaeosphaeria (= Leptosphaeria) avenaria* f. sp. *avenaria*) (CUNFER, 1994).

Effects of *Septoria avenae* f. sp. *avenae* on grain yield of oat

Septoria avenae can attack any above-ground part of oat plant. The attacking of stem results in a heavy lodging. Losses 34–43% were reported from Germany (MIELKE, 1975, MULLER, 1963, 1964), but its occurrence was also announced from the other parts of Europe (NOBLE, MONTGOMERIE, 1956, ŠEBESTA, 1985, cit. HARDER and HABER, 1992). In recent years *Septoria avenae* has been found in Austria, Germany, Italy and Poland (1990–1997). It seems that the fungus is in progress.

Incidence of *Septoria avenae* in Europe

The incidence of *Septoria avenae* f. sp. *avenae* at different localities is obvious from the Table 65. High occurrence of the pathogen was recorded in Austria at the locality Petzenkirchen in 1991, Drauhofen in 1992, Zwettl in 1993 and in Poland at the locality Danko in 1991 and Wielopole in 1991–1993. Moderate occurrence was found in Austria at the locality Fuchsenbigl in 1992 and 1993, at Petzenkirchen in 1993. In Germany the pathogen moderately occurred at Gross Lusewitz in 1993, in Italy at Badia Polesine in 1990 and in Rome in 1992, in Poland at the locality Choryn in 1990. Low occurrence of the pathogen was found in Austria at the locality Zwettl in 1992, in Germany at Berthelsdorf in 1990, at Quedlinburg in 1991 and in Italy in Rome in 1993.

Disease resistance index (DRI) of the oat genotypes included into the EODN

The variation of the resistance index of *Septoria avenae* on the 52 oat genotypes in the EODN trials in Austria, Germany, Italy, and Poland is obvious from the Table 66. The higher index the higher quantitative resistance is supposed. The accession *Avena sterilis* CAV 2648 has the higher value of the resistance index followed by oats Cc 4761, Pc 55, Pc 67, Pc 50-2, Pc 60, Pc 50-4, Pc 54, IL 86-6404, Garland, Pc 58, Pc 48 and Cc 6490. High frequency of R and MR evaluations in these oats as evident from the Table 66 indicates relatively high level of quantitative resistance to *Septoria avenae*.

Recently, CORAZZA ET AL. (1990, 1992) found that some of the oat cultivars grown in Italy were moderately resistant to *Septoria avenae* (Argenitina, Lidia, Manoire, Weibull), moderately susceptible (Angelica, Astra, Ava, Condor, Kalott, Nave, Ombrone, Perona, Vintero) or susceptible (Rogar 8, Sole II, Sonar).

However, some differences in aggressiveness of *Septoria avenae* populations presumably occur. E. g. *A. sterilis* CAV 2648 with the highest frequency of R evaluations was susceptible in Poland, at the locality Wielopole in 1992.

On the other hand, oats such as Rodney E, Roxton, Pc 59, OA 504-6, OA 504-5 and APR 166 being evaluated ten, eight, six, five and four times as moderately susceptible or susceptible, respectively, are supposed to be susceptible.

Our data on the quantitative response of oats to *Septoria avenae* f. sp. *avenae,* obtained from the natural occurrence of the pathogen, are supposed to be of potential importance for the selection of genotypes for disease resistance breeding.

However, exact comparative experiments with inoculum from different regions are very needed.

Strategy of the control of *Septoria avenae* f. sp. *avenae*

The plant disease resistance seems to be adequate to control of Septoria leaf blight and black stem (cit. HARDER and HABER, 1992). As indicated by Clark (1980) the Septoria resistance is polygenically inherited. However, the resistance to Septoria was found to occur in agronomically interesting oats (HOOKER, 1957 a, b, c) (cit. HARDER and HABER, 1992).

JOHNSTON ET AL. (1981) demonstrated the effectiveness of fungicides against Septoria in intensive cereal growing. The seed dressing is not too effective as the main source of infection is stubble and debris from the previous year's crop.

On the other hand, sanitation and crop rotation are recommendable to disease control (HARDER and HABER, 1992).

Helminthosporium leaf blotch, leaf stripe, and seedling blight

The characterization of the pathogen

The fungus *Drechslera avenae (Eidam)* Scharif (*Helminthosporiun avenae* Eidam) (perf. state *Pyrenophora avenae Ito et* Kurib.) is a common and destructive pathogen of oats in humid and cold regions of Europe and North America. 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, TAYLOR, 1943). According to MULLER (1963), *Pyrenophora 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 not noted to be a common fungus in Sweden (OLOFSSON, 1976) and Finland (REKOLA ET AL., 1970).

Pathogenic specialization of the fungus was demostrated (PANDEY and MISRA, 1974, TVEIT, 1956).

Effects of *Helminthosporium avenae* on the host plant

Seedling blight is the primary phase of the disease, arising from infected seed. Plants may be killed before or after amergence (HARDER and HABER, 1992).

Secondary infections (leaf and stem blight) arise on upper leaves from conidia produced following primary infections (HARDER and HABER, 1992).

Infection of the oat panicle results in the penetration of the hull and infection of seed (HARDER and HABER, 1992).

LUKE ET AL. (1957) (cit. Harder, Haber, 1992) attributed a black culm of oat in the southeastern USA to *Helminthosporium avenae.*

The incidence of *Pyrenophora avenae* in Europe as recorded in the EODN trials

The occurrence of the disease in 1990–1993 at the EODN localities is obvious from Table 67 Within 1990–1993 high or moderate high occurrence of the disease was recorded in Poland at the localities Choryn (1990), Danko (1991) and Wielopole (1992), in Austria at the localities Vienna (1991) and St. Donat (1993), in Italy at the locality Rome, in Finland at the locality Anttila (1993) and in Russia at the locality Nemchinovka (1993).

Moderate or weak-moderate incidence of the pathogen was recorded in the Czech Republic at the locality Kromeriz (1990, 1991), in Russia at Nemchinovka (1990), in Sweden at Svalöf (1990), in Germany at the locality Salzmünde (1991) and in Poland at the locality Wielopole (1991). Weak incidence of the pathogen was recorded in Finland at the locality Hankkija (1991), in Italy at the locality Rome (1993) and in Poland at Wielopole (1993).

Disease resistance index (DRI) of the oat genotypes included into the EODN

The quantitative reaction of the oat genotype is remarkable. The resistance index ranges from values of over 60 down to 22. From Table 68 it is obvious that the highest resistance index was found in the majority of Illinois lines (IL 86-1158, IL 85-6467, IL 86-4189, IL 85-2069, IL 86-6404, IL 86-56-98) that are tolerant to Barley Yellow Dwarf (BYDV) and also resistant to crown rust. Furthermore, included in the more resistant category to *Pyrenophora avenae* are the oats Maldwyn, Manod, Cc 3678, Pc 6l, Pc 60, Cc 476l, Pc 67, Pc 58, Orlando Pg 15,

Pc 59, Pc 50, Rodney A, Pg 16, Cc 6490, Jostrain, Garland, Pc 50-2, Pc 55, Roxton, KR 3813/73, Pc 39, Pc 56 and Pc 50-4.

Maybe, also oats APR 166 and Melys, OM 1621 and OM 1387 have some level of resistance but these were tested only in two or one year, respectively.

On the other hand oats such as Rodney ABDH, Maelor, OA 504-5, OA 503-1, Rodney B, Rodney M, Pan, Rodney E, Pc 38, Pc 54, Cc 4146, Pen^{2} x CAV 1376 and Pirol are regarded as moderately susceptible or susceptible but have been evaluated in some cases resistant or moderately resistant in these tests due to a low level of severity of the pathogen.

The quality of resistance is given by the number of resistant or by the sum of resistant and moderate resistant evalutions. The higher number of resistance readings indicates a higher level of resistance. The different range of virulence of the fungus populations can also be responsible for the differences in the resistant index.

The severity of *Pyrenophora avenae* on leaves and panicles in oats in Prague in 1992

Resistant in both leaves and panicles were cultivars (lines) KR 89-18, KR 8122, KR 9046, Explorer, Zlaták, 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 cvs. Lars, IL 86-6404, Semu 3767, Flamingsnova and IL 86-4467

Moderately resistant both in leaves and panicles were cvs. Auron, Wiesel, Tomba, IL 86-4189 and Calibre.

Moderately susceptible in leaves and resistant in panicles were cvs. P 5137, IL 85-2069, IL 86-5698 and Saia. Moderately susceptible in leaves and moderately resistant in panicles was the cv. Ogle.

Moderately susceptible in leaves and moderately susceptible in panicles were cvs. Akiwase, Dolphin, Joycee and Walaroo. The cv. Marloo was susceptible both in leaves and panicles.

The high natural occurrence of *Pyrenophora avenae* in central Bohemia in 1992 enabled to distinguish expressive differences in quantitative response of oat genotypes to this pathogen. However, these data should be considered as preliminary ones because of only one-year observation.

Up to now, our knowledge of the reaction of the host to *Pyrenophora avenae,* especially on panicles is very poor. As obvious from our data, the reaction of panicles can differ significantly from that of leaves as well. Therefore, while selecting parental cultivar for a resistance breeding programme both reaction of leaves and panicles should be taken into consideration. (Tables 69, 70).

It is promising that a number of Czech oat cultivars and advanced lines such as KR 89-18, KR 8122, KR 9046, Zlaták, Adam, David, Ardo and KR 9478 were resistant both in leaves and panicles. The fact might be explained by more or less regular occurrence of the pathogen in the Czech Republic and its negative effect on the grain yield. Selection of oats for the high yield has also been presumably the selection for a higher resistance to *Pyrenophora avenae* under such conditions.

Indications of pathogenic specialization of *Pyrenophora avenae* and differences in aggressiveness among its populations

As TVEIT (1956) and PANDEY and MISRA (1974) have already indicated there is probably pathogenic specialization of the pathogen and differences in aggressiveness among its populations may exist.

Even the oat lines with the highest resistance IL 86-1158, IL 85-6467 and IL 86-4189 were evaluated moderately susceptible or susceptible 2, 3 and 4 times, respectively, out of a total of 19 tests.

The cv. Pan being found to be relatively resistant to the central Bohemian population of the pathogen (ŠEBESTA ET AL., 1994) can be regarded according to resistance index to be relatively susceptible in Europe (Table 68). However, it is interesting to state that the cv. Pan was resistant in the Czech Republic at Kromeriz in 1990 (weak-moderate occurrence) but moderately susceptible at the same locality in 1991 (moderate occurrence), resistant in Finland at Hankkija (weak occurrence) and in Poland at Danko and Wielopole in 1991 (moderate-strong and weak-moderate occurrence, respectively), moderately resistant in Austria at the St. Donat locality and resistant in Italy in 1992 (moderate-strong occurrence) and in 1993 (weak occurrence).

Strategy of control of *Pyrenophora avenae*

Control of *P. avenae* is mainly established on seed treatment. Current treatments, especially with systemic fungicides, are very effective (VIR ET AL., 1970, PEZZALI, PORTA-PUGLIA, 1983, KANAPATHIPILLAI ET AL., 1975).

Cultural methods supporting rapid growth of seedlings reduce disease incidence (HARDER and HABER, 1992). Sanitation and crop rotation are also beneficial (HARDER and HABER, 1992).

Potential sources of *P. avenae* resistance, oats such as IL 86-1158, IL 85-6467, IL 86-4189, IL 85-2069, IL 86-6404 and IL 86-5698 and further ones included into the EODN are supposed to possess some *Pyrenophora avenea* resistance (Table 68).

Therefore, these oats are considered to be of potential importance as sources of *P. avenae* resistance.

However, these data should be considered as tentative ones as they are result of rough screening. Detailed analyses of genotypes reaction of these oats to *P. avenae* cultures under reproducible conditions are needed before making any conclusions as to their precise resistance values (ŠEBESTA ET AL., 1995).

CONCLUSIONS

Oat is subjected to a large number of diseases. Many of them can cause severe damage.

The use of resistant cultivars is the most feasible and economic means of controlling fungal diseases, crown rust, stem rust, and powdery mildew at present time and, maybe in the near future also Septoria leaf blight and black stem and Helminthosporium leaf blotch.

The importance of international cooperation in monitoring the disesases incidence and determining the effectiveness of resistance sources against these diseases is emphasized.

The breeding of multigenic cultivars, consisting of several resistance genes to crown rust and stem rust was proposed and has been practised with success in Canada and also in Europe for a number of years.

In 1990 the international oat disease project, called the European Oat Disease Nursery, was included into the European System of Cooperative Research Networks in Agriculture (ESCORENA) of Food and Agriculture Organization (FAO) and 31 national cooperators in 18 European countries participated in this cooperation.

In relation to crown rust the resistance genes Pc 39, Pc 55, Pc 58 and Pc 68 were effective against all pathotypes isolated in Europe so far. Genes such as Pc 48, Pc 50-2, Pc 50-4, Pc 54-1 and Pc 59, however, are of importance for the European crown rust resistance breeding of oat as well.

In relation to stem rust the gene Pg 13 (Rodney M), was the most effective followed by Pg a, Pg 16, Pg 4, Pg 15 and Pg 9. The effectiveness of stem rust resistance of the cv. Saia *(A. strigosa)* was very high. Effective were also two new *A. sterilis* accessions, VIR 343-1 and VIR 343-2.

Studies of inheritance of oat resistance to crown rust and stem rust are included as well.

Present knowledge on the resistance of oat to powdery mildew and the effects of the disease on the oat crop are reviewed. Adult plant resistant genotypes have been effective during the period under research. However, new sources of mildew resistance are required to protect advances made by breeders in grain yield improvement. Some success in transferring resistance between species has been achieved, most recently the resistance from *A. eriantha* has been incorporated into *A. sativa*.

The oat disease resistance seems to be presumably also adequate to control Septoria leaf blight and black stem and Helminthosporium leaf blotch. However, detailed analyses of genotype reaction of the potential oat donors of resistance under reproducible conditions are needed before making any conclusions as to their resistance values.

Strategies of the genetic control, especially for crown rust, stem rust and powdery mildew are proposed.

Zusammenfassung

Hafer (*Avenae sativa* L.) wird von einer Reihe von pilzlichen Krankheiten befallen. Die Ertrags- und Qualitätsbeeinflussung sind, abhängig vom jeweiligen Krankheitserreger und dem Befallsausmaß, unterschiedlich.

Die Nutzung von resistenten Sorten ist die ökologisch und ökonomisch brauchbarste Methode des Pflanzenschutzes gegen die derzeit wichtigsten Haferkrankheiten wie **Kronenrost** (*Puccinia coronata* Corda), **Schwarzrost** (*Puccinia graminis* subsp. *graminis* f. sp. *avenae* [Erikss. et Henn.]), **Mehltau** (*Erysiphe graminis* f. sp. *avenae* [Em. Marchal]), **Septoriose** – Septoria-Blattfleckenkrankheit des Hafers – Konidien-Form: *Septoria avenae* Frank f. sp. *avenae* (Syn. Stagonospora *avenae* / Frank / Biset f. sp. *avenae*), Hauptfruchtform: *Leptosphaeria avenaria* f. sp. *avenaria* G. F. Weber f. sp. avenaria, Syn.: *Phaeosphaeria avenaria*/G. F. WEBER/O. ERIKSSON f. sp. *avenaria*) – und Streifenkrankheit – Konidienform: *Helminthosporium avenae* Eidam (Syn. *Drechslera avenae* / Eidam / Scharif), Hauptfruchtform: *Pyrenophora avenae* Ito et Kuribay.

Die internationale Kooperation zur Überwachung des Krankheitsauftretens und zur laufenden Bestimmung und Überwachung von wirksamen Resistenzquellen kann als internationale Basisstrategie aus dem nichtchemischen Pflanzenschutz untermauert werden.

Die Züchtung von multigenen-Sorten, die mehrere unterschiedliche Resistenzgene gegen unterschiedliche Pathotypen führen, z. B. gegen Kronenrost und Schwarzrost, wird seit Jahren mit viel Erfolg in Kanada genutzt, aber auch in Europa.

Im Jahre 1990 wurde das Haferkrankheitsprojekt, besser bekannt als das „Europäische Haferkrankheiten Sortiment" in das Europäische System der kooperativen Netzwerk-Forschungsprojekt der Landwirtschaft (ESCORENA) der Nahrungs- und Landwirtschaftsorganisation FAO übernommen, in dem nun 31 nationale Institute aus 18 europäischen Staaten mitwirken.

Was den Kronenrost betrifft, waren bisher die Resistenzgene Pc 39, Pc 55, Pc 58 und Pc 68 gegen alle in Europa isolierten Pathotypen voll wirksam. Daneben wurde eine Anzahl von ebenfalls hoffnugsvollen Resistenzgenen mit hoher Wirksamkeit determiniert: Pc 48, Pc 50-2, Pc 50-4, Pc 54-1 und Pc 59.

Gegenüber Schwarzrost zeigt das Resistenzgen Pg 13 (Rodney M) die höchste Wirksamkeit, gefolgt von weiteren Genen wie Pg a, Pg 16, Pg 4, Pg 15 und Pg 9. Daneben erwiesen sich die Avenae strigosa-Sorte „Saia" und die Avena sterilis-Linien VIR 343-1 und VIR 343-2 als hoch wirksam.

Im Zusammenhang mit Kronenrost und Schwarzrost wurden auch Untersuchungen über die Vererbung der Resistenzgene durchgeführt und gefunden, daß neben dominanten Genen auch Gene mit rezessivem und heterozygotem Erbgang möglich sind.

Ein weiterer Abschnitt befaßt sich mit der gegenwärtigen Kenntnis über die Resistenz gegenüber Mehltau. Dabei wurden neue Resistenzgene sowie der interspezifische Transfer von Resistenzgenen aus *Avenae eriantha* vorgestellt.

Was die Septoriose und die Helminthoriose betreffen, sind die Untersuchungen zur Auffindung und Determination von Resistenzgenen noch nicht sehr spezifisch. Hier wurden vornehmlich im Rahmen der Erhebung des Befallsverhaltens untersucht, inwieweit unter gegenüber Kronenrost, Schwarzrost und Mehltau definierten Resistenzträgern auch Wirksamkeit gegen Septoriose und Helminthoriose auftritt. Es wurden durchaus auch gegen diese Krankheiten Resistenzen nachgewiesen.

Schließlich werden Strategien vorgeschlagen, Kronenrost, Schwarzrost und Mehltau genetisch zu kontrollieren.

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Table 1.

Effect of stem rust and crown rust on 1000-grain weight in oat (ŠEBESTA, 1971)

Rust	Parallel	1967		1968	
		Actual 1000-grain weight	Decrease in relation to C (in %)	Actual 1000-grain weight	Decrease in relation to C (in %)
Stem rust	I.	18,34	–29,35	17,59	–25,04
	II.	21,12	–15,32	18,81	–17,37
Crown rust	I.	20,84	–19,72	21,43	–8,65
	II.	21,74	–12,83	20,92	–8,13
Control	I.	25,96	–	23,46	–
	II.	24,94	–	22,77	–

1967: $md_{0.05}$ for rusts = 0.64, for interaction rust parallels = 0.9
1968: $md_{0.05}$ for rusts = 1.266

Table 2.

Percentage of hulls in oat attacked with stem rust and crown rust (ŠEBESTA, 1971)

Rust	Parallel	1967		1968	
		Actual percentage of hulls	Increase of percentage of hulls	Actual percentage of hulls	Increase of percentage of hulls)
Stem rust	I.	42,25	+39,44	46,70	+30,04
	II.	38,12	+27,65	42,26	+18,71
Crown rust	I.	35,26	+16,36	39,97	+11,30
	II.	36,92	+23,63	40,66	+14,21
Control	I.	30,30	–	35,91	–
	II.	29,86	–	35,60	–

1967: $md_{0.05}$ for rusts = 1.142, for parallels = 0.932, for interaction rusts X parallels = 1.615
1968: $md_{0.05}$ for rusts = 1.784, for interaction rusts X parallels = 2.523

Table 3.

Crude protein content in oat grains from plants attacked with stem rust and crown rust (interactions years X rusts) (ŠEBESTA et al., 1972)

Year	Rust	Content of crude proteins	
		Actual	Decrease in relation to C (in %)
1967	Stem rust	9,14	–24,15
	Crown rust	9,91	–17,8
	Control	12,05	–
1968	Stem rust	7,03	–26,46
	Crown rust	7,89	–17,49
	Control	9,56	–

Table 4a.

Content of bound amino acids in oat grains from plants attacked with stem rust and crown rust (ŠEBESTA et al., 1972)

Amino acid	Stem rust		Crown rust		Control
	Actual content	Decrease in relation to C (%)	Actual content	Decrease in relation to C (%)	
Lysine	4,86	–15,33	5,11	–10,98	5,74
Histidine	2,51	–30,28	2,89	–19,72	3,60
Arginine	6,72	–19,52	7,26	–13,05	8,35
Aspartic acid	8,30	–19,02	9,08	–11,41	10,25
Threonine	3,68	–11,75	3,89	–6,71	4,17
Serine	4,76	–16,78	5,11	–10,66	5,72
Glutamic acid	20,33	–18,16	22,11	–10,99	24,84
Proline	5,74	–7,12	5,52	–10,68	6,18
Glicine	6,09	–10,83	6,33	–7,32	6,83
Alanine	5,27	–15,00	5,54	–10,65	6,20
Cystine/2	1,75	–18,60	1,94	–9,77	2,15
Valine	6,69	–12,66	6,69	–12,66	7,66
Methionine	1,51	–11,70	1,42	–16,96	1,71
Isoleucine	4,60	–18,29	4,86	–13,68	5,63
Leucine	7,96	–17,43	8,50	–11,83	9,64
Tyrosine	3,59	–18,59	3,94	–10,66	4,41
Phenylalanine	5,21	–22,12	5,72	–14,50	6,69

Table 4b.

Effect of oat stem rust and oat crown rust on the proportion of free amino acids, determined by paper chromatography, in oat grains
(KRYZANEK, ŠEBESTA, 1974)

Amino acid	Numerical designation	1967			1968		
		Stem rust	Crown rust	Control	Stem rust	Crown rust	Control
Aspartic acid	1	++	++	++++	++	++	++++
Glutamic acid	2	++++	++++	+++++	++++	++++	+++++
Serine	3	+++	+++	++++	+++	+++	++++
Glycine							
Threonine	4	(+)	(+)	(+)	(+)	(+)	(+)
Alanine	5	+++++	++++	++++	+++++	++++	++++
a – aminobutyric acid	6	+++	++	++	+++	++	++
Phenylalanine	7	(+)	(+)	(+)	(+)	(+)	(+)
Isoleucine	8	(+)	(+)	(+)	(+)	(+)	(+)
Leucine	9	(+)	(+)	(+)	(+)	(+)	(+)
Asparagine							
Glutamine	10	++	++	+++	++	++	+++
Lysine	11	(+)	(+)	+	(+)	(+)	+
Proline	12	+	+	(+)	+	+	(+)

(+) „ – substance occurring in traces;“
\+ „ – small, little intensive spot;“
++ „ – small, intensive spot;“
+++ „ – moderate intensive spot;“
++++ „ – big intensive spot;“
+++++ „ – big and expressively intensive spot;“

Table 5.

Effect of stem rust and crown rust on oat grain composition from feeding point of view (ŠEBESTA, 1974)

Substance determined	Stem rust		Crown rust		Control plot
	Actual content	Relation to control (in %)	Actual content	Relation to control (in %)	
Dry matter	91,15	+ 0,24	90,80	– 0,14	90,93
Nitrogen substances	8,45	–22,83	9,06	–17,26	10,95
Lipids	3,89	+ 1,83	3,89	+ 1,83	3,82
Ash	4,56	– 6,37	4,70	– 3,49	4,87
Fibre	16,43	+19,32	15,32	+11,26	13,77
Extract of non-nitrogen substances	58,11	+ 1,01	57,84	+ 0,54	57,53
Digestible N-substances	6,00	–22,38	6,43	–16,82	7,73
Starch units (in 100 kg)	52,11	– 5,01	53,05	– 3,30	54,86

*) $md_{0.05}$ – nitrogen substances = 0.508 – digestible N-substances = 0.369
– fibre = 1.201 – starch units (in 100 kg) = 1.710

Table 6.

Effect of stem rust and crown rust on oat straw composition from feeding point of view (ŠEBESTA, 1974)

Substance determined	Stem rust		Crown rust		Control plot
	Actual content	Relation to control (in %)	Actual content	Relation to control (in %)	
Dry matter	93,07	0,68	92,32	– 0,12	92,44
Nitrogen substances	5,98	73,33	4,35	26,09	3,45
Lipids	1,67	10,60	1,63	7,95	1,51
Ash	7,96	–16,82	8,26	–13,69	9,57
Fibre	36,09	– 6,24	37,71	– 2,03	38,49
Extract of non-nitrogen substances	41,15	4,34	40,42	2,48	39,44
Digestible N-substances	1,20	73,91	0,87	26,09	0,69
Starch units (in 100 kg)	20,49	7,50	19,78	3,78	19,06

*) $md_{0.05}$ – nitrogen substances = 0.8782 – digestible N-substances = 0.1743
– ash =0.583 – starch units (in 100 kg) = 1.7063
– fibre = 1.186

Table 7

Effect of stem rust races 1 and 6F and the crown rust races 239 and CS 1 on the content of bound amino acids in the grain of the susceptible cv. Cesky zluty (Czech yellow) (in mg per 1 gram of dry substance) (ŠEBESTA, Sykora, 1974)

Amino acid	*Puccinia graminis avenae*				*Puccinia coronata avenae*				Control
	Race 1		Race 6F		Race 239		Race CS 1		
	Actual content	Relation to C (%)	Actual content	Relation to C (%)	Actual content	Relation to C (%)	Actual content	Relation to C (%)	
Lysine	3,64	–14,75	3,76	–11,94	3,76	–11,94	4,09	– 4,22	4,27
Histidine	1,34	–40,44	1,00	–55,56	1,31	–41,78	1,63	–27,56	2,25
Arginine	4,71	– 8,90	4,63	–10,44	4,74	– 8,32	5,72	+10.64	5,17
Aspartic acid	6,47	–18,21	6,63	–16,18	6,82	–13,78	7,31	– 7.59	7,91
Threonine	2,80	–22,01	2,97	–17,27	3,29	– 8,36	3,10	–13.65	3,59
Serine	4,01	–20,91	4,12	–18,74	4,57	– 9,86	4,38	–13.61	5,07
Glatamic acid	14,74	–18,52	14,88	–17,74	16,10	–11,00	17,76	– 1.82	18,09
Proline	3,91	–18,20	4,40	– 7,95	4,38	– 8,37	5,06	+ 5.86	4,78
Glicine	4,29	–13,68	4,38	–11,87	4,69	– 5,63	4,68	– 5.84	4,97
Alanine	4,34	–13,20	4,41	–11,80	4,60	– 8,00	4,76	– 4.80	5,00
Valine	3,90	–26,28	4,38	–17,20	4,45	–15,88	4,97	– 6.05	5,29
Isoleucine	3,25	–17,51	3,52	–10,66	3,57	– 9,39	3,90	– 1.01	3,94
Leucine	6,10	–15,28	6,44	–10,56	7,08	– 1,67	7,18	– 0.28	7,20
Tyrosine	2,41	–26,97	2,65	–19,70	2,84	–13,94	3,02	– 8.48	3,30
Phenylalanine	3,72	–28,87	4,23	–19,12	4,50	–13,96	4,84	7.46	5,23

Table 8.

Crown rust severity on oat cultivars and advanced lines and its effect on 1000-grain weight (ŠEBESTA, 1987)

Cv./line	Reaction	Severity	1000-grain weight	
			Actual	Decrease in relation to C
1 A Pan	HS	16,45	28,77	– 8,84
2 A Veles	HS	17,92	28,44	–13,74
3 A Petkus 7625	S	15,38	27,29	–11,11
4 A KR 2534	R/S	9,30	28,69	– 5,75
5 A KR 2454	HR	6,35	29,43	– 5,22
6 A KR 3975	HR	5,90	28,69	– 6,52
7 A Siluria	HR	9,53	26,33	– 5,25
8 A Orlando	HR	9,80	28,23	– 5,27
9 A Roxton	MR	12,65	35,48	+ 5,25
1 B Tiger	HS	14,70	29,30	– 4,81
2 B Diadém	S	15,30	33,82	– 3,78
3 B Hermes	S	15,90	31,20	– 4,13
4 B Saturn	S	8,08	36,02	– 4,20
5 B Solidor	HS	16,07	30,28	– 8,38
6 B Petkus 7573	S	10,50	30,90	– 2,83
7 B KR 106/31	S/MR	11,32	36,24	– 0,98
8 B KR 4027	R/S	6,27	29,98	– 5,07
9 B KR 2209	HR	0,23	29,96	– 1,29
10 B KR 3799	HR/S	6,42	26,78	– 8,82
11 B KR 3859	MR/S	7,15	29,22	– 6,59
12 B Flämnova	S	19,42	30,44	– 4,52
13 B Szegedi 30	R/S	8,87	32,42	+ 0,53
14 B Maelor	S	7,32	35,50	– 1,47
15 B Maldwyn	S	17,02	23,96	– 7,85

Table 9.

Stem rust severity on oat cultivars and advanced lines and its effect on 1000-grain weight (ŠEBESTA, 1987)

Cv./line	Reaction	Severity	1000-grain weight	
			Actual	Decrease in relation to C
1 A Pan	HS	47,50	23,52	–25,48
2 A Veles	HS	47,00	18,38	–44,25
3 A Petkus 7625	HS	44,00	19,02	–38,05
4 A KR 2534	HR/M	1,27	22,19	–27,10
5 A KR 2454	M	8,95	24,56	–20,90
6 A KR 3975	HS	44,00	21,74	–29,16
7 A Siluria	HS	40,17	15,95	–42,61
8 A Orlando	HS	40,00	17,62	–40,87
9 A Roxton	HR/M	0,85	25,40	–24,65
1 B Tiger	HS	51,83	21,68	–29,56
2 B Diadém	HS	44,17	27,70	–21,19
3 B Hermes	HS	44,83	25,78	–21,04
4 B Saturn	HS	49,00	27,86	–25,90
5 B Solidor	HS	45,50	28,26	–14,49
6 B Petkus 7573	HS	46,00	24,32	–23,52
7 B KR 106/31	HS/M	16,72	31,90	–12,84
8 B KR 4027	M	8,58	24,78	–21,53
9 B KR 2209	M	12,93	22,34	–26,39
10 B KR 3799	HS	42,17	19,38	–34,01
11 B KR 3859	HS	39,00	20,18	–35,49
12 B Flämnova	HS	44,33	25,42	–20,26
13 B Szegedi 30	M	26,75	29,74	– 7,78
14 B Maelor	HS	30,08	23,40	–35,05
15 B Maldwyn	HS	24,42	19,82	–23,77

Table 10.

The severity of powdery mildew and crown rust on oat cultivars and its effect on 1000-grain weight (ŠEBESTA, 1987)

Cv./line	Powdery mildew		Crown rust		1000-grain weight	
	Reaction	Severity	Reaction	Severity	Actual	Decrease in relation to C
1 A Pan	HS	18,68	HS	12,75	26,61	–15.68
2 A Veles	HS	21,25	HS	18,17	26,31	–20.20
3 A Petkus 7625	HS	17,43	S	12,12	26,62	–13.29
4 A KR 2534	R/S	5,78	MR	5,05	28,44	– 6.57
5 A KR 2454	R/HS	7,23	HR	1,28	28,87	– 7.02
6 A KR 3975	HR	0,05	MR	5,42	28,50	– 7.14
7 A Siluria	S	2,80	M	2,83	25,50	– 8.24
8 A Orlando	S	2,82	M	3,87	26,65	–10.57
9 A Roxton	S	5,18	R	2,68	32,38	– 3.95
1 B Tiger	HS	20,65	HS	11,87	27,30	–11.31
2 B Diadém	HS	12,67	S	10,83	32,20	– 8.39
3 B Hermes	HS	16,10	HS	13,23	30,80	– 5.67
4 B Saturn	HS	26,55	HS	10,93	33,83	–10.03
5 B Solidor	HS	29,25	HS	9,82	29,53	–10.65
6 B Petkus 7573	HS	14,28	S	8,03	29,45	– 7.39
7 B KR 106/31	HS	23,42	M	6,47	35,42	– 3.22
8 B KR 4027	HS	21,08	MR	2,58	29,02	– 8.11
9 B KR 2209	R/HS	2,82	HR	0,52	28,70	– 5.44
10 B KR 3799	S	10,87	MS	5,65	26,62	– 9.36
11 B KR 3859	HR	0,00	MS	5,28	26,82	–14.26
12 B Flämnova	HS	23,97	S	15,52	29,95	– 6.05
13 B Szegedi 30	HS	23,22	R	6,03	31,45	– 2.48
14 B Maelor	S	1,30	MS	5,58	33,77	– 6.27
15 B Maldwyn	HS	11,72	HS	8,40	22,85	–12.12

Table 11.

National cooperators and countries in which the European oat disease nursery was established in 1997 (ŠEBESTA, 1997)

National cooperator	Country
Hofrat Dr. B. Zwatz, Director	Austria
Dr. A. Shishlova	Belarus
Dr. N. Antonova	Bulgaria
Dr. P. Momchilova	Bulgaria
Dr. F. Machan	Czech Republic
Ing. M. Chourova	Czech Republic
Dr. J. Šebesta	Czech Republic
Dr. M. Koppel	Estonia
Dr. M. Saastamoinen	Finland
Dr. P. Franck	Germany
Dr. M. Herrmann	Germany
Dr. U. Stephan	Germany
Dr. Franz Stoiber	Germany
Dr. G. Zimmermann	Germany
Dr. R. Clothier	Great Britain
Dr. K. Bladenopoulos	Greece
Ing. T. E. Wouda	Holland
Dr. O. Weisz	Hungary
Dr. J. Manisterski	Israel
Dr. L. Corazza	Italy
Dr. B. Ezzahiri	Morocco
Dr. L. Reitan	Norway
Dr. Ing. J. Krolikowski	Poland
Ing. I. Longauer	Slovakia
Dr. J. Martin Lobo	Spain
Dr. B. Mattsson	Sweden
Dr. I. Loskutov	Russia
Dr. E. Lyzlov	Russia
Professor Dr. S. Stojanovic	Yugoslavia
Dr. R. Jevtic	Yugoslavia

Table 12.

Incidence (+ = low, ++ = moderate, +++ = high) of oat crown rust (*Puccinia coronata* f. sp. *avenae*) in Europe during 1990–1997 as observed in the European oat disease nursery trials (SEBESTA et al., 1997)

Country	Locality	Year							
		1990	1991	1992	1993	1994	1995	1996	1997
Austria	Drauhofen			++	++		+++	+++	
	Edelhof			++	++	++			
	Fuchsenbigl			+		+	+	++	
	Petzenkirchen			+	+++	+++	++	+++	++
	St. Donat			++	+	+++	+	++	+++
	Vienna	++	+++						
Bulgaria	Sadovo				++	+			
	Rousse							++	+++
Czech Rep.	Krukanice	++	++	++	++				
	Bystrice n. P.		++		(+)		+	+	
	Prague-East							++	++
	Prague-West				+	+	+	+	+
Estonia	Jogeva						+++	+++	+++
Finland	Jokioinen						++	+	+
France	Rheu	+++	++	+++					
Germany	Groß Lüsewitz							+	
	Freising								+
G. Britain	Aberystwyth	+++	+++	+++	++	++	+	+++	++
Hungary	Martonvasar								+
Israel	Bet Dagan						+++	+++	
Italy	Rome	+++		+	++	++	+++	+++	
Morocco	Rabat								+++
Poland	Borow			+		+	+	++	+++
	Polanowice		++		+	++	+++	+++	+++
	Strzelce					++	+	++	+++
	Wielopole		+++	++	+++	++	+++	+++	++
Russia	Nemchinovka				+++	+++			
	St. Petersburg					+++	+++	+++	+++
Slovakia	Pstrusa				+	+++			++
Sweden	Svalöf				+				
Yugoslavia	Kragujevac		+++	+	+	+++	+++		+++
	Novi Sad								+

Table 13.

Avirulence/virulence combinations of oat crown rust (*Puccinia coronata* f. sp. *avenae*) in Europe during 1992 to 1993 in relation to Pc-genes derived from *Avena sterilis* L. (ŠEBESTA et al., 1997)

Designation of sample	Number of virulence genes	Country avirulence/virulence combination
Austria		
40-92	3	38,39,48,50-2,50-4,54-1,55,58,59,60,61,62,64,67,68/54-2,56,63
46-92	4	38,39,48,50-2,50-4,54-1,55,56,58,59,62,63,67,68/54-2,60,61,64
51-92	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/67
58-92	4	38,39,48,50-2,50-4,54-1,55,56,58,59,62,63,67,68/54-2,60,61,64
17-93	1	38,39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,64,67,68/56
47-93	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,64,67,68/63
79-93	2	38,39,48,50-2,54-1,54-2,55,58,59,60,61,62,63,64,67,68/50-4,56
80-93	4	39,48,50-2,54-1,54-2,55,56,58,59,60,61,63,67,68/38,50-4,62,64
81-93	5	39,48,54-1,54-2,55,56,58,59,60,61,63,67,68/38,50-2,50-4,62,64
82-93	4	38,39,48,54-1,54-2,55,56,58,59,60,61,63,67,68/50-2,50-4,62,64
Belgium		
12-93	1	38,39,48,50-2,50-4,54-1,55,56,58,59,60,61,62,63,67,68/64
Czech Republic		
80-92	2	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,64,67,68/38,63
80-92/1	3	38,39,48,50-2,54-1,54-2,55,56,58,59,60,61,62,64,68/50-4,63,67
85-92	2	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,67,68/38,64
87-92/1	2	38,39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,64,68/56,67
92-92	3	38,39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,63,67,68/56,62,64
93-92	1	38,39,48,50-2,50-4,54-1,55,56,58,59,60,61,62,63,64,67,68/54-2
94-92	4	38,39,48,50-2,50-4,54-1,55,56,58,59,62,63,64,68/54-260,61,67
95-92	5	39,50-2,54-1,55,58,59,60,61,62,64,67,68/38,48,50-4,54-2,56
95-92/1	3	39,50-2,50-4,54-1,54-2,55,55,58,59,60,61,62,64,67,68/38,48,63
95-92/2	4	39,50-2,50-4,54-1,54-2,55,55,58,59,60,61,62,64,68/38,48,63,67
95-92/3	3	39,50-2,50-4,54-1,54-2,55,55,58,59,60,61,62,64,67,68/38,48,63
97-92	2	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,63,67,68/62,64
112-92	0	38,39,48,50-2,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
116-92	4	39,48,50-2,54-1,54-2,55,58,59,60,61,62,64,68/38,56,63,67
116-92/1	4	39,48,50-2,54-1,54-2,55,58,59,60,61,62,64,68/38,56,63,67
117-92	3	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,64,67,68/38,56,63
70-93	3	39,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,64,67,68/38,48,63
72-93	1	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/38
73-93	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-

Table 13 (Contd. 2)

Designation of sample	Number of virulence genes	Country avirulence/virulence combination
Czech Republic		
74-93	2	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/38,67
75-93	5	38,39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,68/56,62,63,64,67
92-93	4	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,68/38,56,64,67
93-93	2	39,48,50-2,54-1,54-2,55,56,58,59,60,61,62,63,67,68/38,64
110-93	2	39,48,50-2,54-1,54-2,55,56,58,59,60,61,62,64,67,68,/38,63
124-93	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,67,68/64
127-93	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/67
Estonia		
125-93	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
France		
1-92	2	38,39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,68/64,67
2-92/1	3	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,64,68/38,63,67
2-92/2	6	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,68/38,65,62,63,64,67
2-92/3	3	38,39,48,50-2,50-4,54-1,55,56,58,59,61,62,63,64,68/54-2,60,67
2-92/4	3	38,39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,68/56,64,67
6-93	2	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,68/64,67
7-93	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,67,68/64
Great Britain		
104-92	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,67,68/64
104-92/1	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
105-92	3	38,39,48,50-2,54-1,54-2,55,56,58,59,60,61,63,67,68/50-4,62,64
105-92/1	2	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,68/64,67
106-92	3	38,39,48,50-2,50-4,54-2,55,56,58,59,62,63,64,67,68/54-2,60,61
107-92/1	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
108-92	2	38,39,50-2,50-4,54-1,55,56,58,59,60,61,62,63,64,67,68/48,54-2
26-93	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,67,68/64
27-93	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,67,68/64
28-93	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
29-93	2	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,68/64,67
31-93	2	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,68/64,67
Italy		
43-92/2	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/67
44-92	5	38,39,48,50-2,50-4,54-2,55,56,58,59,63,67,68/54,2,60,61,62,64
44-92/1	5	38,39,48,50-2,50-4,54-2,55,56,58,59,62,63,68/54-2,60,61,64,67

Table 13 (Contd. 3)

Designation of sample	Number of virulence genes	Country avirulence/virulence combination
Italy		
44-92/1-1	6	38,39,48,50-2,50-4,55,56,58,59,62,64,68/54-1,54-2,60,61,63,67
44-92/3	5	38,39,48,50-2,50-4,54-2,55,56,58,59,63,64,68/54,2,60,61,62,67
Poland		
130-92	1	38,39,48,50-2,50-4,54-1,54-2,55,56,59,60,61,62,63,64,67,68/56
77-93	5	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,67,68/38,56,62,63,64
Slovakia		
41-92	3	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,67,68/63,64
42-92	6	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,68/38,56,62,63,64,67
42-92/1	5	39,54-1,54-2,55,56,58,59,60,61,62,64,67,68/38,48,50-2,50-4,63
77-92	1	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,63,64,68/67
82-92	1	38,39,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/67
83-92	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
84-92	3	39,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,64,67,68/38,48,63
89-92	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
90-92	1	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/38
91-92	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
103-92/1	2	38,39,48,50-2,50-4,54-1,55,56,58,59,60,61,62,63,67,68/54-2,64
123-92	4	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,68/38,56,64,67
123-92/1	1	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/38
68-93	4	39,48,50-2,54-1,54-2,55,58,59,60,61,62,64,67,68/38,50-4,56,63
Spain		
72-92	2	38,39,50-2,50-4,54-1,55,56,58,59,60,61,62,63,64,67,68/48,54-2
72-92/2	5	38,39,48,50-2,50-4,55,56,58,59,62,63,64,68/54-1,54-2,60,61,67
Sweden		
118-93	1	38,39,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/67
Yugoslavia		
15-92	4	38,39,48,50-2,50-4,54-1,55,56,58,59,62,63,67,68/54-2,60,61,64
20-92	0	38,39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,67,68/-
21-92/1	2	39,48,50-2,50-4,54-1,54-2,55,56,58,59,60,61,62,63,64,68/38,67
21-92/2	3	39,48,50-2,50-4,54-1,54-2,55,58,59,60,61,62,63,64,68/38,56,67
27-92	6	38,39,48,50-2,50-4,55,56,61,63,68/54-1,54-2,59,60,64,67
Israel		
2-93	5	38,39,48,54-1,54-2,55,56,58,59,62,63,64,68/50-2,50-4,60,61,67
3-93	3	38,39,48,50-2,50-4,54-1,55,56,58,59,62,63,64,67,68/54-2,60,61
5-93	6	50-2,50-4,56,58,59¨,60,61,62,63,64,68,68/38,39,48,54-1,54-2,55

Table 14.

Effectiveness of eighteen Pc-genes transferred from *Avena sterilis* L. accessions against European populations of oat crown rust (*Puccinia coronata* f. sp. *avenae*) in 1992 and 1993 (ŠEBESTA et al., 1997)

Pc-gene	Efectiveness (percentage of avirulent isolates)
Pc-38	65,1
Pc-39	100,0
Pc-48	89,5
Pc-50-2	96,5
Pc-50-4	89,5
Pc-54-1	96,5
Pc-54-2	79,1
Pc-55	100,0
Pc-56	79,1
Pc-58	100,0
Pc-59	98,8
Pc-60	86,1
Pc-61	89,5
Pc-62	87,2
Pc-63	74,4
Pc-64	60,5
Pc-67	61,6
Pc-68	100,0

Table 15.

Disease resistance index (DRI) to oat crown rust (*Puccinia coronata* f. sp. *avenae*) in 71 oat lines and cultivars included into the European oat disease nursery trials during 1990–1994 (After adjusting to the same number of evaluations) (ŠEBESTA et al.)

Line/Cultivar	Resistance index	Number of evaluations			
		R	MR	MS	S
Pc 68	185	36	5	1	1
Pc 58	183	38	3	1	2
Rodney ABDH	183	38	3	2	1
Rodney E	179	24	5	1	1
Pc 50-2	175	37	2	3	2
Pc 59	172	34	4	2	3
Pc 39	170	32	5	4	1
Garland	168	34	4	4	2
Pc 50	167	36	–	4	3
Pc 63	167	29	7	4	1
IL85-2069	167	29	7	2	3
Pc 55	165	27	7	3	2
Pen²x CAV1376	163	30	10	4	2
Rodney H	163	32	4	4	3
Pc 62	162	29	10	4	2
A. Sterilis CAV2648	161	22	4	3	2
IL86-5698	157	30	6	3	5
OA 504-5	156	22	5	4	2
Pc 56	155	31	4	6	3
KR288/73L/569	155	28	7	4	4
OA503-1	154	21	9	2	4
Cc 4761	149	29	4	7	3
IL86-1158	145	29	4	7	4
Pg 16	142	29	3	7	5
KR3813/73	141	28	3	8	4
IL86-6404	141	28	3	4	8
Pc 50-4	140	27	5	9	3
Rodney F	140	18	4	5	3
Roxton	140	24	8	8	3
Rodney D	139	20	2	5	4
Pc 61	138	24	4	5	6
Pg a	138	28	3	8	5
Rodney B	136	27	2	7	6
Pg 15	136	27	4	9	4
Pc 54-1	135	20	3	5	5
Pc 38	135	26	3	8	5
IL86-4467	134	25	5	12	1
Pc 60	134	30	1	7	8
Minrus	134	17	5	6	3
Pc 48	132	26	5	6	8
Rodney A	132	24	4	6	7
Jostrain	127	16	5	6	4
Vermiou	127	4	4	–	3
Kasadra	120	6	-	2	2

Table 15 (Contd. 2)

Line/Cultivar	Resistance index	Number of evaluations			
		R	MR	MS	S
Manod	116	25	–	6	12
IL85-6467	116	21	6	4	13
Pc 54-2	113	6	1	2	3
Pc 64	113	22	6	8	11
Rodney M	112	20	7	9	9
Pirol	106	20	5	11	9
OA 504-6	106	12	8	8	6
Pc 67	104	19	6	10	10
Cc4146	99	16	7	7	13
IL86-4189	98	20	2	5	17
Cc6490	97	14	9	10	10
Pc 54	75	3	2	5	2
APR 122	64	5	7	6	14
OM1621	62	4	5	6	10
OM1387	58	3	6	2	15
Cc3678	58	7	5	2	23
Orlando	51	6	7	8	23
APR 166	47	3	6	8	15
Mostyn	46	5	7	9	24
Maelor	45	6	5	11	21
KR9046	45	1	2	2	6
Adam	42	1	2	2	7
Melys	38	4	1	5	15
Maldwyn	35	3	6	12	22
Zlaták	30	–	2	5	3
Pan	23	2	4	9	29
KR8122	13	–	1	4	7

Table 16.

Reaction of 133 cultivars and lines of oat in the seedling stage to races 228, 231, 239, 240 and CS l of oat crown rust (*Puccinia coronata* f. sp. *avenae*) (ŠEBESTA et al., 1970)

Cv. / line	C. I. / C. A. N.	Sample obtained from	Physiologic race				
			228	231	239	240	CS 1
Abegweit	C. I. 4970	CZ	R-S/S	R	S	R/R-S	S
Ag 313	C. I. 7145	Israel	R	R	R	R	R/R-S
Ag 313	C. I. 7145	Rumania	R	R	R	R	R/R-S
Ag 313		Yugoslavia	R	R	R	R	R
Ag 331		Yugoslavia	R	R	R	R	R/R-S
Ag 354		Yugoslavia	R	R	R	R	R
Ajax	C. I. 4157	D	S/R-S	R/R-S	S	R-S	S
Alber	C. I. 2766	USA	R	R	R	R	S
Andrew	C. I. 4170	D	R	R	R	R	S
Anthony	C. I. 7001	USA	R	S	R	S	R/R-S
Appler	C. I. 7003	USA	R	R	R	R	S
Arlington	C. I. 4657	D	R	R	R	R	S
Arl 3x Wkt 2x Ctn x Sf 4x Flr	C. I. 7908	Rumania	R	R	R	R	R/R-S
(Atlantic x Cl-SF) x H-J	C. I. 6922	USA	R	R	R	R	R/R-S
Avena sterilis L.		CZ	S	S	S	S	S
Avon		CZ	R	R	R	R	S
Bage Sel. 364 Klein		CZ	R	R	R	R	S
Bambu II		CZ	S	S	S	S	S
Banner	C. I. 1729	CZ	S	S	S	S	S
Beacon		CZ	S	S	S	S	S
Beaver		CZ	S	S	S	S	S
Bentland	C. I. 6930	D	R	R	R	R	R/R-S
Blenda		CZ	S	S	S	S	S
Bond	C. I. 7004	USA	R	R	R	R	S
Bonda	C. I. 4329	D	R	R	R	R	S
Bondvic	C. I. 7009	USA	R	R	R	R	R/R-S
Bonham	C. I. 4676	D	R	R	R	R	S
Bonkee		CZ	R	R	R	R	S
Boone	C. I. 3305	D	R	R	R	R	S
Borne TT		Yugoslavia	R	R	R	R	S
Branch		CZ	R	R	R	R	S
Brighton		CZ	S	S	S	S	S
Buck 152		CZ	R	R	R	R	S
Burnett		Israel	R	R	R	R	S
Camelia	C. I. 4079	USA	R	R	R	R	S
Canuck		Israel	R	S	R	S	R/R-S
Cartier		CZ	S	S	S	S	S
Cenad 88		CZ	S	S	S	S	S
Centore	C. I. 3865	D	R	R	R	R	S
Clarion	C. I. 5647	D	R	R	R	R	S
Cleo	C. I. 6740	USA	R	R	R	R	R/R-S
Clintafe	C. I. 5869	USA	R	R	R	R	R/R-S
Clintland	C. I. 6701	D	R	R	R	R	R/R-S

Table 16 (Contd. 2)

Cv. / line	C. I. / C. A. N.	Sample obtained from	Physiologic race				
			228	231	239	240	CS 1
Clinton	C. I. 3971	D	R	R	R	R	S
Clinton x Arkansas		Yugoslavia	R	R	R	R	S
Clinton x Arkansas 674		Rumania	R	R	R	R	S
Cody II		CZ	R	R/R-S	R-S/S	R	S
Crater		CZ	R	R	R	R	S
Curt	C. I. 7424	D	R	R	R/R-S	R	R-S
Cesky zluty		CZ	S	S	S	S	S
Delair	C. I. 4653	USA	R	R	R	R	S
Dodge		CZ	R	R	R	R	R
Eagle$_2$		CZ	S	S	S	S	S
Eagle$_2$ x C. I. 4023		Rumania	S	S	S	S	S
Eagle$_2$ x C. I. 7438		Rumania	S	S	S	S	S
Exeter		Canada	S	S	S	S	S
Ferguson 560	C. I. 7161	D	R	R	R	R	S
Floriland	C. I. 6588	D	R	R	R	R	R/R-S
Fulghum		D	S	S	S	S	S
Fulgrain	C. I. 3693	D	R	R	R	R	S
Garland (KHC/2x/Cl/4x/ Land/3x/)	C. I. 8040	USA	R	R	R	R	R
Garland		CZ	R	R	R	R	R
Garry	C. I. 6662	D	R	R	R	R	S
Gopher	C. I. 2027	Yugoslavia	S	S	S	S	S
Green Russian		CZ	R	S	R	S	R/R-S
H-J (Fulwin x Lee-Vict) (Bond x Ant) x Land	C. I. 6666	USA	R	R	R	R	R/R-S
Hajira x Banner	C. I. 7438	Rumania	S	S	S	S	S
Hajira x Joanette	C. I. 4023	Israel	R	S	R	S	R/R-S
Hajira x Joanette	C. I. 4023	Rumania	R	S	R	S	R/R-S
Hadmerslebener Auswuchsfester Gelb		CZ	S	S	S	S	S
Hurron		CZ	S	S	S	S	S
Jackson	C. I. 5441	D	R	R	R	R	R/R-S
Jefferson	C. I. 7624	USA	R	R	R	R	R
Joanette		CZ	R	R	R	R	S
Jostrain	C. I. 2660	Yugoslavia	R	S	R	S	R/R-S
Kanota	C. I. 839	D	R	R	R	R	S
Krukanicky bezpluchy		CZ	S	S	S	S	S
La Premision		CZ	R	R	R	R	S
Landhafer	C. I. 7005	USA	R	R	R	R	R/R-S
Mabel	C.A.N. 542	D	R	R	R	R	S
Macon		D	S	S	S	S	S
Magistral		CZ	R	S	R	S	R/R-S
Markton	C. I. 2053	D	S	S	S	S	S
Minland	C. I. 6765	D	R	R	R	R	R/R-S
Minnesota II-47-12		Israel	R	R	R	R	R/R-S
Minnesota II-47-17		Israel	R	R	R	R	R/R-S

Table 16 (Contd. 3)

Cv. / line	C. I. / C. A. N.	Sample obtained from	Physiologic race				
			228	231	239	240	CS 1
Minnesota Oat Sel. 643114		USA	R	R	R	R	R
Minrus		Yugoslavia	R	S	R	S	R
Minton		CZ	R	R	R	R	S
Mohawk	C. I. 4327	D	R	R	R	R	S
Mo 0-205		CZ	R	R	R	R	S
Mutant = Rosen's Mutant		Israel	S/R-S	S	R	R	S
Nalzovsky		CZ	S	S	S	S	S
Nemaha	C. I. 4301	D	R	R	R	R	S
Newton	C. I. 6642	D	R	R	R	R	S
Nodoway		CZ	R	R	R	R	S
Nora (Ark. 3-74-H 3)	C. I. 8163	USA	R	R	R	R	R/R-S
O. g. 313		Yugoslavia	R	R	R	R	R/R-S
O. g. 331		Yugoslavia	R	R	R	R	R/R-S
O. g. 354		Yugoslavia	R	R	R	R	R/R-S
Onward		CZ	S	S	S	S	S
Onward 56		N. Zealand	S	S	S	S	S
Onward 63		N. Zealand	S	S	S	S	S
Ora	C. I. 7916	USA	R	R	R	R	R/R-S
Palestine	C. I. 2696	D	R/R-S	R	R/R-S	R	S/R-S
Purdue Selection	C. I. 7921	USA	R	R	R	R	R/R-S
Purdue Selection	C. I. 8153	USA	R	R	R	R	S
Putnam 61	C. I. 7531	D	R	R	R	R	R/R-S
Radar 2		CZ	R	R	R	R	R/R-S
Rodney	C. I. 6661	D	R	R	R	R	S
Rychlik		CZ	S	S	S	S	S
Sachalinskij 1		CZ	S	S	S	S	S
Saia	C. I. 7010	USA	R	R	R	R	S/R-S
Santa Fe	C. I. 7006	USA	R	R	R	R	R/R-S
Sol II		CZ	S	S	S	S	S
Sunland	C. I. 6600	USA	R	R	R	R	R/R-S
Sumavsky		CZ	S	S	S	S	S
Taggart	C. I. 4652	D	R	R	R	R	R
Tonka	C. I. 7192	D	R-S	R/R-S	R-S	R/R-S	S
Trispernia	C. I. 7008	USA	R	R	R	R	R/R-S
Ukraine	C. I. 7007	USA	S/R-S	S	R	R	S
Vicar	C.A.N. 827	D	R	R	R	R	S
Victoria	C. I. 7002	USA	R	R	R	R	S
Waubay	C. I. 5440	D	R	R	R	R	S
White Tartar		D	S	S	S	S	S
Winema	C. I. 4373	D	R	R	R	R	S/R-S
80,02		N. Zealand	R	R	R	R	S
	C. I. 4529	Canada	R	R	R	R	S
	C. I. 5844	Canada	R	R	R	R	S
	C. I. 6558	Canada	R	R	R	R	S
	C. I. 6574	USA	R	R	R	R	S
	C. I. 6829	Canada	R	R	R	R	S
	C. I. 6849	Canada	S	S	R	R	S

Table 17

Reaction of 133 cultivars and lines of oat in the seedling stage to races 201, 203, 214, 229 and 265 of oat crown rust (*Puccinia coronata* f. sp. *avenae*) (ŠEBESTA, 1972)

Cv. / line	C. I. / C. A. N.	Sample obtained from	Physiologic race				
			201	203/216	214	229	265
Abegweit	C. I. 4970	CZ	S	S	S	S	S
Ag 313	C. I. 7145	Israel	R	R	R	R	S
Ag 313	C. I. 7145	Rumania	R	R	R	R	S
Ag 313		Yugoslavia	R	R	R	R	S
Ag 331		Yugoslavia	R	R	R	R	S
Ag 354		Yugoslavia	R	R	R	R	S
Ajax	C. I. 4157	D	S	S	S	S	S
Alber	C. I. 2766	USA	S	S	S	R	S
Algerian		D	S	S	S	R	S
Amuri (80,02)		N. Zealand	S	S	S	R/R-S	S
Anthony	C. I. 7001	USA	R	S	S	R	R
Appler	C. I. 7003	USA	S	S	S	R	S
Arkwin	C. I. 5850	D	S	S	S	R+S	S
Avena sterilis L.		CZ	S	S	S	S	S
Avena strigosa x Abegweit		USA	R	R	R-S	S	R
Avon		CZ	S	S	S	R	S
Bage Sel. 364 Klein		CZ	R+S	S	S	R	S
Bambu II		CZ	S	S	S	S	S
Banner	C. I. 1729	CZ	S	S	S	S	S
Beacon		CZ	S	S	S	S	S
Beaver		CZ	S	S	S	S	S
Bentland	C. I. 6930	D	R	R	R	R	S
Blenda		CZ	S	S	S	S	S
Bond	C. I. 7004	USA	S	S	S	R	S
Bonda	C. I. 4329	D	S	S	S	R	S
Bondvic	C. I. 7009	USA	R	R	R	R	S
Bonham	C. I. 4676	D	S	S	S	R	S
Bonkee		CZ	S	S	S	R	S
Boone	C. I. 3305	D	R	R-S/S	S	R	R-S
Borne TT		Yugoslavia	S	S	S	R	S
Branch		CZ	S	S	S	R	S
Brighton		CZ	S	S	S	S	S
Buck 152		CZ	R	S	S	R	S
Burnett		Israel	S	S	S	R	S
Camelia	C. I. 4079	USA	S	S	S	R	S
Canuck		Israel	R	S	S	R	R
Cartier		CZ	S	S	S	S	S
Cenad 88		CZ	S	S	S	S	S
Clarion	C. I. 5647	D	S	S	S	R	S
Cleo	C. I. 6740	USA	R	R	R	R	S
Clintafe	C. I. 5869	USA	R	R	R	R	S
Clintland	C. I. 6701	D	R	R	R	R	S
Clinton	C. I. 3971	D	S	S	S	R	S
Clinton x Arkansas		Yugoslavia	R+S	S	S	R	R+S
Clinton x Arkansas 674		Rumania	R+S	S	S	R	R+S

Table 17 (Contd. 2)

Cv. / line	C. I. / C. A. N.	Sample obtained from	Physiologic race				
			201	203/216	214	229	265
Crater		CZ	R	S	S	R+S	S
Curt	C. I. 7424	D	R	S	S	S	S
Cesky zluty		CZ	S	S	S	S	S
Delair	C. I. 4653	USA	R	S	S	R	S
Dodge		CZ	R	R	R	R	R
Eagle		CZ	S	S	S	S	S
Eagle2 x C. I. 4023		Rumania	S	S	S	S	S
Eagle2 x C. I. 7438		Rumania	S	S	S	S	S
Exeter		Canada	S	S	S	S	S
Ferguson 560	C. I. 7161	D	S	S	S	R	S
Floriland	C. I. 6588	D	R	R	R	R	S
Fortune	C.A.N. 686	CZ	S	S	S	S	S
Fulghum		D	S	S	S	S	S
Fundy		CZ	S	S	S	S	S
Garland	C. I. 8040	USA	R	R	R	R	R
Garland		CZ	R	R	R	R	R
Garry	C. I. 6662	D	S	S	S	R	R-S/S
Gopher	C. I. 2027	Yugoslavia	S	S	S	S	S
Hadmerslebener Auswuchsfester Gelb		CZ	S	S	S	S	S
Hurron		CZ	S	S	S	S	S
Jackson	C. I. 5441	D	R+S	R+S	R+S	R	S
Joanette		CZ	S	S	S	R+S	S
Jostrain	C. I. 2660	Yugoslavia	R	S	S	R-(S)	R
Krukanicky bezpluchy		CZ	S	S	S	S	S
La Prevision 13		CZ	R	S	S	R	S
Landhafer	C. I. 7005	USA	R	R	R	R	S
Mabel	C.A.N. 542	D	S	S	S	R	S
Macon		D	S	S	S	S	S
Magistral		CZ	R	S	S	R-S	R
Markton	C. I. 2053	D	S	S	S	S	S
Minland	C. I. 6765	D	R	R	R	R	S
Minnesota II-47-12		Israel	R	R	R	R	S
Minnesota II-47-17		Israel	R+S	R+S	R+S	R	S
Minnesota Oat Sel. 643114		USA	R	R	R	R	R
Minrus		Yugoslavia	R	S	S	R-S	R
Minton		CZ	S	S	S	R	S
Mohawk	C. I. 4327	D	S	S	S	R	S
Mo 0-205		CZ	S	S	S	R	S
Mutant = Rosen's Mutant		Israel	R	S	S	S	R
Nalzovsky		CZ	S	S	S	S	S
Newton	C. I. 6642	D	S	S	S	R	S
Nodoway		CZ	S	S	S	R	S
Nora (Ark. 3-74-H 3)	C. I. 8163	USA	R	R	R	R	S/R-S
O. g. 313		Yugoslavia	R	R	R	R	S
O. g. 331		Yugoslavia	R	R	R	R	S

Table 17 (Contd. 3)

Cv. / line	C. I. / C. A. N.	Sample obtained from	Physiologic race				
			201	203/216	214	229	265
O. g. 354		Yugoslavia	R	R	R	R	S
Onward		CZ	S	S	S	S	S
Onward 56		N. Zealand	S	S	S	S	S
Onward 63		N. Zealand	S	S	S	S	S
Ora	C. I. 7916	USA	R	R	R	R	S/R-S
Palestine	C. I. 2696	D	S	S	S	S	S
Purdue Selection	C. I. 7921	USA	R	R	R	R	S
Purdue Selection	C. I. 8153	USA	R	S	S	R	R
Putnam 61	C. I. 7531	D	R	R	R	R	S
Radar 2		CZ	R	R	R	R	S
Richland	C. I. 787	Yugoslavia	S	S	S	S	S
Rodney	C. I. 6661	D	R	S	S	R	S
Rychlik		CZ	S	S	S	S	S
Sachalinskij 1		CZ	S	S	S	S	S
Saia	C. I. 7010	USA	R	R	S	S	R
Santa Fe	C. I. 7006	USA	R	R	R	R	S
Seminole	C. I. 5924	D	R	R	R	R	S
Simcoe	C.A.N. 742	CZ	S	S	S	S	S
Sol II		CZ	S	S	S	S	S
Sunland	C. I. 6600	USA	R	R	R	R	S
Sumavsky		CZ	S	S	S	S	S
Taggart	C. I. 4652	D	R	R	R	R	S
Tonka	C. I. 7192	D	S	S	S	S	S
Torch		CZ	R+S	S	S	R+S	R+S
Trispernia	C. I. 7008	USA	R	R	R	R	S
Ukraine	C. I. 7007	USA	R	S	R-S/S	S	R
Vanguard		CZ	S	S	S	S	S
Vicar	C.A.N. 827	D	S	S	S	R	S
Victoria	C. I. 7002	USA	R	R-S	S	R	R/R-S
White Tartar		D	S	S	S	S	S
	C. I. 4023	Israel	R	S	S	R	R
	C. I. 4023	Rumania	R	S	S	R	R
	C. I. 5844	Canada	R	S	S	R	R
	C. I. 6558	Canada	R	S	S	R	R
	C. I. 6666	USA	R	R	R	R	S
	C. I. 6829	Canada	R	S	S	R	R
	C. I. 6849	Canada	R	S	S	S	R
	C. I. 6922	USA	R	R	R	R	S
	C. I. 7438	Rumania	S	S	S	S	S
	C. I. 7908	Rumania	R	R	R	R	S

Table 18.

Reaction of oat collection in adult plant stage to a race population of oat crown rust (*Puccinia coronata* f. sp. *avenae*) (ŠEBESTA, 1970)

Cv. / line	C. I. / C. A. N.	Origin of sample	Reaction	Severity
Abegweit	C.A.N. 693	CZ	S	30
Ag 313	C. I. 7145	Israel	R^b	15
Ag 313	C. I. 7145	Rumania	R^a	t
Ag 313		Yugoslavia	R	5
Ag 331		Yugoslavia	R	5
Ag 354		Yugoslavia	R	10
Alber	C. I. 2766	USA	M^a	10
Andrew	C. I. 4170	D	M	10
Appler	C. I. 7003	USA	M	15
Arkwin	C. I. 5850	D	S	25
Arlington	C. I. 4657	D	M^b	15
Avena strigosa x Abegweit		USA	M	15
Bage Sel. 364 Klein		CZ	M^b	10
Bentland	C. I. 6930	D	R	10
Bond	C. I. 7004	USA	M	15
Bonda	C. I. 4329	D	M	5
Bondvic	C. I. 7009	USA	R	10
Bonham	C. I. 4676	D	R	5
Boone	C. I. 3305	D	M^b	10
Borne TT		Yugoslavia	M	10
Buck 152		CZ	M^b	25
Burnett		Israel	M	10
Camelia	C. I. 4079	USA	M^a	10
Canuck		Israel	M	15
Centore	C. I. 3865	D	M	15
Clarion	C. I. 5647	D	M	10
Cleo	C. I. 6740	USA	R^a	2
Clintafe	C. I. 5869	D	R	10
Clintland	C. I. 6701	D	M	10
Clinton	C. I. 3971	D	M	10
Clinton x Arkansas		Yugoslavia	M	5
Cl^2 x Arkansas 674		Rumania	M	10
Curt	C. I. 7424	D	M^b	10
Cesky zluty		CZ	S	60
Delair	C. I. 4653	USA	R^a	2
Dodge		CZ	R^a	t
$Eagle_2$ x C. I. 4023		Rumania	S	40
$Eagle^2$ x C. I. 7438		Rumania	S	45
Ferguson 560	C. I. 7161	D	M	10
Floriland	C. I. 6588	D	R	10
Fulgrain	C. I. 3693		M^b	10
Garland	C. I. 8040	USA	R	t-10
Garland		CZ	R^a	t
Garry	C. I. 6662	D	R	5
Hadmerslebener Auswuchsfester Gelb		CZ	S	40
Indio	C. I. 7292	CZ	M	25
Jackson	C. I. 5441	D	M	5
Jefferson	C. I. 7624	USA	R^a	t
Kanota	C. I. 839	D	M^b	5
Krukanicky bezpluchy		CZ	S	40
La Prevision 13		CZ	M^b	5

Table 18. (Contd. 2)

Cv. / line	C. I. / C. A. N.	Origin of sample	Reaction	Severity
Landhafer	C. I. 7005	USA	R	10
Mabel	C.A.N. 542	D	M	10
Markton	C. I. 2053	D	S	40
Minland	C. I. 6765	D	R	10
Minnesota II-47-12		Israel	R	5
Minnesota II-47-17		Israel	R	3
Minnesota Oat Sel. 643114		USA	R	2
Minton		CZ	M	5
Mohawk	C. I. 4327	D	M	5
Mutant = Rosen,s Mutant		Israel	M	10
Nalzovsky		CZ	S	45
Nemaha	C. I. 4301	D	M[b]	10
Newton	C. I. 6642	D	M	3
Nodoway		CZ	M[a]	5
Nora	C. I. 8163	USA	R[a]	5
O.g. 313		Yugoslavia	R	3
O.g. 331		Yugoslavia	R	2
O.g. 354		Yugoslavia	R	5
Onward		CZ	S[a]	20
Onward 56		N. Zealand	S[a]	55
Onward 63		N. Zealand	S[b]	45
Ora	C. I. 7916	USA	R[a]	2
Putnam 61	C. I. 7531	D	R	5
Radar 2		CZ	R[a]	t
Rodney	C. I. 6661	D	M	10
Rychlik		CZ	S	30
Sachalinskij 1		CZ	S[b]	20
Saia	C. I. 7010	USA	R	3
Santa Fe	C. I. 7006	USA	R	5
Seminole	C. I. 5924	D	M	5
Sunland	C. I. 6600	USA	R[a]	2
Sumavsky		CZ	S	40
Taggart	C. I. 4652	D	R	3
Tonka	C. I. 7192	D	M	15
Torch		CZ	M	40
Trispernia	C. I. 7008	USA	R	5
Ukraine	C. I. 7007	USA	M	5
Vicar	C.A.N. 827	D	M	10
Victoria	C. I. 7002	USA	M	10
Waubay	C. I. 5440	D	M	3
Winema	C. I. 4373	D	R	5
	C. I. 4529	Canada	R[a]	t
	C. I. 5844	Canada	R[a]	2
	C. I. 6558	Canada	M[a]	5
	C. I. 6574	USA	R[a]	5
	C. I. 6829	Canada	R[a]	t
	C. I. 6922	USA	R[a]	t
	C. I. 7438	Rumania	S	30
	C. I. 7908	Rumania	R	2
	C. I. 7921	USA	R	10
	C. I. 8153	USA	R	5

[a] tested in 1968
[b] tested in 1967

Table 19.

Segregation of F_2 generation of oat adult plants according to reaction to oat crown rust (*Puccinia coronata* f. sp. avenae), races 239 and 265 in crosses of resistant cvs. Dodge and Garland with the susceptible cvs. Bento and Tiger (ŠEBESTA, 1977a)

Test/Cross	Race/ Culture	Plants			Expected ratio	X^2	P
		R	S	n			
1. Dodge x Bento	239/ 44–73/1	317	13	330	61 : 3	0,41	0.7–0.5
2. Dodge x Tiger	265/ 66–66/1	216	70	286	3 : 1	0,042	0.9–0.8
3. Garland x Tiger	265/ 66–66/1	232	83	315	3 : 1	0,31	0.7–0.5

Table 20.

Segregation of F_2 generation of oat seedling plants according to reaction to oat crown rust (*Puccinia coronata* f. sp. avenae), races 201, 228, 229, 230, 232, 238, 239 and 240 in crosses of resistant cvs. Dodge and Garland with the susceptible cvs. Bento and Diadém

Test/Cross	Race/ Culture	Plants			Expected ratio	X^2	P
		R	S	n			
1. Dodge x Bento	201/ 42–73/2	197	11	208	61 : 3	0,17	0.7–0.5
2. Dodge x Diadém	228/ 32–72/1	203	8	211	61 : 3	0,38	0.7–0.5
3. Bento x Dodge	229/ 34–73/3	109	4	113	61 : 3	0,33	0.7–0.5
4. Bento x Dodge	239/ 44–73/1	198	9	207	61 : 3	0,053	0.9–0.8
5. Dodge x Diadém	240/ 9–71/2	245	11	256	61 : 3	0,087	0.8–0.7
6. Bento x Garland	228/ 73–73/1 229/ 34–73/1	218	9	227	61 : 3	0,26	0.7–0.5
7. Bento x Garland	230/ 12–73/2 239/ 44–73/1	145	9	154	61 : 3	0,46	0.5
8. Bento x Garland	232/ 67–67/2	229	11	240	61 : 3	0,006	0.95-0.90
9. Diadém x Garland	238/ 128–72/2	128	5	133	61 : 3	0,26	0.7–0.5

Table 21.

Segregation of F_2 generation of seedling oat plants according to reaction to oat crown rust (*Puccinia coronata* f. sp. *avenae*), races 216, 265 and CS 1 in crosses of resistant cvs. Dodge and Garland with the susceptible cvs. Bento and Diadém (ŠEBESTA, 1977a)

Test/Cross	Race/ Culture	Plants			Expected ratio	X^2	P
		R	S	n			
1. Dodge x Bento	216/ 58–67/1	158	50	208	3 : 1	0,1	0.8–0.7
2. Dodge x Diadém	265/ 48–72/1	156	55	211	3 : 1	0,13	0.8–0.7
3. Dodge x Diadém	265/ 83–72/1	144	42	186	3 : 1	0,58	0.5–0.3
4. Dodge x Bento	265/ 66–66/1	158	54	212	3 : 1	0,025	0.9–0.8
5. Bento x Dodge	265/ 66–66/1	87	26	113	3 : 1	0,24	0.7–0.5
6. Bento x Dodge	CS 1/ 153–65/1	155	52	207	3 : 1	0,002	1.0–0.95
7. Bento x Garland	265/ 66–66/1	116	38	154	3 : 1	0,009	0.95–0.90
8. Bento x Garland	CS 1/ 153–65/1	169	58	227	3 : 1	0,037	0.9–0.8

Table 22.

Segregation of F_2 generation of seedling oat plants in crosses of resistant cvs. Dodge and Garland with the susceptible cvs. Bento and Diadém according to reaction to oat crown rust (*Puccinia coronata* f. sp. *avenae*). Relationship of the genetic background

Test/Cross	Race/ Culture	Plants with combination			
		R R	R S	S R	S S
1. Dodge x Diadém	228/ 32–72/1 265/ 48–72/1	156	47	–	8
2. Dodge x Diadém	240/ 9–71/2 265/ 48–72/1	139	42	–	8
3. Bento x Garland	228/ 73–73/1 CS 1/ 153–65/1	169	49	–	9
4. Bento x Garland	239/ 44–73/1 265/ 66–66/1	116	29	–	9

Table 23.

Reaction of the seedlings of F_3 families of the crosses Dodge x Bento, Garland x Tiger and Dodge x Tiger to oat crown rust (*Puccinia coronata* f. sp. *avenae*), races 239 and 265. (ŠEBESTA, 1977)

Test/Cross	Race/ Culture	Number of families				Expected ratio	X^2	P
		R	Segr.	S	Total			
1. Dodge x Bento	239/ 44–73/1	67	32	1	100	45:18:1	0,89	0.7–0.5
2. Garland x Tiger	239/ 44–73/1	64	33	2	99	45:18:1	1,54	0.5–0.3
3. Dodge x Tiger	265/ 66–66/1	22	57	21	100	1:2:1	1,98	0.5–0.3

Table 24.

F_2 seedling segregation for crown rust reaction in crosses involving cv. Delphin and susceptible cultivars. (ŠEBESTA, 1979)

Test	Cross	Race/ Culture	Plants			Expected ratio	P
			R	S	T		
1	Delphin x Diadém	240/ 87–71/1	243	186	429	9 : 7	0.9–0.8
2	Delphin x Diadém	239/ 3–71/3	143	103	246	9 : 7	0.7–0.5
3	Cabot x Delphin	231/ 47–73/1	149	116	265	9 : 7	1.0–0.95
4	Cabot x Delphin	234/ 54–73/2	169	126	295	9 : 7	0.8–0.7
5	Cabot x Delphin	238/ 60–73/2	92	208	300	5 : 11	0.9–0.8
6	Cabot x Delphin	238/ 59–73/1	33	66	99	5 : 11	0.7–0.5
7	Cabot x Delphin	238/ 59–73/1	27	55	82	5 : 11	0.8–0.7

Table 25.

Seedling reaction to crown rust races 239 (the 1st group) and 230 (the 2nd group) in F_3 lines of the cross Delphin x Diadém. (ŠEBESTA, 1979)

Test	Race/ Culture	Number of lines			P value 1 : 8 : 7
		R	Segr.	S	
1	239/ 44–73/1	1	27	22	0.5–0.3
2	230/ 12–73/2	5	53	42	0.8–0.7

Table 26.

Relationship of the reaction in the Delphin cross to the two race groups of oat crown rust.
(ŠEBESTA, 1979)

Cross	Race/culture	Segregation ratio						P
		R		S	R	S		
		5 :	4 :	7	5 :	4 :	7	
Delphin	228/17–73/1	82	61	105				0.9–0.8
x Bento	230/12–73/2				82	61	105	

Table 27.

F_2 seedling segregation for crown rust reaction in the cross Pc 50-2 x Pc 50-4. Tested to oat crown rust isolate 7-77 P.
(ŠEBESTA, 1983)

Cross	Plants			Expected ratio	P
	R+M*	S	n		
319–1	227	85	312	3 : 1	0.5–0.3
319–4	257	95	352	3 : 1	0.5–0.3
321–1	228	82	310	3 : 1	0.7–0.5

M* = mesothetic (resistant and susceptible infection types present).

Table 28.

F_2 seedling segregation for crown rust reaction in the cross Pc 50-2 x Flamingsnova. Tested to crown rust isolate 9-77 P.
(ŠEBESTA, 1983)

Cross	Plants			Expected ratio	P
	R	S	n		
324–2	242	90	332	3 : 1	0.5–0.3
325–1	266	99	365	3 : 1	0.5–0.3
325–3	246	98	344	3 : 1	0.3–0.1

Table 29.

F_2 seedling segregation for crown rust reaction in the cross Pc 50-4 x Flamingsnova. Tested to crown rust isolate 9-77 P.
(ŠEBESTA, 1983)

Cross	Plants			Expected ratio	P
	R	S	n		
326–1	272	94	366	3 : 1	0.8–0.7
328–1	239	79	318	3 : 1	0,95

Table 30.

F_2 adult plant segregation for crown rust reaction in the cross Pc 50-2 x Flamingsnova and Pc 50-4 x Flamingsnova. Tested to crown rust isolate 9-77 P.
(ŠEBESTA, 1983)

Cross	Plants			Expected ratio	P
	R	S	n		
324–2	177	73	250	3 : 1	0.3–0.1
326–1	337	111	448	3 : 1	0.95–0.90

Table 31.

Seedling reaction to crown rust isolates 9-77 P and 7-77 P in F_3 lines of the crosses Pc 50-2 x Flamingsnova and Pc 50-2 x Pc 50-4.
(ŠEBESTA, 1983)

Cross	Crown rust isolate	Number of lines			P
		R	Segr.	S	(1 : 2 : 1)
324–2	9-77 P	23	54	23	0.8–0.7
319–1	7-77 P	21	56	23	0.5–0.3

Table 32.

Infection types and frequency of crown rust lesions in F_2 plants between oat lines Pc 50-2 and Pc 50-4 inoculated with crown rust isolate 7-77 P.
(ŠEBESTA, 1983)

Infection type (s)	Frequency of visible lesions					Number of plants
	0[1]	a	b	c	d	
0	3	–	–	–	–	3
„0;“	–	13	–	–	–	13
„0–1;“	–	19	4	1	–	24
„0–2;“	–	24	29	13	3	69
„0–3;“	–	8	29	32	15	84
„0–4;“	–	–	–	8	26	34
4	–	–	11	23	51	85
Number of plants	3	64	73	77	95	312

0[1] = no visible symptoms, a = very low, b = low, c = moderate, d = high frequency.

Table 33.

Segregation of oat plants in F_2 for reaction to race 265 of crown rust (*Puccinia coronata* f. sp. *avenae*) in seedling and adult plant stage in crosses of *A. fatua* CS 1 and *A. sativa* cultivars (ŠEBESTA, KUHN, 1990)

Cross	Plants			Expected ratio	P
	R	S	n		
Seedling Tests at Low Temperature					
A. fatua x Weikuss 2a/74	78	168	246	5 : 11	0.9–0.8
A. fatua x Dodge 481d	176	356	532	5 : 11	0.5–0.3
Rodney A x A. fatua 494a	50	129	179	5 : 11	0.5–0.3
Seedling Tests at High Temperature					
A. fatua x Rodney A 486a	98	401	499	3 : 13	0.7–0.5
A. fatua x Rodney A 489a	63	219	282	3 : 13	0.3–0.1
A. fatua x Rodney B 491b	61	228	289	3 : 13	0.5–0.3
A. fatua x Rodney M 493a	20	72	92	3 : 13	0.5–0.3
Adult Plant Tests					
A. fatua x Leanda 513g/74	41	153	194	3 : 13	0.5–0.3
A. fatua x Mona 518a/74	29	112	141	3 : 13	0.7–0.5
A. fatua x Weikuss 2a/74	24	145	169	3 : 13	0.3–0.1
A. fatua x Leanda 606b	42	178	220	3 : 13	0.9–0.8
A. fatua x Rodney A 486a	33	155	188	3 : 13	0.7–0.5
A. fatua x Rodney B 490a	17	71	88	3 : 13	0.9–0.8
Rodney B x A. fatua 497a	9	54	63	3 : 13	0.5–0.3

Table 34.

Seedling reaction to crown rust race 265 in F_3 lines of the cross with *A. fatua* CS 1. (ŠEBESTA, KUHN, 1990)

Cross	Plants			Expected ratio	P
	R	Segr.	S		
A. fatua x Weikuss 1b/74	3	26	21	1 : 8 : 7	1.0–0.95
A. fatua x Weikuss 2a/74	5	28	17	1 : 8 : 7	0.3–0.1
A. fatua x Leanda 513g/74	2	29	19	1 : 8 : 7	0.5–0.3
A. fatua x Leanda 513g/74–2	2	29	19	1 : 8 : 7	0.5–0.3
A. fatua x Weikuss 2a/74–2	3	30	17	1 : 8 : 7	0.5–0.3
A. fatua x Weikuss 3a/74	1	9	6	1 : 8 : 7	0.9–0.8
A. fatua x Leanda 514a/74	3	27	20	1 : 8 : 7	0.9–0.8
A. fatua x Mona 518a/74	1	28	21	1 : 8 : 7	0.5–0.3
A. fatua x Leanda 518a/74	3	27	20	1 : 8 : 7	0.9–0.8
A. fatua x Mona 518a/74	2	30	18	1 : 8 : 7	0.3–0.1

Table 35.

Seedling reaction to crown rust race 265 of segregation F_3 families of the crosses *A. fatua L.* and susceptible *A. sativa L.* cultivars. (ŠEBESTA, KUHN, 1990)

Cross	Plants			Expected ratio	P
	R	S	n		
A. fatua x Weikuss 2a/74–10	13 25	14	52[1]	1 : 2 : 1	0.95–0.90
A. fatua x Weikuss 2a/74–13	39	100	139	5 : 11	0.5–0.3
A. fatua x Weikuss 1b/74–18	47 30	163	240[1]	3 : 2 : 11	0.95–0.90
A. fatua x Weikuss 2a/74–21	178	366	544	5 : 11	0.5–0.3
A. fatua x Weikuss 2a/74–59	220	73	293	3 : 11	1.0–0.95
A. fatua x Weikuss 2a/74–75	85 171	76	332[1]	1 : 2 : 1	0.7–0.5
A. fatua x Weikuss 2a/74–34	113	486	599	3 : 13	0.9–0.8
A. fatua x Weikuss 2a/74–72	140	330	470	5 : 11	0.5–0.3
A. fatua x Weikuss 3a/74–59	55	164	219	1 : 3	1.0–0.95
A. fatua x Mona 518a/74–65	98	306	404	1 : 3	0.8–0.7

Table 36.

Relation of Pc-genes of *A. fatua* CS 1 to genes Pg-2 (A) and Pg-4 (B) (ŠEBESTA, KUHN, 1990)

Cross	Race of P. cor. P. gram.	Plants					Expected ratio	P
		R R	R S	S R	S S	n		
A. fatua x Rodney A (Pg 2) 486a	265 77	23	12	101	28	164	9:3:39:13	0.5–0.3
A. fatua x Rodney A (Pg 2) 489a	265 77	35	16	174	54	279	9:3:39:13	0.8–0.7
Rodney A (Pg 2) x A. fatua 494a	265 77	18	9	122	34	183	9:3:39:13	0.5–0.3
Rodney A (Pg 2) x A. fatua 491a	265 72	37	10	173	63	271	9:3:39:13	0.7–0.5

Table 37.

Inheritance of crown rust resistance in two *A. fatua L.* CS 1 derivatives. Segregation of seedlings in F_2 for reaction to race 265 of crown rust in crosses (*A. fatua L.* x Leanda) x KR 316 and (*A. fatua* L. CS 1 x Leanda) x Leanda. (ŠEBESTA, KUHN, 1990)

Cross	Plants			Expected ratio	P
	R	S	n		
A. fatua x Leanda (513g/74–95–49) x KR 316	29 69	440	538[1]	1 : 2 : 13	0.8–0.7
A. fatua x Leanda (513g/74 -2/77) x Leanda	211	450	661	5 : 11	0.8–0.7

[1] Differentiated 3 categories of plant reaction (0; 0–2; 3,4)

Table 38.

Comparison of the *A. fatua L.* CS 1 reaction to crown rust (*P. coronata* f. sp. *avenae*) with eight *A. sterilis* Pc-genes.
(ŠEBESTA, KUHN, 1990)

Race	Isolate	Reaction of								
		Pc 35	Pc 38	Pc 39	Pc 40	Pc 45	Pc 46	Pc 47	Pc 48	A. fa.
201	1	4	0,	2	3	3	3	4	0,	2
	3	0,	0,	0,	4	4	3	4	3	0,
214	1	3	0,	0,	2	4	2	3	3	2
228	1	0,	4	0,	0,	0,	0,	2	0,	4
	2	4	0,	0,	0,	0,	0,	2	0,	4
	3	4	0,	0,	0,	0,	0,	2	2	0,
	4	0,	2	0,	1	0,	0,	1	2	1
	5	0,	3	0,	0,	0,	0,	2	0,	2
229	1	4	0,	0,	0,	0,	2	2	0,	2
230	1	0,	0,	0,	4	3	0,	4	4	0,
232	1	4	4	0,	2	2	0,	2	0,	2
238	1	2	0,	0,	4	3	0,	3	2	0,
	2	4	0,	2	4	4	0,	4	3	1
239	1	4	2	0,	0,	2	0,	2	0,	4
	4	4	0,	0,	2	2	0,	2	0,	2
	5	4	4	1	2	1	0,	2	0,	2
240	1	4	0,	0,	2	2	2	2	2	2
	2	3	3	0,	2	2	0,	2	2	2
	3	0,	4	0,	2	2	2	2	2	2
	4	4	2	0,	2	2	2	2	2	2
	5	3	3	0,	2	0,	1	2	0,	4
259	1	2	0,	0,	3	3	0,	3	3	0,
264	1	2	0,	0,	4	3	4	4	2	2
265	1	4	0,	0,	2	4	4	4	2	0,
	2	0,	0,	0,	2	4	4	4	4	0,
	3	4	0,	0,	2	4	3	4	2	0,
282	1	4	0,	0,	2	4	4	4	3	0,
294	1	2	0,	0,	4	3	3	4	2	0,
CS 1	1	4	0,	0,	4	4	4	4	2	0,

0, = highly resistant, 1 = resistant, 2 = moderately resistant, 3 = moderately susceptible, 4 = highly susceptible

Table 39.

Incidence of oat crown rust (*Puccinia graminis* f. sp. *avenae*) in Europe during 1988–1996 as recorded at some localities of the European oat disease Nursery

Country	Locality	Year								
		1988	1989	1990	1991	1992	1993	1994	1995	1996
Austria	Drauhofen					+++	+++			++
	Edelhof					+				
	Fuchsenbigl					+				+
	Petzenkirchen					+				
	St. Donat					++	++			+
	Vienna	+++	++	+++	+++					
Bulgaria	Rousse									++
	Sadovo					++		++	+	
Czech Rep.	Bystrice n. P.				++					
	Kromuric								+	++
	Krukranice			+++[1]	++[1]	+++[1]	++[1]	+++[1]		++
	Prague-East									+
	Prague-West									+
Estonia	Jógeva								+++	++
Finland	Jokioinen								+	
Germany	Groß Lüsewitz									+
Italy	B. Polesine			++						
	Rome					+++		+++	++	+++
Poland	Wielopole	+++		+++						+
Russia	St. Petersburg								+++	+++
Slovakia	Pstrusa		++					+++		
Spain	Madrid	++				+++				
Gr. Britain	Aberyswyth					+				
Yugoslavia	Kragujevac	++[1]	+++[1]		+++[1]	++[1]	++[1]	+++[1]	+++	+++

+ = low incidenbce, ++ = moddrate, +++ = high
[1] inoculated

Table 40.

Disease resistance index (DRI) of oat cultivars and lines to oat stem rust *Puccinia graminis* Pers. f. sp. *avenae Erikss.* et *Henn.*) included into the EODN trials in 1988–1996 (after adjusting to 40 evaluations per line)

Cv. / Line	DRI	Number of evalutions				
		R	MR	MS	S	Total
Rodney M	139	28	9	2	1	40
Garland	131	26	9	4	1	40
Pg a	130	28	6	5	1	40
OA 504-5	127	20	4	4	1	29
KR 288/73L/569	124	18	8	5	–	31
Rodney ABDH	121	19	9	5	1	34
KR 3813/73	119	23	4	6	2	35
IL 86-1158	109	18	6	7	2	33
IL 85-2069	108	17	7	7	2	33
Kasadra	105	7	1	4	1	13
Pc 54-2	104	6	5	3	1	15
Pc 62	103	19	8	7	5	39
Jostrain	99	6	6	4	1	17
Pc 59	94	16	7	6	7	36
Pc 58	92	16	9	7	5	37
Pc 63	87	15	4	6	8	33
Pc 54	83	6	3	3	4	16
Rodney B	79	17	8	13	1	39
Pg 16	78	16	6	12	8	42
Roxton	75	13	7	12	7	39
Cc 4761	70	12	8	14	7	41
Minrus	70	4	4	7	1	16
A. sterilis CAV 2648	68	2	7	6	2	17
Pc 54-1	67	7	4	7	6	24
Pg 15	66	12	8	14	8	42
OA 503-1	63	8	4	9	7	28
OA 504-6	63	7	7	13	4	31
Rodney A	63	9	9	14	8	40
Rodney H	63	10	7	15	7	39
Vermiou	61	2	5	4	4	15
Rodney F	59	4	3	5	5	17
IL 86-6404	55	6	7	10	10	33
Rodney D	52	4	2	7	4	17
Cc 3678	51	5	4	7	9	25
Cc 6490	51	10	5	10	18	43
SG-K 95708	50	1	2	4	1	8
Cc 4146	49	6	8	13	12	39
IL 86-4189	48	7	4	10	12	33
Pirol	48	7	4	8	14	33
Maldwyn	47	6	8	12	15	41

Table 40 (Contd. 2)

Cv. / Line	DRI	Number of evalutions				
		R	MR	MS	S	Total
Pc 50-2	47	7	7	8	20	42
Rodney E	47	2	4	5	6	17
POB 1429/93	47	1	3	4	3	11
Maelor	45	6	7	15	12	40
Pc 39	45	8	5	10	19	42
Melys	42	2	4	6	7	19
Pc 48	42	7	3	8	17	35
Manod	41	5	5	7	17	34
Pc 60	40	8	3	13	17	41
Pc 67	39	5	4	12	12	33
APR 166	38	4	3	8	11	26
Pc 38	38	5	5	11	16	37
Pc 64	38	5	4	7	18	34
Pc 55	36	6	2	10	15	33
OM 1621	36	3	2	5	10	20
Pen^{2}xCAV 1376	36	6	2	12	13	33
Pc 56	35	4	4	13	11	32
IL 85-6467	34	2	6	8	15	31
Pc 68	34	4	4	8	17	33
Pc 50-4	32	5	4	14	17	40
APR 122	30	3	3	8	14	28
Pc 50	31	4	4	10	18	36
Pc 61	31	5	4	14	18	41
OM 1387	30	1	4	10	6	21
POB 14391	28	1	1	5	3	10
KR 9046	27	1	2	4	8	15
IL 86-4467	23	3	2	12	14	31
Mostyn	23	3	4	16	19	42
Orlando	20	2	3	15	14	34
Zlasák	17	1	2	7	13	23
Adam	16	–	2	4	9	15
KR 8122	15	–	2	5	9	16
Pan	4	–	1	13	14	28

Table 41.

Avirulence / virulence combinations of oat stem rust (*Puccinia graminis* f. sp. *avenae*) in Europe during 1989 to 1996 in relation to Pg-genes and some other sources of stem rust resistance

Designation of sample	Country	Avirulence/Virulence combination
48 - 92	A	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
50 - 92	A	4, 9, 13, 15, a, Sa, V1, V2 / 1, 2, 3, 8, 12, 16
57 - 92	A	2, 3, 4, 9, 12, 13, 15, a, Sa, V1, V2 / 1, 8, 16
60 - 92	A	4, 9, 13, a, Sa, V1, V2 / 1, 2, 3, 8, 12, 15, 16
34 - 92	A	1, 4, 8, 9, 12, 13, 15, a, Sa, V1, V2 / 2, 3, 16
49 - 92	A	2, 3, 4, 9, 12, 13, a, Sa, V1, V2 / 1, 8, 15, 16
52 - 92	A	1, 3, 8, 9, 12, 13, 15, 16, a, Sa, V2 / 2, 4, V1
70 - 92 / 1	A	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
38 - 92	A	1, 4, 9, 13, 15, a, Sa, V1, V2 / 2, 3, 8, 12, 16
39 -92	A	1, 2, 4, 8, 12, 13, 15, 16, A, Sa, V1, V2 / 3, 9
51 - 92	A	1, 3, 8, 13, 15, a, Sa, V1, V2 / 2, 4, 9, 12, 16
32 - 92	A	1, 2, 4, 8, 13, 15, a, Sa, V2 / 3, 9, 12, 16, V1
34 -92 / 2	A	3, 8, 9, 12, 15, 16, a, Sa, V2 / 1, 2, 4, V1
9 - 92	A	4, 9, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8, 12
34 - 92 / 3	A	1, 9, 13, a, Sa, V1, V2 / 2, 3, 4, 12, 15, 16
49 - 92 / 2	A	2, 4, 12, 15, a, Sa, V1, V2 / 1, 3, 8, 9, 16
34 - 92 / 2	A	1, 8, 9, 12, 13, 15, a, Sa, V1, V2 / 2, 3, 4, 16
51 -92	A	1, 3, 8, 12, 13, 15, a, Sa, V1, V2 / 2, 4, 9, 16
102 - 93	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
105 - 93	A	1, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2, 3
34 - 93	A	1, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2, 3
105 - 93 / 2	A	1, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2, 3
102 - 93 / 1	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
61 - 93	A	3, 4, 9, 12, 13, 15, a, Sa, V1, V2 / 1, 2, 8, 16
62 - 93	A	1, 8, 12, 13, 15, 16, a, Sa, V2 / 2, 3, 4, 9, V1
64 - 93	A	1, 2, 3, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 4, 9
101 - 93	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
108 - 93	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
103 - 93	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
65 - 93	A	3, 4, 9, 12, 13, 15, a, Sa, V1, V2 / 1, 2, 8, 16
105 - 93 / 1	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
64 - 93 / 1	A	3, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 4, 8, 9, 12
65 - 93 / 1	A	3, 9, 12, 13, 15, 16, a, sa, V1, V2 / 1, 2, 4, 8
2 - 94	A	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 2
30 - 94	A	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9
48 - 94	A	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9
33 - 94	A	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 15
46 - 94	A	1, 2, 3, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 9
46 - 94 / 2	A	1, 2, 3, 4, 12, 13, a, Sa, V2 / 8, 9, 15, 16, V1
43 - 94	A	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3, 9
50 - 94	A	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
33 - 94 / 2	A	1, 2, 3, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 9, 15
40 - 94	A	1, 2, 4, 8, 12, 13, Sa, V1, V2 / 3, 9, 15, 16

Table 41 (Continued 1)

Designation of sample	Country	Avirulence/Virulence combination
2 - 94 / 1	A	1, 3, 8, 9, 12, 13, a, Sa, V1, V2 / 2, 4, 15, 16
33 - 94 / 1 - 2	A	1, 2, 3, 4, 8, 13, 15, 16, a, Sa, V1, V2 / 9, 12
40 - 94 / 1	A	1, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
2 - 94 / 1 - 2	A	1, 2, 3, 8, 9, 12, 13, 15, 16, a, Sa, V2 / 2, 4, V1
33 - 94 / 1 - 2 - 1	A	1, 2, 3, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 9
40 - 94 / 1 - 1	A	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3, 9
33 - 94 / 1 - 2	A	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3, 9
96 - 96	A	4, 9, 13, 15, 16, a, Sa, V1, V2/1,2,3,12
104 -9 6	A	3, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2/1,2
13 - 90	BG	9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 4, 8
11 - 93	BG	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
64 - 96	BG	1, 2, 4, 9, 12, 13, 16, a, Sa, V1, V2/3,15
33 - 89	CS	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
33 - 89 / 1	CS	4, 9, 12, 13, 15, 16, a, Sa, V1 / 1, 2, 3, 8, V2
33 - 89 / 2	CS	4, 9, 12, 13, 16, a, Sa, V1 / 1, 2, 3, 8, 15, V2
3 - 90	CS	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
6 - 90	CS	4, 9, 12, 13, 16, a, Sa, V1 / 1, 2, 3, 8, 15, V2
15 - 90	CS	9, 13, 15, 16, a, Sa, V2 / 1, 2, 3, 4, 8, 12, V1
17 - 90	CS	9, 12, 13, 15, 16, a, Sa, V2 / 1, 2, 3, 4, 8, V1
22 - 90	CS	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
23 - 90	CS	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
36 - 90	CS	4, 9, 12, 13, 16, a, Sa, V1, V2 / 1, 2, 3, 8, 15
30 - 90	CS	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
2 - 91	CS	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
55 - 91	CS	2, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 3
61 - 91	CS	1, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
63 - 91	CS	3, 4, 9, 12, 13, a, Sa, V1, V2 / 1, 2, 8, 15, 16
65 - 91	CS	2, 4, 9, 12, 13, 15, a, Sa, V1, V2 / 1, 3, 8, 16
27 - 91	CS	3, 9, 12, 13, 15, a, Sa, V1 / 1, 2, 4, 8, 16, V2
41 - 91	CS	4, 9, 13, 15, Sa, V1, V2 / 1, 2, 3, 8, 12, 16, a
68 - 91	CS	9, 13, 15, Sa / 1, 2, 3, 4, 8, 12, 16, a, V1, V2
38 - 91	CS	13, Sa, V2 / 1, 2, 3, 4, 8, 9, 12, 15, 16, a, V1
57 - 91	CS	13, Sa / 1, 2, 3, 4, 8, 9, 12, 15, 16, a, V1, V2
69 - 91	CS	13, Sa / 1, 2, 3, 4, 8, 12, 15, 16, a, V1, V2
96 - 92	CS	3, 4, 8, 9, 13, 15, 16, a, Sa, V1, V2 / 1, 2
78 - 92	CS	3, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2
86 - 92	CS	2, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1
93 - 92	CS	2, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 3, 8
79 - 92	CS	9, 13, 15, 16, Sa, V2 / 1, 2, 3, 4, 8, 12, V1
78 - 92 / 1	CS	4, 9, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 12
93 - 92 / 1	CS	1, 4, 9, 13, 15, 16, a, Sa, V1, V2 / 2, 3, 12
78 - 92 / 2	CS	4, 9, 13, 15, 16, a, SA, V1, V2 / 1, 2
96 - 92 / 1	CS	4, 9, 13, 16, a, Sa, V1, V2 / 1, 2, 3, 8, 12, 15
93 -92 / 2	CS	9, 13, a, Sa, V1 / 1, 2, 3, 4, 8, 12, 15, 16, V2
87 - 92 / 2	CS	1, 8, 12, 13, 16, a, Sa / 2, 3, 4, 9, 15, V1, V2

Table 41 (Continued 2)

Designation of sample	Country	Avirulence/Virulence combination
111 - 93	CZ	3, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 8
25 - 93	CZ	1, 3, 8, 9, 12, 13, 15, a, Sa, V1, V2 / 2, 4, 16
72 - 93	CZ	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
111 - 93 / 1	CZ	3, 4, 8, 9, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 12
115 - 93	CZ	3, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 4, 8
115 - 93 / 1	CZ	13, 16, Sa / 1, 2, 3, 4, 8, 9, 12, 15, a, V1, V2
24 - 94	CZ	2, 3, 4, 8, 9, 12, 13, a / 1, 15, 16, Sa, V1, V2
1 - 94 / 1	CZ	1, 3, 4, 8, 9, 12, 13, 15, a, Sa, V1, V2 / 2, 16
24 - 94 / 2	CZ	2, 3, 4, 8, 9, 12, 13, a, V1 / 1, 15, 16, Sa, V2
22 - 94 / 1	CZ	1, 2, 3, 4, 8, 13, 16, a, Sa, V1, V2 / 9, 12, 15
23 - 94 / 1	CZ	1, 2, 4, 8, 13, 15, 16, Sa, V1, V2 / 3, 9, 12
100 - 95	CZ	4, 9, 13, 16, a, Sa, V1, V2 / 1, 2, 3, 12, 15
28 - 96	CZ	2, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2/1,3
124 - 96 / 1	CZ	4, 9, 13, 15, Sa, V1, V2/1,2,3,12,16, a
126 - 96 / 2	CZ	4, 9, 13, 15, 16, a, Sa, V2/1,2,3,8,12,V1
125 - 96	CZ	4, 13, 15, Sa, V1, V2/ 1, 2, 3, 9, 12, 16, a
135 - 96	CZ	2, 4, 13, a, Sa, V1, V2 /1, 3, 8, 9, 12, 15, 16
126 - 93	EE	2, 3, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 8
126 - 93 / 2	EE	12, 16, a, Sa / 1, 2, 3, 4, 8, 9, 13, 15, V1, V2
86 - 95	SF	2, 3, 4, 8, 9, 13, Sa, V1, V2 / 1, 12, 16, a
86 - 95 / 3	SF	1, 2, 3, 4, 8, 9, 13, 15, 16, Sa, V1, V2 / 12, a
86 - 95 / 2	SF	1, 2, 3, 4, 8, 9, 13, 15, 16, V1, V2 / a
86 - 95 / 1	SF	1, 2, 3, 4, 8, 13, 15, 16, Sa, V1, V2 / 9, 12, a
86 - 95 / 3 - 1	SF	1, 2, 3, 4, 8, 9, 13, 16, Sa, V1, V2 / 12, 15, a
111 - 92	D	2, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 3
111 - 92 / 3	D	4, 9, 13, 15, a, Sa, V1, V2 / 1, 2, 3, 8, 12, 16
111 - 92 / 1	D	9, 13, 15, a, Sa, V1, V2 / 1, 2, 3, 4, 8, 12, 16
111 - 92 / 2	D	13, 15, a, Sa, V1, V2 / 1, 2, 3, 4, 8, 9, 12, 16
131 - 92	GB	2, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 3
131 - 92 / 1	GB	4, 9, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8, 12
131 - 92 / 2	GB	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
43 - 92	I	1, 2, 4, 8, 13, a, Sa, V1, V2 / 3, 9, 12, 15, 16
43 - 92 / 1	I	1, 2, 4, 8, 12, 13, 15, 16, Sa, V1, V2 / 3, 9
45 - 92	I	2, 4, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 3, 8, 9
43 - 92 / 3	I	1, 2, 4, 8, 13, 15, 16, a, Sa, V1, V2 / 3, 9, 12
45 - 92 / 2	I	1, 2, 4, 8, 13, Sa, V1, V2 / 3, 9, 12, 15, 16, a
43 - 92 / 1 - 1	I	2, 4, 8, 13, 15, 16, a, Sa, V1, V2 / 1, 3, 9, 12
45 - 92 / 3	I	9, 13, 15, 16, Sa, V1, V2 / 1, 2, 3, 4, 8, a, 12
45 - 92 / 1	I	4, 13, Sa, V1, V2 / 1, 2, 3, 8, 9, 12, 15, 16, a
3 - 94	I	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
4 - 94	I	1, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 2, 3, 9, 15
5 - 94	I	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
6 - 94	I	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
7 - 94	I	1, 2, 4, 8, 12, 13, 16, a, Sa, V2 / 3, 9, 15, V1

Table 41 (Continued 3)

Designation of sample	Country	Avirulence/Virulence combination
7 - 94 / 1	I	1, 2, 3, 4, 8, 13, 15, 16, a, Sa, V1, V2 / 9, 12
4 - 94 / 1	I	1, 2, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / -
6 - 94 / 2	I	1, 2, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 3
4 - 94 / 2	I	1, 2, 3, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 9
6 - 94 / 1	I	1, 2, 8, 13, a, Sa, V1, V2 / 3, 4, 9, 12, 15, 16
3 - 95	I	1, 2, 3, 4, 8, 13, 15, 16, a, Sa, V1, V2 / 9, 12
3 - 95 / 2	I	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
24 - 96	I	3, 4, 12, 13, 15, 16, a, V1, V2 /1, 2, 8, 9, Sa
24 - 96 / 1	I	4, 9, 13, 15, a, Sa, V1, V2 /1, 2, 3, 12, 16
26 - 96 / 2	I	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3
77 - 93	P	13, 15, 16, a, Sa, V2 / 1, 2, 3, 4, 8, 9, 12, V1
76 - 93	P	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3, 9
115 - 96	P	4, 12, 13, 15, 16, a, Sa, V1, V2/ 1, 2, 3, 9
115 - 96 / 2	P	4, 9, 13, 15, a, Sa, V1, V2/ 1, 2, 3, 12, 16
130 - 93	RU	12, 13, 15, 16, a, Sa, V2 / 1, 2, 3, 4, 8, 9, V1
90 - 95	RU	2, 3, 4, 13, 15, 16, a, Sa, V1, V2 / 1, 8, 9, 12
90 - 95 / 1	RU	13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 4, 8, 9, 12
69 - 93	SK	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3, 9
69 - 93 / 1	SK	1, 2, 3, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 9
84 - 96 / 2 - 1	SK	3, 4, 9, 13, 15, 16, a, Sa, V1, V2 /1, 2, 12
84 - 96 / 2 - 3	SK	4, 9, 12, 13, 15, a, Sa, V1, V2 /1, 2, 3, 8, 16
87 - 96 / 2	SK	4, 8, 9, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 12
131 - 96	SK	3, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2
72 - 92	E	2, 3, 4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1
73 - 92	E	2, 9, 12, 13, 15, 16, V1, V2 / a
76 - 92	E	2, 4, 9, 12, 15, a, V1, V2 / -
74 - 92 / 1	E	4, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
76 - 92 / 1	E	4, 9, 12, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 8
73 - 92 / 1	E	9, 13, Sa, V1, V2 / 1, 2, 3, 4, 8, 12, 15, 16, a
76 - 92 / 2	E	4, 9, 12, 13, a, Sa, V1, V2 / 1, 2, 3, 8, 15, 16
73 - 92 / 2	E	4, 9, 13, 15, a, Sa, V1, V2 / 1, 2, 3, 8, 12, 16
117 - 93	S	2, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 3, 4, 8
116 - 93	S	4, 9, 13, 15, a, Sa, V1, V2 / 1, 2, 3, 8, 12, 16
116 - 93 / 1	S	3, 13, 16, Sa / 1, 2, 4, 8, 9, 12, 15, a, V1, V2
116 - 93 / 2	S	13, Sa / 1, 2, 3, 4, 8, 9, 12, 15, 16, a, V1, V2
89 - 95 / 1 - 1	S	1, 2, 3, 4, 13, 16, a, Sa, V1, V2 / 8, 9, 12, 15
127 - 96	S	1, 2, 3, 4, 9, 12, 13, 15, 16, a, Sa / V1, V2
127 - 96 /3	S	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 /3, 9, 12, 15
29 - 92	YU	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3
30 - 92	YU	1, 2, 4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 3
21 - 92	YU	2, 4, 8, 13, 16, a, Sa, V1, V2 / 1, 3, 9, 12, 15
26 - 92	YU	1, 2, 3, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 9
28 - 92	YU	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
16 - 92	YU	1, 3, 4, 8, 9, 12, 13, 15, 16, a, Sa, V2 / 2

Table 41 (Continued 4)

Designation of sample	Country	Avirulence/Virulence combination
29 - 92 / 2	YU	4, 8, 13, a, Sa, V1, V2 / 3, 9, 12, 16
26 - 92 / 1	YU	1, 2, 8, 13, a, Sa, V1 / 3, 4, 9, 12, 15, 16, V2
29 - 92 / 1	YU	1, 2, 4, 8, 13, 16, a, Sa, V1 / 3, 9, 12, 15, V2
23 - 92	YU	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
28 - 92 / 2	YU	1, 2, 4, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
30 - 92 / 2	YU	1, 2, 3, 4, 8, 13, 16, a, Sa / 9, 12, 15, V1, V2
28 - 92 / 1	YU	1, 2, 4, 8, 13, a, Sa, V1 / 3, 9, 12, 15, 16, V2
26 - 92 / 2	YU	1, 2, 4, 8, 12, 13, a, Sa, V1 / 3, 9, 15, 16, V2
26 - 92 / 2	YU	1, 2, 8, 13, 16, a, Sa, V1 / 3, 4, 9, 12, 15, V2
109 - 93	YU	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1, V2 / 3, 9
9 - 93	YU	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
13 - 94	YU	4, 8, 9, 12, 13, 15, 16, a, Sa, V1, V2 / 1, 2, 3
14 - 94	YU	1, 2, 4, 8, 12, 13, 15, 16, a, Sa, V1 / 3, 9, V2
17 - 94	YU	1, 2, 4, 8, 12, 13, 16, a, Sa, V1, V2 / 3, 9, 15
17 - 94 / 1	YU	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
13 - 94 / 1	YU	1, 3, 4, 13, 15, a, Sa, V1, V2 / 2, 8, 9, 12, 16
67 - 95	YU	1, 2, 3, 4, 13, 15, a, Sa, V1, V2 / 8, 9, 12, 16
69 - 95	YU	13, 15, 16, a, Sa, V1, V2 / 1, 2, 3, 4, 9, 12
67 - 95 / 2	YU	1, 2, 4, 13, 16, a, Sa, V1, V2 / 3, 8, 9, 12, 15
67 - 95 / 1	YU	1, 2, 4, 8, 9, 13, 15, 16, a, Sa, V1, V2 / 3, 12
74 - 96	YU	1, 2, 3, 4, 12, 13, 15, 16, a, Sa, V1, V2 /9
75 - 96	YU	2, 3, 4, 9, 12, 13, 15, a, Sa, V2 /1, 16, V1

A = Austria, BG = Bulgaria, CS = Czechoslovakia, CZ = Czech Republic, E = Spain, EE = Estonia, D = Germany, GB = Great Britain, I = Italy, P = Poland, RU = Russia, S = Sweden, SF = Finland, SK = Slovakia, YU = Yugoslavia

Table 42.

Avirulence / virulence combinations of oat stem rust (*Puccinia graminis* f. sp. *avenae*) in Israel in 1993 and 1995 in relation to Pg-genes and some other sources of stem rust resistance

Designation of sample	Country	Avirulence/Virulence combination
1 - 93	IL	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15
36 - 95 / 2	IL	2, 4, 8, 9, 12, 13, a, Sa, V1, V2 / 1, 3, 15, 16
38 - 95 / 1	IL	1, 2, 4, 8, 13, 16, a, Sa, V1, V2 / 3, 9, 12, 15

Table 43.

Effectiveness of Pg - genes and some other sources of stem rust resistance against European populations of *Puccinia graminis* f. sp. *avenae* in 1989 - 1996

Cv. / Line	major gene(s)	Country													
		A	BG	CS	CZ	EE	SF	D	GB	I	P	RU	SK	E	S	YU
Rodney D	Pg 1	73	33	12	28	0	75	0	33	**70**	25	0	33	0	43	**81**
Rodney A	Pg 2	42	33	15	33	50	100	25	67	**74**	25	33	33	38	57	**79**
Jostrain	Pg 3	46	0	12	50	50	100	0	0	**22**	0	33	60	12	43	**21**
Rodney B	Pg 4	77	67	58	78	50	100	50	100	**78**	75	33	100	75	57	**89**
Ea²xCAV4023	Pg 8	69	0	12	44	0	100	25	33	**74**	25	0	50	0	14	**75**
Rodney H	Pg 9	56	100	85	72	50	75	75	67	**22**	25	0	67	100	43	**18**
Kyto	Pg 12	75	100	61	50	100	0	25	33	**57**	50	33	67	75	29	**46**
Rodney M	Pg 13	100	100	100	100	50	100	100	100	**100**	100	100	100	100	100	**100**
Pg 15	Pg 15	71	67	67	67	50	75	100	67	**57**	100	100	100	75	43	**46**
Pg 16	Pg 16	67	100	73	61	100	75	25	100	**78**	75	100	83	50	80	**68**
Pg a	Pg a	100	100	85	83	100	0	100	100	**91**	100	100	100	75	71	**100**
Saia	?	100	100	100	89	100	100	100	100	**96**	100	100	100	100	100	**100**
Avena sterilis VIR 343 1 (V1)	?	88	100	76	83	50	100	100	100	**96**	75	67	100	100	57	**93**
Avena sterilis VIR 343 2 (V2)	?	100	100	73	83	50	100	100	100	**100**	100	100	100	100	57	**75**

Table 44.

Reaction of 130 cultivars and lines of oat tested in the seedling stage to races 2, 3, 4 and 6 of *Puccinia graminis* f. sp. *avenae* (ŠEBESTA, 1969)

Cv. / line	C. I. / C. A. N.	Country	Race			
			2	3	4	6
Abegweit	C. I. 4970	CZ	R	R	S	S
Ag 313	C. I. 7145	Israel	R	R	R	R
Ag 313	C. I. 7145	Rumania	R	R	R	R
Ag 313		Yugoslavia	R	R	S	S
Ag 331	C. I. 7144	Rumania	R	R	S	S
Ag 331		Yugoslavia	R	R	S	S
Ag 354		Yugoslavia	R	R	R	R
Ajax	C. I. 4157	D	R	R	S	S
Alber	C. I. 2766	USA	S	S	S	S
Algerian		D	S	S	S	S
Anthony	C. I. 7001	USA	R	S	S	S
Appler	C. I. 7003	USA	S	S	S	S
Avena sterilis L.		CZ	S	S	S	S
Bage Sel. 364 Klein		CZ	S	S	S	S
Banner	C. I. 1729	CZ	S	S	S	S
Bentland	C. I. 6930	D	R	S	S	S
Bond	C. I. 7004	USA	S	S	S	S
Bonda	C. I. 4329	D	R	S	S	S
Bonham	C. I. 4676	D	R	S	S	S
Boone	C. I. 3305	D	R	R	S	S
Borne TT		Yugoslavia	R	R	R	R
Branch		CZ	R	R	S	S
Bringhton	C. I. 4160	CZ	S	S	S	S
Buck 152		CZ	S	S	S	S
Burnett	C. I. 6537	Israel	R	R	R	R
Camelia	C. I. 4079	USA	S	S	S	S
Canuck	C. I. 4024	Israel	R	R	R	R
Cartier	C. I. 2565	CZ	S	S	S	S
Cenad 88		CZ	S	S	S	S
Centore		D	R	R	S	S
Clarion	C. I. 5647	D	R	R	S	S
Cleo	C. I. 6740	USA	R	S	S	S
Clintafe	C. I. 5869	USA	R	S	S	S
Clintland	C. I. 6701	D	R	S	S	S
Clinton	C. I. 3971	D	R	S	S	S
Clinton x Arkansas		Yugoslavia	R	S	S	S
Clinton[2] x Arkansas 674		Yugoslavia	R	S	S	S
Crater	C. I. 7295	D	S	S	S	
Curt	C. I. 7424	D	R	R	S	S
Cesky zluty		CZ	S	S	S	S
Delair	C. I. 4623	USA	S	S	S	S
Dodge		CZ	R	R	R	R
Eagle	C. I. 4113	CZ	S	S	S	

Table 44. (Contd. 2)

Cv. / line	C. I. / C. A. N.	Country	Race			
			2	3	4	6
Eagle² x C. I. 4023		CZ	R	S	R	S
Eagle² x C. I. 7438		Rumania	R	S	R	S
Exeter	C. I. 4158	Canada	R	R	S	S
Ferguson 560	C. I. 7161	D	S	S	S	S
Floriland	C. I. 6588	D	S	S	S	S
Fulghum	C. I. 708	D	S	S	S	S
Fulgrain	C. I. 3693	D	S	S	S	S
Fundy		CZ	R	R	S	S
Garland		CZ	R	R	R	R
Garry	C. I. 6662	D	R	R	R	R
Gopher	C. I. 2027	Yugoslavia	S	S	S	S
Hadmerslebener Auswuchsfester Gelb		CZ	S	S	S	S
Hurron		CZ	S	S	S	S
Charkovskij 596		CZ	S	S	S	S
Indio	C. I. 7292	D	R	R	S	S
Jackson	C. I. 5441	D	R	S	S	S
Jefferson	C. I. 7624	USA	R	S	S	S
Joanette		CZ	S	S	S	S
Jostrain	C. I. 2660	Yugoslavia	S	R	R	S
Krukanicky bezpluchy		CZ	S	S	S	S
La Prevision 13		CZ	S	S	S	S
Landhafer	C. I. 7005	USA	S	S	S	S
Lgovskij 1026		D	S	S	S	S
Mabel	C. A. N. 542	D	S	S	S	S
Macon	C. I. 6625	D	R	R	S	S
Markton	C. I. 2053	D	S	S	S	S
Mgh 462		Yugoslavia	S	S	S	S
Mgh 856		Yugoslavia	S	S	S	S
Minnesota II-47-12		Israel	R	R	R	R
Minnesota II-47-17		Israel	R	R	R	R
Minnesota Oat Sel. 643114		USA	R	R	R	R
Minrus	C. I. 2144	Yugoslavia	R	S	S	S
Minton		CZ	R	R	S	S
Mo 0-205		CZ	R	R	S	S
Mohawk	C. I. 4327	D	R	S	S	S
Nalzovsky		CZ	S	S	S	S
Nemaha	C. I. 4301	D	R	R	S	S
Nemcinovskij		D	S		S	S
Newton	C. I. 6642	D	R	R	S	S
Nodoway		CZ	R	R	R	R
Nora	C. I. 8163	USA	S	S	S	S
Novi Pazar		Yugoslavia	S	S	S	S
O. g. 313		Yugoslavia	R	R	R	R
O. g. 331		Yugoslavia	R	R	S	S
O. g. 354		Yugoslavia	R	R	R	R
Onward		CZ	S	S	S	S

Table 44. (Contd. 3)

Cv. / line	C. I. / C. A. N.	Country	Race			
			2	3	4	6
Onward 56		N. Zealand	S	S	S	S
Onward 63		N. Zealand	S	S	S	S
Ora	C. I. 7976	USA	S	S	S	S
Palestine	C. I. 2696	D	S	S	S	S
Putnam 61	C. I. 7531	D	R	R	R	R
Radar 2		CZ	R	R	R	R
Rauhhafer aus Hamstedt		D	S	S	S	S
Resistance		CZ	S	S	S	S
Richland	C. I. 787	Yugoslavia	R	R	S	S
Rodney	C. I. 6661	D	R	R	R	R
Ruakura	C. I. 2025	D	S	S	S	S
Rychlik		CZ	S	S	S	S
Sachalinskij 1		CZ	S	S	S	S
Saia	C. I. 7010	USA	R	R	R	R
Santa Fe	C. I. 7006	USA	S	S	S	S
Seminole	C. I. 5924	D	S	S	S	S
Simcoe	C. I. 6767	D	R	R	S	S
Sol II		CZ	S	S	S	S
Sovetskij		D	S	S	S	S
Sunland	C. I. 6600	USA	S	S	S	S
Szekacs 8		CZ	S	S	S	S
Sumavsky		CZ	S	S	S	S
Taggart	C. I. 4652	D	R	R	R	R
Tonka	C. I. 7192	D	R	R	S	S
Torch	C. A. N. 812	CZ	R	R	R	R
Trispernia	C. I. 7008	USA	S	S	S	S
Vicar	C. A. N. 827	D	R	R	R	R
Waubay	C. I. 5440	D	R	S	S	S
White Tartat		D	S	S	S	S
Winema	C. I. 4373	D	R	R	S	S
80,02		N. Zealand	S	S	S	S
	C. I. 4023	Israel	R	R	R	R
	C. I. 6666	USA	R	R	R	R
	C. I. 6922	USA	R	R	S	S
	C. I. 7438	Rumania	R	R	R	R
	C. I. 7908	Rumania	S	S	R	S
	C. I. 7921	USA	R	R	R	R
	C. I. 8040	USA	R	R	R	R
	C. I. 8153	USA	R	R	R	R

Table 45.

Reaction and severity of oat collection in adult stage to race population of oat stem rust (*Puccinia graminis* f. sp. *avenae*) (SEBESTA, 1970)

Cv. / line	C. I. / C. A. N.	Origin	Reaction	Severity
Abegweit	C. A. N. 693	CZ	M	50
Ag 313	C. I. 7145	Israel	R^b	10
Ag 313	C. I. 7145	Rumania	R^a	60
Ag 313		Yugoslavia	M	15
Ag 331		Yugoslavia	M	50
Ag 354		Yugoslavia	R	20
Alber	C. I. 2766	USA	S^a	80
Algerian		D	S	50
Amuri (80,02)		N. Zealand	S^a	30
Andrew	C. I. 4170	D	M^b	40
Appler	C. I. 7003	USA	S	40
Arkwin	C. I. 5850	D	S	70
Arlington	C. I. 4657	D	S^b	40
Avena strigosa x Abegweit		USA	R	25
Bage Sel. 364 Klein		CZ	S^b	30
Bentland	C. I. 6930	D	S	60
Bond	C. I. 7004	USA	S	50
Bonda	C. I. 4329	D	M	30
Bondvic	C. I. 7009	USA	M	40
Bonham	C. I. 4676	D	M	25
Boone	C. I. 3305	D	M	25
Borne TT		Yugoslavia	R	20
Brighton		CZ	S^b	30
Buck 152		CZ	S^b	20
Burnett		Israel	R	30
Camelia	C. I. 4079	USA	S^a	60
Canuck	C. I. 4024	Israel	R	20
Cartier		CZ	S^b	70
Centore	C. I. 3865	D	M	40
Clarion	C. I. 5647	D	M	50
Cleo	C. I. 6740	USA	S^a	20
Clintafe	C. I. 5869	USA	M^b	40
Clintland	C. I. 6701	D	M	50
Clinton	C. I. 3971	D	M	50
Clinton x Arkansas		Yugoslavia	M	40
Cl^2 x Arkansas 674		Rumania	M	40
Curt	C. I. 7424	D	M	35
Cesky zluty		CZ	S	80
Delair	C. I. 4653	USA	S^a	40
Dodge		CZ	R^a	30
$Eagle^2$ x C. I. 4023		Rumania	M	50
$Eagle^2$ x C. I. 7438		Rumania	M	50
Exeter		CZ	M	50
Ferguson 560	C. I. 7161	D	S	25

Table 45. (Contd. 2)

Cv. / line	C. I. / C. A. N.	Origin	Reaction	Severity
Floriland	C. I. 6588	D	S	30
Fulghum		D	S[b]	40
Fulgrain	C. I. 3693		S[b]	30
Fundy		CZ	M[b]	40
Garland	C. I. 8040	USA	R	15
Garry	C. I. 6662	D	R	25
Hadmerslebener Auswuchsfester Gelb		CZ	S	70
Indio	C. I. 7292	D	M	20
Jackson	C. I. 5441	D	M	30
Jefferson	C. I. 7624	USA	S	20
Kanota	C. I. 839	D	S[b]	20
Krukanicky bezpluchy		CZ	S	60
La Prevision 13		CZ	S[b]	20
Landhafer	C. I. 7005	USA	S	60
Mabel	C. A. N. 542	D	S	45
Minland	C. I. 6765	D	R	15
Minnesota II-47-12		Israel	R	10
Minnesota II-47-17		Israel	R	10
Minnesota Oat Sel. 643114		USA	R	15
Minton		CZ	M	50
Mohawk	C. I. 4327	D	S	30
Mutant = Rosen's Mutant		Israel	M	60
Nalzovsky		CZ	S	70
Nemaha	C. I. 4301	D	M	25
Newton	C. I. 6642	D	M	40
Nodoway		CZ	R[a]	25
Nora	C. I. 8163	USA	S[a]	30
O. g. 313		Yugoslavia	R	10
O. g. 331		Yugoslavia	M	50
O. g. 354		Yugoslavia	R	10
Onward5		CZ	S[b]	50
Onward 56		N. Zealand	S[a]	45
Onward 63		N. Zealand	S[a]	45
Ora	C. I. 7916	USA	S[a]	35
Putnam 61	C. I. 7531	D	R	20
Radar 2		CZ	R	20
Rodney	C. I. 6661	D	R	30
Rychlik		CZ	S	60
Sachalinskij 1		CZ	S[b]	30
Saia	C. I. 7010	USA	R	5
Santa Fe	C. I. 7006	USA	S	45
Seminole	C. I. 5924	D	S	25
Sunland	C. I. 6600	USA	S[a]	55
Sumavsky		CZ	S	65
Taggart	C. I. 4652	D	R	30
Tonka	C. I. 7192	D	M	20
Torch		CZ	R	25
Trispernia	C. I. 7008	USA	S	30

Table 45. (Contd. 3)

Cv. / line	C. I. / C. A. N.	Origin	Reaction	Severity
Ukraine	C. I. 7007	USA	M	35
Vanguard		CZ	M	65
Vicar	C. A. N. 827	D	R	15
Victoria	C. I. 7002	USA	S	30
Waubay	C. I. 5440	D	S	40
Winema	C. I. 4373	D	M	15
	C. I. 4023	Israel	R[b]	5
	C. I. 4023	Rumania	R	10
	C. I. 4529	Canada	S[a]	65
	C. I. 5844	Canada	M[a]	45
	C. I. 6558	Canada	S[a]	70
	C. I. 6574	USA	S[a]	45
	C. I. 6666	USA	R[a]	15
	C. I. 6829	Canada	M[a]	45
	C. I. 6849	Canada	M[a]	65
	C. I. 6922	USA	S[a]	35
	C. I. 7438	Rumania	R	25
	C. I. 7908	Rumania	M	30
	C. I. 7921	USA	R	35
	C. I. 8153	USA	R	10

[a] = tested in 1968
[b] = tested in 1967

Table 46.

Segregation of seedlings of the F_2 generation according to reaction to races 68, 76 and 77 of oat stem rust (*Puccinia graminis* f. sp. *avenae*) in crosses of resistant cvs. Garry, C. I. 6662, new release 290-1-1 and 292-1-3 and susceptible cvs. Diadém and Tarpan (ŠEBESTA, CERVENKA, 1977)

Test	Cross	Race/ Isolate	Plants			Expected ratio	X^2	P
			R	S	n			
1.	Tarpan x Garry	68/ 88–67/3	415	28	443	15 : 1	0,004	0,95
2.	290-1-1 x Diadém	76/ 80–73/1	241	85	326	3 : 1	0,200	0.70–0.50
3.	290-1-1 x Diadém	77/ 67–73/1	222	72	294	3 : 1	0,041	0.90–0.80
4.	292-1-3 x Diadém[1])	76/ 80–73/1	99	6	105	15 : 1	0,051	0.90–0.80
5.	292-1-3 x Diadém[2])	76/ 80–73/1	147	48	195	3 : 1	0,015	0.95–0.90

Table 47.

Segregation of adult plants of F_2 generation according to reaction to races 68, 72 and 74 of oat stem rust in cross of resistant cvs. Garry, new release 290-1-1 and 292-1-3 and susceptible cultivars
(ŠEBESTA, CERVENKA, 1977)

Test	Cross	Race/ Isolate	Plants			Expected ratio	X^2	P
			R	S	n			
1.	Tarpan x Garry	68/ 88–67/3	311	17	328	15 : 1	0,64	0.5–0.3
2.	Tarpan x Garry	(74)/ 264–65/12	201	52	253	3 : 1	2,67	0.3–0.1
3.	Garry x Diadém	(74)/ 264–65/12	204	77	281	3 : 1	0,86	0.5–0.3
4.	Diadém x Garry	72/ 86–69/2	70	31	101	3 : 1	1,75	0.3–0.1
5.	Garry x Diadém	72/ 86–69/2	115	41	156	3 : 1	0,14	0.8–0.7
6.	Garry x Flamingsstahl	(74)/ 264–65/12	87	22	109	3 : 1	1,35	0.3–0.1
7.	Garry x Flamingsstahl	72/ 86–69/2	124	36	160	3 : 1	0,53	0.5–0.3
8.	Permit x Garry	(74)/ 264–65/12	151	40	191	3 : 1	1,68	0.3–0.1
9.	Garry x Permit	(74)/ 264–65/12	28	14	42	3 : 1	1,56	0.3–0.1
10.	290-1-1 x Diadém 71	(74)/ 264–65/12	385	123	508	3 : 1	0,17	0.7–0.5
11.	290-1-1 x Diadém 71	72/ 86–69/2	318	110	428	3 : 1	0,11	0.8–0.7
12.	292-1-3 x Diadém 71	72/ 86–69/2	360	111	471	3 : 1	0,52	0.5–0.3

Table 48.

Reaction of seedling F_2 generation to races of oat stem rust (*Puccinia graminis* f. sp. *avenae*) in the crosses of resistant cvs. Dodge and Garland with the line Pc 39 (ŠEBESTA, 1977)

Test	Cross	Race/ Isolate	Plants			Expected ratio	X^2	P
			R	S	n			
1.	Dodge x Garland	72/ 33–73/1	278	–	278	–	–	–
2.	Dodge x Garland	77/ 67–73/1	278	–	278	–	–	–
3.	Pc 39 x Dodge	72/ 33–73/1	236	79	315	3 : 1	0,001	1.00–0.95
4.	Pc 39 x Dodge	76/ 37–73/1	295	20	315	15 : 1	0,005	0.95–0.90
5.	Pc 39 x Garland	72/ 33–73/1	232	77	309	3 : 1	0,001	1.00–0.95
6.	Pc 39 x Garland	76/ 37–73/1	288	21	309	15 : 1	0,16	0.70–0.50

Table 49.

Segregation of adult plants of F2 according to reaction to races 72 and 74 of *P. graminis* f. sp. *avenae* in the crosses of resistant cvs. Dodge and Garland with susceptible cvs. Diadém, Flamingsstahl and Permit (ŠEBESTA, 1977)

Test	Cross	Race/ Isolate	Plants			Expected ratio	X^2	P
			R	S	n			
1.	Dodge x Diadém	72/ 86–69/2	76	20	96	3 : 1	0,890	0.5–0.3
2.	Dodge x Diadém	(74)/ 264–65/12	281	104	385	3 : 1	0,830	0.5–0.3
3.	Diadém x Dodge	72/ 86–69/2	166	49	215	3 : 1	0,560	0.5–0.3
4.	Diadém x Dodge	(74)/ 264–65/12	286	94	380	3 : 1	0,014	0.95–0.90
5.	Dodge x Flamingsstahl	(74)/ 264–65/12	106	45	151	3 : 1	1,860	0.3–0.1
6.	Flamingsstahl x Dodge	72/ 86–69/2	85	27	112	3 : 1	0,048	0.9–0.8
7.	Flamingsstahl x Dodge	(74)/ 264–65/12	109	36	145	3 : 1	0,002	1.0–0.95
8.	Dodge x Permit	72/ 86–69/2	100	42	142	3 : 1	1,590	0.3–0.1
9.	Dodge x Permit	(74)/ 264–65/12	157	47	204	3 : 1	0,420	0.7–0.5
10.	Permit x Dodge	(74)/ 264–65/12	369	128	497	3 : 1	0,150	0,70
11.	Garland x Diadém	72/ 86–69/2	92	27	119	3 : 1	0,340	0.7–0.5
12.	Garland x Diadém	(74)/ 264–65/12	158	53	211	3 : 1	0,002	1.0–0.95
13.	Garland x Flamingsstahl	72/ 86–69/2	152	47	199	3 : 1	0,200	0.7–0.5
14.	Garland x Flamingsstahl	(74)/ 264–65/12	95	30	125	3 : 1	0,067	0.8–0.7
15.	Flamingsstahl x Garland	(74)/ 264–65/12	29	13	42	3 : 1	0,790	0.5–0.3
16.	Garland x Permit	72/ 86–69/2	179	58	237	3 : 1	0,035	0.9–0.8
17.	Garland x Permit	(74)/ 264–65/12	174	63	237	3 : 1	0,320	0.7–0.5
18.	Permit x Garland	72/ 86–69/2	182	65	247	3 : 1	0,230	0.7–0.5
19.	Permit x Garland	(74)/ 264–65/12	361	109	470	3 : 1	0,820	0.5–0.3

Table 50.

Relationship of the genes Pg-2 and Pg-4 for resistance to *Puccinia graminis* f. sp. avenae in the cvs. Dodge and Garland with the gene Pc 39 for resistance to *P. coronata* f. sp. *avenae*
(ŠEBESTA, 1977)

Test	Cross	Race/ Isolate	Plants				Expected ratio	X^2	P
		P. gram. av. P. cor. av.	R R	R S	S R	S S			
1.	Pc 39 x Garland	72/ 33–73/1 264/ 60–68/1	172	54	60	23	9 : 3 : 3 : 1	1,06	0.8–0.7
2.	Pc 39 x Garland	76/ 37–73/1 264/ 60–68/1	218	70	14	7	45 : 15 : 3 : 1	1,07	0.8–0.7

Table 51.

Comparison of infection types of *P. graminis* f.sp. *avenae* in *Avena sterilis L.* WYR 343-1, WYR 343-2, cv. Szegedi Korai and Pc 54-1 with genes Pg 4 (Rodney), Pg 13 (Rodney M), Pg 15, Pg 16 and Pg a
(ŠEBESTA, 1986)

Line/Cv.	Isolates of Puccinia graminis avenae									
	23–84	39–84	47–84	56–84	75–84	77–84	90–84	91–84	94–84	96–84
A. sterilis, WYR 343-1	1	1	1	1	1	1	1	1	1	1
A. sterilis, WYR 343-2	2	2	2	2	2	2	2	2	2	2
Szegedi Korai	4	1	1	1	4	4	1	1	1	1
Pc 54-1	2	4	2	2	1	2	2	2	2	2
Pg 4 (Rodney)	4	1	1	1	4	4	1	1	0,	1
Pg 13 (Rodney M)	0–1,	0–1,	0–1,	0–1,	0–1,	0–1,	0–1,	1	0–1,	1
Pg 15	2–3	4	2–3	3	2	2	2	2–3	2–3	3
Pg 16	0–2	1	1	1	1	1	1	1	0–1	1
Pg a	2	1	0–2	0–2	2	2	1	0–2	0–2	0–2

Table 52.

Segregation of F_2 seedlings of the cross Flamingsnova x A. sterilis L., WYR 343 to Austrian isolates 93–84 and 94–84 of *P. graminis* f. sp. *avenae* (ŠEBESTA, 1986)

Cross	Plants				Expected ratio	P
	R	MR	S	n		
1a/84-1	387		132	519	3:1	0.9-0.8
1b/84-4a	239		74	313	3:1	0.7-0.5
1c/84-1	194[a]		79	273	3:1	0.3-0.1
1c/84-6	156[a]		61	217	3:1	0.3-0.1
1b/84-3	190[a]		46	236	13:3	0.8-0.7
1b/84-9	236[a]		62	298	13:3	0.5-0.3
1c/84-2	133[a]		31	164	13:3	1.0-0.95
1c/84-4	161[a]		40	201	13:3	0.7-0.5
1b/84-5	169	13	50	232	12:1: 3	0.7-0.5
1b/84-6	351	24	97	472	12:1: 3	0.5-0.3

Table 53.

Reaction and infection type of cv. Minrus, C. I. 2144 and nearly isogenic line Rodney D (Pg 1) to races of *P. graminis* f. sp. *avenae* (ŠEBESTA, 1980)

Race	Origin/ Year	Minrus		Rodney D	
		Reaction	Infection type	Reaction	Infection type
24	A/1976	R	„0;“	S	4
68	A/1979	R	„0;“	S	4
68	CS/1967	R	„0–2;“	S	3–4
76	A/1979	R	„0;“	R	2
76	CS/1979	R	„0;“	R	2
A 1	A/1976	R	„0;“	R	2
A 2	A/1976	R	„0–2;“	S	4
A 3	A/1978	R	„0;“	R	2
A 4	A/1978	R	„0;“	S	4
CS 1	CS/1979	R	„0;“	S	4

Table 54.

Segregation of F_2 oat seedlings to *P. graminis* f. sp. *avenae* races in the cross Minrus x Rodney D (ŠEBESTA, 1980)

Cross	Race	Plants			Expected ratio	P
		R	S	n		
661 a, b	A 1	209	90	299	45:19	0.9-0.8
662 a, 3a, b	A 2	239	110	349	45:19	0.5-0.3
145/79-I	CS 1	196	78	274	45:19	0.8-0.7
145/79-II	CS 1	296	131	427	45:19	0.7-0.5

Table 55.

The cross Minrus x Rodney D. Reaction of F_3 lines to *P. graminis* f. sp. *avenae*, isolate CS 1 (ŠEBESTA, 1980)

Cross	Race	Plants			Expected ratio	P
		R	Segr.	S		
663 c/77	CS 1	6	38	16	7:38:19	0.9-0.8

Table 56.

Reaction and the infection type of the cv. Jostrain, C. I. 2660 and nearly isogenic line Rodney E (Pg 3) to races of *P. graminis* f. sp. *avenae* (ŠEBESTA, 1980)

Race	Origin/ Year	Jostrain		Rodney E	
		Reaction	Infection type	Reaction	Infection type
71	P/1978	R	0,	R	„0-1;“
76	A/1979	R	0,	R	„0-1;“
77	A/1977	M	X	S	4
A 1	A/1976	R	0,	R	„0-1;“
A 2	A/1976	R	„0-1;“	S	4
A 3	A/1978	R	„0-2;“	M	X
A 4	A/1978	R	0,	R	„0;“
P 1	P/1978	R	0,	R	„0;“

A = Austria
P = Poland

Table 57

Groupings of the oat powdery mildew resistance and differential cultivars used to identify virulence in isolates od *Erysiphe graminis* f. sp. *avenae* (ŠEBESTA, et al. 1997)

OMR* group	Differential cultivar	Gene designation	Source of resistance
0	Milford		
1	Manod		*Avena byzantina* variety Red Algerian
2	Cc4146	Eg-1	*A. sativa* x *A. ludoviciana* (natural hybrid)
3	9065Cn 6/3/74	Eg-3	*A. ludoviciana* (Cc4346)
4	Cc6490	Eg-4	*A. barbata* (Cc4897)
(5)†	7718Cn 20/3/1		Cc4146

Oat Mildew Resistance Group
† Proposed by Jones (1982)

Table 58.

Host reaction scale for the assessment of virulence pattern of *E. graminis* f. sp. *avenae* or specific resistance (ŠEBESTA et al. 1997)

0	*immune* – no visible symptoms
0n/0N	*highly resistant* – small or large necrotic areas respectively
1	*resistant* – mycelium visible with slight sporulation and chlorosis or necrosis
2	*moderately resistant* – moderate pustules with chlorosis or necrosis
3	*moderately susceptible* – large pustules with slight chlorosis or necrosis
4	*highly susceptible* – large pustules, no chlorosis or necrosis

Table 59

Incidence of oat powdery mildew (*Erysiphe graminis* f. sp. *avenae*) in Europe in 1988–1994 as recorded at some localities of the European oat disease nursery (ŠEBESTA et al., 1997)

Country	Locality	Year						
		1988	1989	1990	1991	1992	1993	1994
Austria	Vienna	++	++	++				
	Drauhofen					+++		
	Edelhof							+++
	St. Donat					+++		+++
Bulgaria	Sadovo					++		
Czech Republic	Bystrice	+		+	++	+	++	
	Krukanice				+++	+++	+++	+++
France	Le Rheu		++	+++	+++	+++	+++	
Germany	Petkus	+++	+	+++				
	Quedlinburg		+++	+++	+++			
	Salzmunde			+++	++			
	Gross Lusewitz					+++	+++	
Greece	Thessaloniki			+++	+++	+++	+	++
Holland	Ulrum		+++					
Italy	Badia Polesine			+++				
	Rome						++	++
Norway	Stjordal			++				
Poland	Strzelce							+
	Wielopole						++	
Slovakia	Pstrusa							++
Spain	Madrid					++		
Sweden	Svalov						++	
United Kingdom	Aberystwyth	+++	+++	+++	+++	+++	+++	+++
Yugoslavia	Kragujevac	+	++	++		+++		++

+ = low occurrence ++ = moderate occurrence +++ = high occurrence

Table 60.

Disease resistance index to oat powdery mildew (*Erysiphe graminis* f.s p. avenae) in 67 oat genotypes included in the European oat disease nursery in 1988–1994 (after adjusting to the same number of evaluations) (ŠEBESTA et al. 1997)

Genotype	DRI	No. of evaluations				Genotype	DRI	No. of evaluations			
		R†	MR	MS	S			R	MR	MS	S
APR 166	190	19	5	–	–	Minrus	93	8	11	7	9
APR 122	173	16	4	2	–	Pc 54-1	93	12	14	18	6
Cc 6490	171	32	11	3	1	Rodney ABDH	93	13	10	13	8
Cc 4761	165	33	11	3	3	Pc 58	92	13	12	14	9
OM 1621	160	9	4	2	–	OA 503-1	91	7	7	6	7
Roxton	150	24	18	7	1	IL 86-1158	90	13	8	10	11
Cc 4146	149	29	11	9	1	KR 288/73L/569	89	12	9	14	7
Melys	140	9	2	3	1	Rodney A	89	14	11	14	11
Maelor	136	24	15	11	2	Pc 60	87	13	11	14	11
Orlando	134	20	10	9	2	IL 86-4189	84	9	12	13	9
Cc 3678	131	16	11	3	7	IL 85-6467	85	13	6	15	7
Pg 15	131	17	17	13	5	Pc 62	84	9	17	17	9
OM 1387	129	6	4	4	–	Pc 63	80	10	9	15	8
Avena sterilis 2648	128	12	13	7	2	IL 86-4467	79	9	9	11	11
Pg 16	127	17	14	11	8	KR 3813/73	74	6	14	10	13
Kasadra	125	3	1	2	–	Pc 50-4	73	9	13	16	13
Mostyn	125	21	13	8	7	Pc 50-2	72	9	12	14	15
Pc 39	122	20	15	10	6	IL 86-5698	71	9	7	12	12
Pg a	116	21	12	14	4	Pc 38	70	7	14	5	24
Rodney E	113	13	9	7	6	Vermiou	70	1	1	3	–
Adam	107	3	1	2	1	Jostrain	67	6	8	13	9
KR 8122	107	3	1	2	1	Pan	66	5	15	10	19
Manod	106	14	9	12	4	IL 86-6404	64	6	9	11	14
Rodney M	106	18	12	14	7	Pen^2 x CAV 1376	59	5	10	13	12
Maldwyn	102	16	12	17	4	Pc 67	57	6	8	16	12
Garland	101	17	11	10	12	Pirol	56	5	9	12	16
KR 9046	100	2	2	1	2	Pc 68	52	5	8	13	16
OA 504-5	100	7	10	8	4	Pc 48	51	2	12	12	17
Rodney D	100	10	10	11	4	Pc 59	51	4	7	12	13
Rodney F	100	12	6	7	8	Zlatak	50	1	1	2	3
Rodney H	100	14	14	14	7	Pc 64	43	3	8	10	21
Pc 61	99	9	17	17	9	Pc 55	35	2	7	15	17
IL 85-2069	98	15	6	12	7	Pc 56	33	3	6	21	15
OA 504-6	98	3	13	8	2						

† R = resistant, MR = moderately resistant, MS = moderately susceptible and S = susceptible.

Table 61.

Oat powdery mildew virulence (OMV) and resistance (OMR) groups identified in Europe up to 1994.
(ŠEBESTA et al., 1997)

		Virulence groups of powdery mildew (OMV)							
		0	1	1,2,5	1,3	1,2,3,5	1,2,4,5	1,3,4,5	1,2,3,4,5
OMR Group	UK Race No. Differentials	1	2	3	4	5	6	8	7
0	Milford	+	+	+	+	+	+	+	+
1	Manod	–	+	+	+	+	+	+	+
2	Cc4146	–	–	+	–	+	+	–	+
3	9065 Cn	–	–	–	+	+	–	+	+
4	Cc6490	–	–	–	–	–	+	+	+
5	7718 Cn	–	–	+	–	+	+	+	+

+ = virulent – = avirulent

Table 62. **Sources of oat resistance to powdery mildew (*Erysiphe graminis* f. sp. *avenae*) (ŠEBESTA et al., 1997)**

Accession/Cv	Species	OMR Group?†	Remarks	References
CAV 6773, 6794	A. atlantica (2x = 14)			ŠEBESTA (1990), HERRMANN & RODERICK (1996)
Cc 3678	A. hirtula (2x)		Gene Eg-2	JONES & GRIFFITHS (1952); SIMONS et al. (1978)
CAV 5264	A. macrostachya (2x)			HOPPE & POHLER (1991)
Qu 8	A. longiglumis (2x)			HERRMANN & RODERICK (1996)
Cc 4852	A. ventricosa (2x)			JONES et al. (1982)
Cc 6557	A. prostrata (2x)			JONES et al. (1982)
AVE 128, 264, 488	A. strigosa (2x)			HERRMANN & RODERICK (1996)
S.171	A. strigosa x A. brevis (2x)			JONES & RODERICK (1986)
Cc 4093	A. strigosa s. sp glabrota (2x)			JONES et al. (1982)
Cc 6558	A. murphyi (4x = 28)			JONES et al. (1982)
Cc 4146	A. sativa/A. ludoviciana (6x)	OMR 2	Gene Eg-1	JONES & GRIFFITHS (1952); SIMONS et al. (1978)
Cc 4761	A. byzantina (6x)		Selection from Creme	JONES (1975)
APR 122/166	A. sativa (6x)		Derived from A. eriantha CAV 0128	HOPPE (1991); HOPPE & KUMMER (1991)
AV 1860, Cc 6490	A. sativa	OMR 4	Translocation lines, resistance from A. barbata Cc4897 Gene Eg-4.	THOMAS et al. (1975, 1980); 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 (1984)
CAV 2648	A. sterilis	OMR 1+2		ŠEBESTA et al. (1987)

Table 62 (Continued 2)

Accession/Cv	Species	OMR Group?†	Remarks	References
Cc 4346 & 4347	A. sterilis var. ludoviciana (6x)	OMR 3	Incorporated into 9065Cn. Gene Eg-3	LAWES & HAYES (1965); HAYES & JONES (1966); SIMONS et al. (1978)
CAV 2107	A. byzantina (6x)			ŠEBESTA (1990)
CAV 3891, CAV 3889	A. occidentalis (6x)			ŠEBESTA et al. (1987), HERRMANN & 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 (1986), 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 (6x = 42)		Derived from A. prostrata addition line	THOMAS & GRIFFITHS (1985), NAQUI (1990)
93–2–4	A. sativa		High level of APR	JONES & RODERICK (1986)
Bage sel Klein	A. byzantina (6x)		High level of APR	JONES & RODERICK (1986)
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, JONES & CLOTHIER (1995)

Table 63.

Areas under the mildew progress curves of 38 spring oat genotypes and lines. (HERMANN, RODERICK, 1996, ŠEBESTA et al., 1997)

Cultivar/line	AUMPC
Cc6490	0 [a]
Creme	15,0 [ab]
93–2–4	86,3
S. 171 (A. Strigosa)	94,3 [abc]
Bage sel Klein	141,0 [abcd]
OM 1711	162,4 [bcde]
Manod	165,8 [bcde]
Pc 54	179,2 [cdef]
Rouge d'Algerie	202,6 [cdefg]
OM 1621 (Mostyn x Maldwyn line)	211,5 [cdefg]
Ruakura	221,3
Roxton	221,5 [cdefg]
OM 2084	226,5 [cdefg]
Maelor	257,3 [defgh]
07772CnII (Menai x Minn. 72001-65)	262,5 [defgh]
OM 1387 (Selma x Maldwyn line)	265,1 [defgh]
Saia (A. Strigosa)	270,6 [defgh]
Cc6989 (A. Prostrata addition line)	298,0 [defghi]
Minn. 72001–65	318,5 [efghij]
Minn. 721011	333,6 [fghijk]
Forward	350,3 [ghijk]
Leanda	356,0 [ghijk]
OM 2086	413,3 [hijkl]
Maldwyn	436,0 [hijkm]
Saladin	438,3
Mostyn	465,3
Milo	465,3
Mutant 6 (selection from cv. Selma)	476,5 [jklm]
Pc 39	486,3 [klm]
Dula	547,7 [lmn]
Milford	576,7 [mno]
Rollo	581,5 [mno]
Trafalgar	673,8 [no]
Maris Tabard	703,8 [no]
Cabana	720,0 [o]
Avalanche	904,0 [p]
Selma	1115,8 [q]

Table 64.

Areas under the mildew progress curves (AUMPC) for a selection of ten cultivars or lines of spring oats with different levels of APR and five advanced lines or new cultivars. (HERMANN, RODERICK, 1996, ŠEBESTA et al., 1997)

Cultivar/line	AUMPC
Cc7422 (Pc 54)	0,24 a*
Roxton	3,21 b
OM 1711	3,29 b
OM 1387	4,20 bc
Maelor	6,34 cde
Forward	6,36 cde
Leanda	6,44 cde
Maldwyn	6,81 def
Saladin	7,88 ef
Selma	13,57 g
10352Cn	5,32 bcd
Aberglen	5,95 cde
Melys	6,49 cde
10026Cn	7,00 def
Valiant	8,86 f

* means followed by the same letter are not significantly different (P = 0.05) according to Duncan's Multiple Range Test.

Table 65.

Incidence of *Septoria avenae* in the European oat disease nursery in 1990–1993 as recorded by the national cooperators (ZWATZ et al. 1994)

Country	Locality	1990	1991	1992	1993
Austria	Drauhofen			+++	++
	Zwettl			+	+++
	Fuchsenbigl			++	++
	Petzenkirchen	++	+++		++
	St. Donat				
Bulgaria	Sadovo				
Czech Republic	Bystrice n. P.				
	Krukanice				
	Praha				
Finland	Anttila				
France	Rheu				
Germany	Berthelsdorf	+			
	Gross Lusewitz				++
	Quedlinburg		+		
	Weihenstephan				
Greece	Thessaloniki				
Great Britain	Aberystwyth				
Italy	Badia Polesine	++			
	Rome				
Poland	Danko		+++	++	+
	Choryn	++			
	Polanowice				
	Wielopole		+++	+++	+++
Russia	Nemchinovka				
Slovakia	Pstrusa				
Sweden	Svalof				
Yugoslavia	Kragujevac				

Table 66.

Resistance index of *Septoria avenae* in oat genotypes included into the EODN in the period 1990-1993. (ZWATZ et al., 1994)

Line/Cultivar	Resistance index	Number of Evaluations		Reaction of the genotype to other diseases
		R	MR	
Avenae sterilis				
CAV 2648	48	9	4	R to Pc and Eg
Cc 4761	47	8	5	R to Eg
Pc 55	46	4	10	R to Pc
Pc 67	45	6	7	R to Pc
Pc 50-2	43	4	9	R to Pc
Pc 60	43	4	9	R to Pc
Pc 50-4	42	6	6	R to Pc
Pc 54	40	4	8	R to Pc, Pg, Eg
IL 86–6404	40	7	4	T to BYDV
Garland	38	5	6	R to Pc, Eg
Pc 58	38	5	6	R to Pc
Pc 48	38	5	6	R to Pc
Cc 6490	37	7	3	R to Eg
Pc 56	36	3	8	R to Pc
Cc 3678	35	5	5	R to Eg
IL 86–4189	34	4	6	R to Pc, T to BYDV
IL 86–1158	33	3	7	R to Pc, T to BYDV
Pg 15	33	3	7	R to Pg
Pc 68	32	5	4	R to Pc
Pen2x CAV1376	32	2	8	R to Pc
IL 85–2069	30	3	6	R to Pc, T to BYDV
IL 85–6467	30	6	2	R to Pc, T to BYDV
Orlando	30	3	6	R to Pc, Eg
Pc 39	29	2	7	R to Pc, Eg
Manod	29	5	3	R to Eg
Pc 61	28	4	4	R to Pc
IL 86–5698	26	2	6	T to BYDV
Pirol	26	2	6	R to Pc
Jostrain	26	2	6	R to Pg
Pg a	25	1	7	R to Pg

Table 66 (Contd. 2)

Line/Cultivar	Resistance index	Number of Evaluations		Reaction of the genotype to other diseases	
		R	MR		
IL B6–4467	25	4	3	T to BYDV	
KR 3813 (CZ)	25	4	3	R to Pc, Pg	
Cc 4146	25	4	3	R to Eg	R to Eg
Mostyn	24	3	4	R to Eg	
Pg 16	24	3	4	R to Pg	APR to Eg
Maldwyn	24	3	4	APR to Eg	
Pc 62	22	4	2	R to Pc	
Minrus	20	2	4	R to Pg	
Rodney F	20	2	4	R to Pg	R to Pg
Pc 63	19	1	5	R to Pc	
Maelor	19	1	5	APR to Eg	R to Pc
Pc 64	17	2	3	R to Pc	
Rodney B	17	2	3	R to Pc, Pg	
OA 503–1	17	2	3	R to Pg	T to BYDV
Rodney A	17	2	3	R to Pg, Pc	
Rodney M	16	1	4	R to Pg, Pc	
Pc 38	16	1	4	R to Pc	
Pan	16	1	4	R to Pc	
Rodney ABDH	15	0	5	R to Pg, Pc	
KR 288/73L (CZ)	13	1	3	R to Pg, Pc	R to Pg, Pc
Rodney H	11	2	1	R to Pg, Pc	
Rodney D	10	1	2	R to Pg, Pc	R to Pc, Pg, Eg

Resistance index = sum of R and MR evaluation (R = 4, MR = 3, MS = 2, S = 1)
R = resistant, M = moderately resistant, MS = moderately susceptible, S = susceptible,
T = tolerant
APR = Adult plant resistance
Pc = *Puccinia coronata* avenae
Pg = *Puccinia graminis* avenae
Eg = *Erysiphe graminis* avenae
BYDV = Barley Yellow Dwarf Virus

Table 67

Incidence of *Pyrenophora avenae* in the European oat disease nursery in 1990–1993 (ŠEBESTA et al., 1995)

Year	Country	Locality	The level of incidence
1990	Czech Republic	Kromeriz	weak-moderate
	Poland	Choryn	strong
	Russia	Nemchinovka	moderate
	Sweden	Svalof	moderate
1991	Austria	Vienna	moderate-strong
	Czech Republic	Kromeriz	moderate
	Finland	Hankkija	weak
	Germany (East)	Salzmunde	moderate
	Poland	Danko	moderate-strong
		Wielopole	weak-moderate
1992	Austria	St. Donat	moderate
	Italy	Rome	moderate-strong
	Poland	Wielopole	strong
1993	Austria	Drauhofen	moderate
		St. Donat	moderate-strong
	Finland	Anttila	strong
	Italy	Rome	weak
	Poland	Wielopole	weak
	Russia	Nemchinovka	strong

Table 68.

Resistance index of oat genotypes to *Pyrenophora avenae* in the EODN trials in the period 1990–1993 (ŠEBESTA et al., 1995)

Line/Cultivar	Resistance index	Number of Evaluations		Reaction of the genotype to other diseases
		R	MR	
IL68-1158	64	13	4	R to Pc, T to BYDV
IL85-6467	61	13	3	R to Pc, T to BYDV
IL86-4189	57	12	3	R to Pc, T to BYDV
Maldvin	56	11	4	APR to Eg
Manod	56	8	8	R to Eg
Cc3678	54	12	2	R to Eg
Pc 61	52	10	4	R to Pc
Pc 60	52	7	8	R to Pc
IL85-2069	51	12	1	R to Pc T to BYDV
IL86-6404	51	9	5	T to BYDV
Cc4761	50	11	2	R to Eg
Pc 67	50	8	6	R to Pc
Pc 58	49	7	7	R to Pc
IL86-5698	49	10	3	T to BYDV
Orlando	48	9	4	R to Pc, Eg
Pg 15	47	8	5	R to Pg
Pc 59	46	7	6	R to Pc
Pc 50	46	7	6	R to Pc
Rodney A	45	6	7	R to Pg, Pc
Pg 16	45	9	3	R to Pg
Cc6490	45	6	7	R to Eg
Jostrain	43	7	5	R to Pg
Garland	42	9	2	R to Pg, Pc
Pc 50-2	42	3	10	R to Pc
Pc 55	42	3	10	R to Pc
Roxton	42	6	6	APR to Eg
KR3813/73	41	8	3	R to Pc, Pg
Pc 39	41	5	7	R to Pc, APR to Eg
Pc 56	41	5	7	R to Pc
Pc 50-4	40	7	4	R to Pc
Mostyn	39	6	5	R to Eg
Rodney D	38	8	2	R to Pg, Pc
Pc 68	37	7	3	R to Pc
A. sterilis CAV2648	37	7	3	R to Pc, Eg
Rodney F	36	6	4	R to Pg, Pc
Pc 63	36	6	4	R to Pc
Rodney H	36	6	4	R to Pg, Pc
Minrus	35	5	5	R to Pg, Pc

Table 68 (Contd. 2)

Line/Cultivar	Resistance index	Number of Evaluations		Reaction of the genotype to other diseases
		R	MR	
IL86-4467	34	4	6	T to BYDV
Pc 62	33	6	3	R to Pc
Pc 64	32	5	4	R to Pc
KR288/73L/569	32	5	4	R to Pg, Pc
Pc 48	31	4	5	R to Pc
Pg a	31	4	5	R to Pg
Rodney ABDH	30	6	2	R to Pg, Pc
Maelor	29	5	3	R to Eg
OA 504-5[1/]	29	5	3	R to Pc
OA 503-1[1/]	28	7	–	R to Pg, Pc
APR 122[2/]	28	4	4	APR to Eg
Rodney B	28	7	–	R to Pg, Pc
Rodney M	27	6	1	R to Pg, Pc
Pan	27	6	1	–
Rodney E	26	5	2	R to Pg, Pc
Pc 38	26	5	2	R to Pc
Pc 54	25	4	3	R to Pc, Pg, Eg
Cc4146	24	3	4	R to Eg
Pen²x CAV1376	23	2	5	R to Pc
Pirol	22	4	2	APR to Eg
APR 166[2/]	22	4	2	APR to Eg
Melys[3/]	21	3	3	APR to Eg T to BYDV
OA 504-6[1/]	19	4	1	R to Pc
OM1621[3/]	16	4	–	R to Eg
OM1387[3/]	12	3	–	R to Eg

[1/], [2/], [3/] including into the EODN trials since 1991, 1992 and 1993, respectively
Resistance index = sum of R and MR evaluation (R = 4, MR = 3, MS = 2, S = 1)
R = resistant, M = moderately resistant, MS = moderately susceptible, S = susceptible,
T = tolerant
APR = Adult plant resistance
Pc = *Puccinia coronata* avenae
Pg = *Puccinia graminis* avenae
Eg = *Erysiphe graminis* avenae
BYDV = Barley Yellow Dwarf Virus

Table 69.

Severity of *Pyrenophora avenae* on leaves and panicles in oats tested in the RICP in Prague in 1992 (ŠEBESTA et al., 1994)

Cv. / line	Leaves		Panicles
	s	vs	0–4
KR 89–18	0,10	0,32 a	0,33 a
KR 8122	0,15	0,38 a	0,20 a
KR 9046	0,15	0,38 a	0,58 abc
Auron	0,23	0,45 ab	1,17 abcde
Explorer	0,23	0,45 ab	0,50 abc
Zlatak	0,32	0,50 ab	0,75 abc
Adam	0,28	0,51 abc	0,25 a
David	0,28	0,51 abc	0,42 ab
Wiesel	0,28	0,51 abc	1,67 cdefg
Ardo	0,28	0,51 abc	0,92 abc
Fuchs	0,27	0,52 abc	0,83 abc
KR 9478	0,37	0,58 abcd	0,25 a
Trafalgar	0,37	0,58 abcd	0,03 a
Pan	0,42	0,64 abcd	0,50 abc
IL 86–1158	0,42	0,64 abcd	0,58 abc
Hirondel	0,50	0,67 abcd	0,42 ab
Tomba	0,58	0,74 abcde	1,25 abcde
Bulban	0,75	0,86 abcdef	2,50 fgh
IL 86–6467	1,17	0,91 abcdef	1,00 abcd
Arne	1,00	1,00 abcdef	0,15 a
Nero	1,00	1,00 abcdef	0,67 abc
Lars	1,67	1,24 abcdefg	0,67 abc
IL 86–6404	1,67	1,24 abcdefg	0,67 abc
SEMU 3767	2,33	1,49 abcdefgh	0,50 abc
IL 86–4189	2,67	1,63 abcdefgh	1,67 cdefg
Calibre	3,00	1,66 abcdefgh	1,17 abcde
Flamingsnova	3,75	1,69 abcdefgh	0,83 abc
IL 86–4467	4,33	2,07 abcdefgh	0,67 abc
P 5137	7,42	2,13 abcdefgh	1,00 abcd
IL 85–2069	7,67	2,63 bcdefghi	0,83 abc
IL 86–5698	7,33	2,68 cdefghi	0,75 abc
Akiwase	9,00	2,71 defghi	2,83 ghi
Saia	8,33	2,85 efghi	1,00 abcd
Ogle	9,33	2,92 fghi	1,58 bcdef
Walaroo	11,67	3,40 ghij	3,17 hi
Dolphin	13,33	3,64 hij	2,17 defgh
Joycee	20,00	4,45 ij	2,33 efgh
Marloo	30,00	5,34 j	4,00 i

Leaves evaluated according to James scale (s), panicles by means of five category key (0–4)
Values followed by the same letter are not significantly different at P = 0.05 according to Turkey test

Table 70.

Changes in position of oat cultivars and advanced lines according to severity of *Pyrenophora avenae* on leaves and panicles (ŠEBESTA et al., 1994)

Leaves	s	Panicles	0–4
Cv. / line		Cv. / line	
KR 89-18	0,10	Trafalgar	0,03
KR 8122	0,15	Arne	0,15
KR 9046	0,15	KR 8122	0,20
Auron	0,23	Adam	0,25
Explorer	0,23	KR 9478	0,25
Fuchs	0,27	KR 89-18	0,33
Adam	0,28	David	0,42
David	0,28	Hirondel	0,42
Wiesel	0,28	Pan	0,50
Ardo	0,28	SEMU 3767	0,50
Zlatak	0,32	Explorer	0,50
KR 9478	0,37	KR 9046	0,58
Trafalgar	0,37	IL 86-1158	0,58
Pan	0,42	Nero	0,67
IL 86-1158	0,42	Lars	0,67
Hirondel	0,50	IL 86-6404	0,67
Tomba	0,58	IL 86-4467	0,67
Bulban	0,75	Zlatak	0,75
Nero	1,00	IL 86-5698	0,75
Arne	1,00	IL 85-2069	0,83
IL 86-6467	1,17	Fuchs	0,83
Lars	1,67	Flamingsnova	0,83
IL 86-6404	1,67	Ardo	0,92
SEMU 3767	2,33	Saia	1,00
IL 86-4189	2,67	P 5137	1,00
Calibre	3,00	IL 86-6467	1,00
Flamingsnova	3,75	Auron	1,17
IL 86-4467	4,33	Calibre	1,17
IL 86-5698	7,33	Tomba	1,25
P 5137	7,42	Ogle	1,58
IL 85-2069	7,67	Wiesel	1,67
Saia	8,33	IL 86-4189	1,67
Akiwase	9,00	Dolphin	2,17
Ogle	9,33	Joycee	2,33
Walaroo	11,67	Bulban	2,50
Dolphin	13,33	Akiwase	2,83
Joycee	20,00	Walaroo	3,17
Marloo	30,00	Marloo	4,00

Leaves evaluated according to James scale (s), panicles by means of five category key (0–4)

Figures

Fig. 1. Oat plants attacked with crown rust (*Puccinia coronata* Corda f. sp. *avenae* Eriks.)

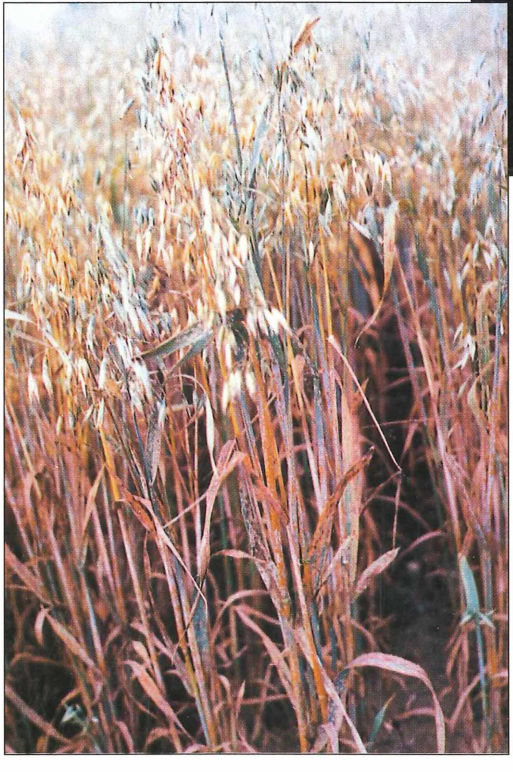


Fig. 2. Oat plants attacked with stem rust (*Puccinia graminis* Pers. f. sp. *avenae* Eriks. et Henn.) (Photo J. Cervenka)

Fig. 3. Oat plants attacked with powdery mildew (*Erysiphe graminis* D. C. f. sp. *avenae* Em. Marchal) (Photo: IGER, Aberystwyth, UK)

Fig. 4. Oat plants attacked with Septoria leaf blight and black stem (*Phaeosphaeria avenaria* G. F. Weber/ O. Eriksson f. sp. *avenaria*)

Fig. 5. Oat plants attacked with Helminthosporium leaf blotch (*Pyrenophora avenae* Ito et Kurib.) (Photo: IGER, Aberystwyth, UK)

Fig. 6. Effect of stem rust attack on oat plants.
Left: Plants infected with *Puccinia graminis* f. sp. *avenae*.
Right: Control plants, without stem rust infection.

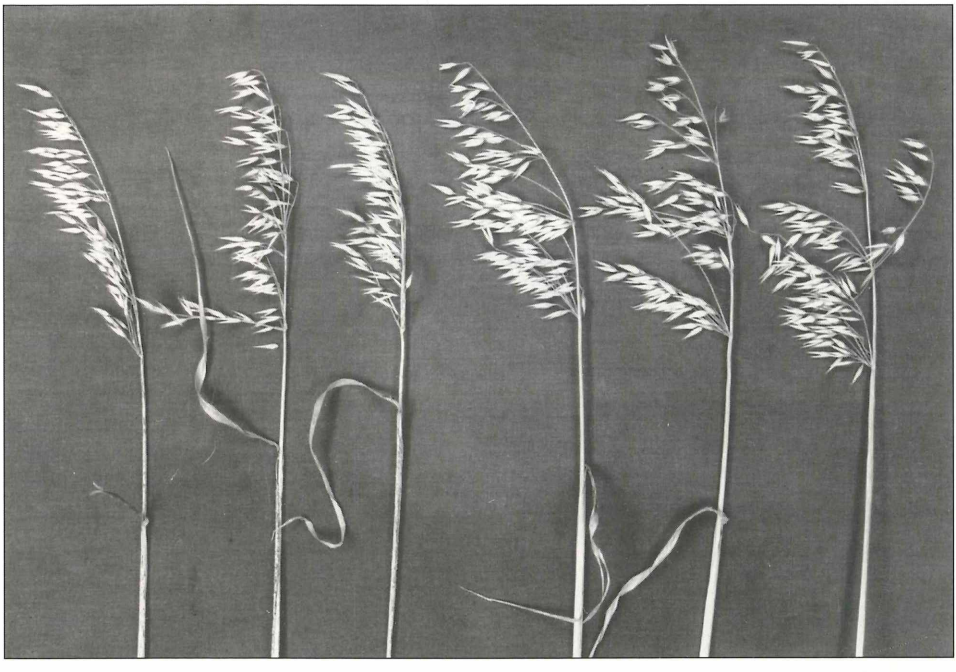


Fig. 7 Effect of stem rust infection on the appearance of oat grains.
Left: Grains from oat plants attacked with stem rust.
Right: Grains from the control plants, without stem rust infection.

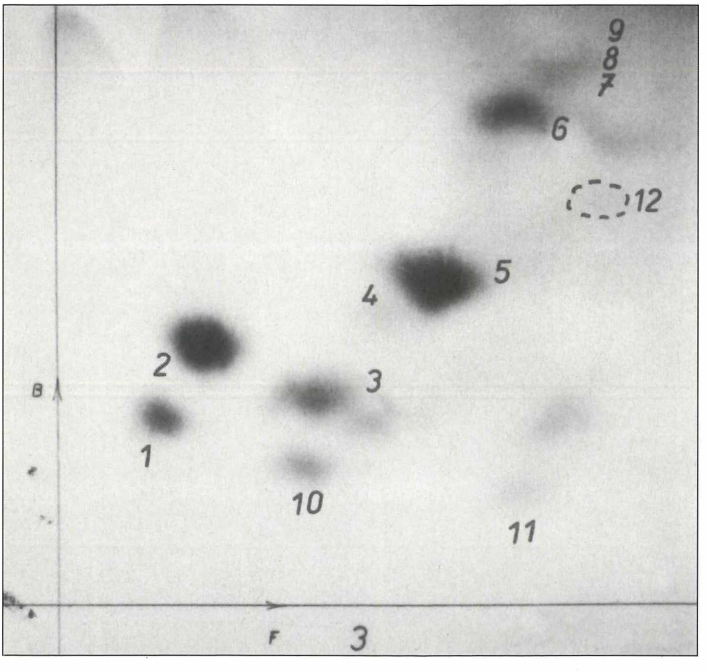


Fig. 8. Chromatogram of free amino acids in grains of the susceptible oat cv. Cesky zluty. Plants attacked with stem rust. (Numerical designation of amino acids – see Table 4b) (KRYZANEK, ŠEBESTA, 1974)

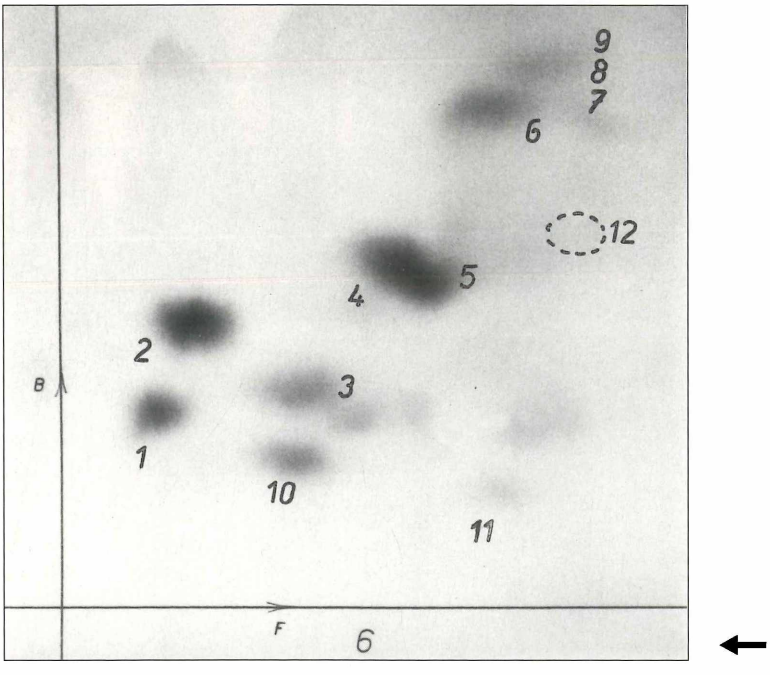


Fig. 9. Chromatogram of free amino acids in grains of the susceptible oat cv. Cesky zluty. Plants attacked with crown rust. (Numerical designation of amino acids – see Table 4b) (KRYZANEK, ŠEBESTA, 1974)

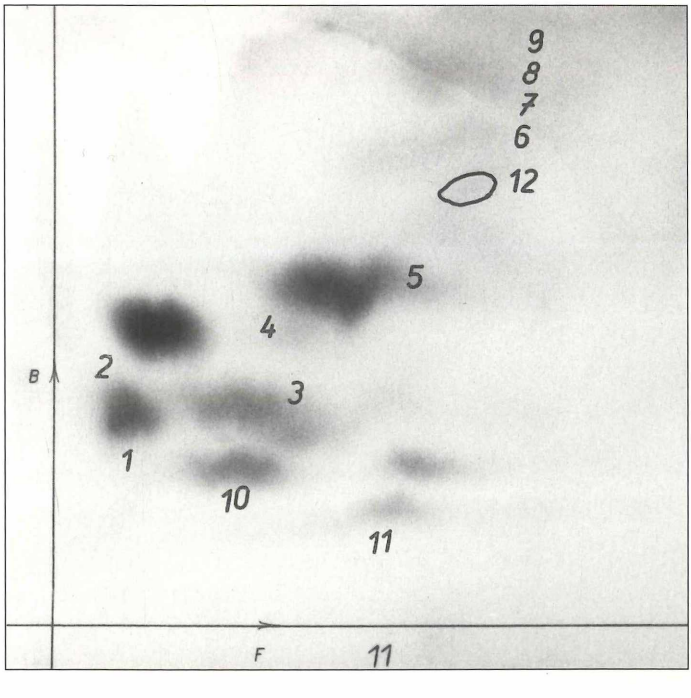


Fig. 10. Chromatogram of free amino acids in grains of the susceptible oat cv. Cesky zluty. Control (unattacked) plants: (Numerical designation of amino acids – see Table 4b) (KRYZANEK, ŠEBESTA, 1974)

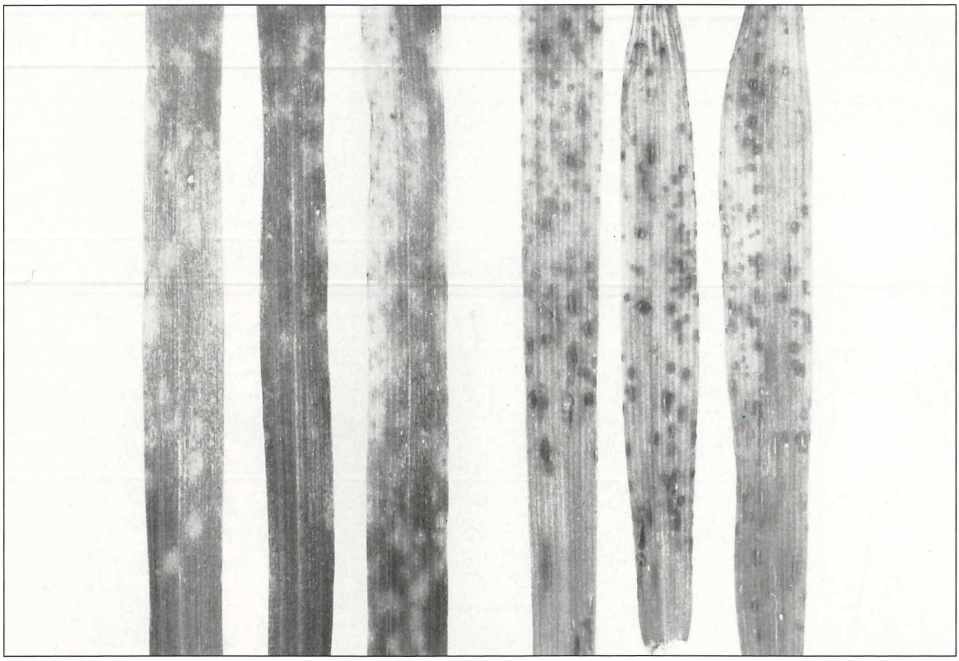


Fig. 11. Seedling reaction of oat to stem rust.
Leaves of resistant (left) and susceptible (right) plants.

Fig. 12. Effect of stem rust infection on the appearance of oat stand.
Left: resistant line (Photo Cervenka, 1998).

Notice to Figs. 13 – 20. Effectiveness of Pc-major genes in isogenic lines and the other sources of crown rust resistance. (behind each line there is the infection type) 0 = immune, 0; = highly resistant, 1 = resistant, 2 = moderately resistant, 3 = moderately susceptible, 4 = highly susceptible).

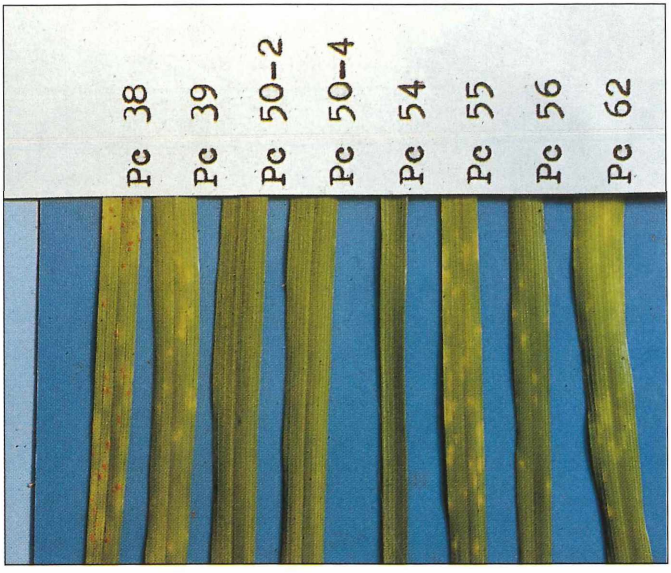
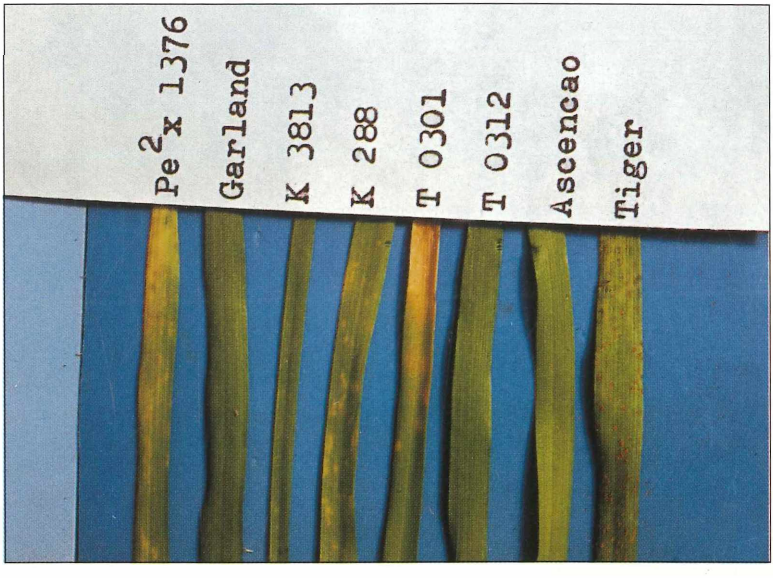


Fig. 13. Tested to the crown rust isolate 23–80/1 from Austria. The first group of genotypes: Pc 38 (4), Pc 39 (0;), Pc 50-2 (0), Pc 50-4 (0), Pc 54 (0), Pc 55 (0;), Pc 62 (0;).

Fig. 14. Tested to the crown rust isolate 23–80/1 from Austria. The second group of genotypes: Pen² x CAV 1376 (0–1;), Garland (0), KR 3813 (0;), KR 288 (0;), TAM 0301 (Pc 58) (0;), TAM 0312 (Pc 59)(0;), Ascencao (Pc 14)(0), Tiger (4).



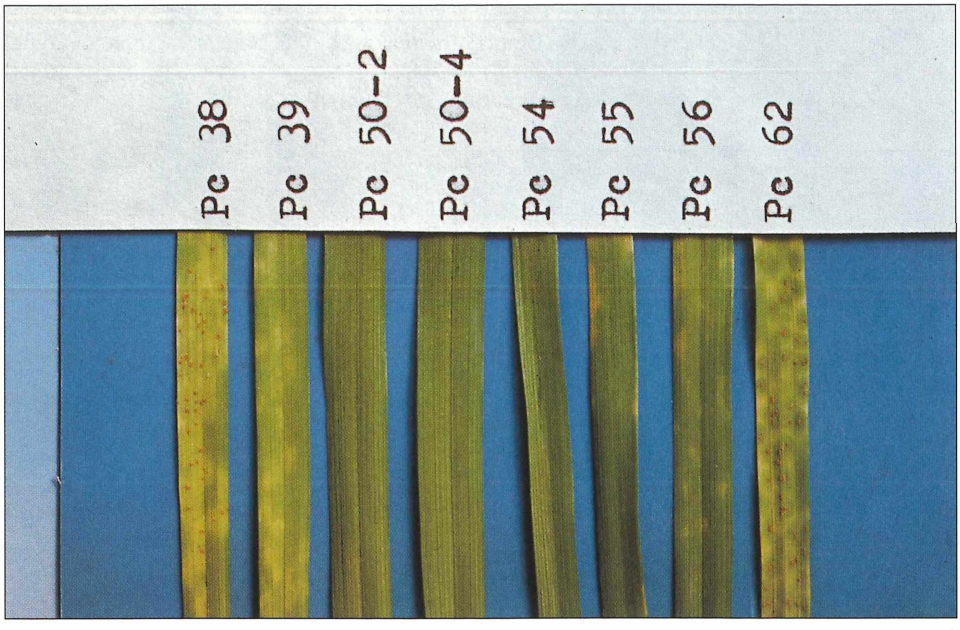
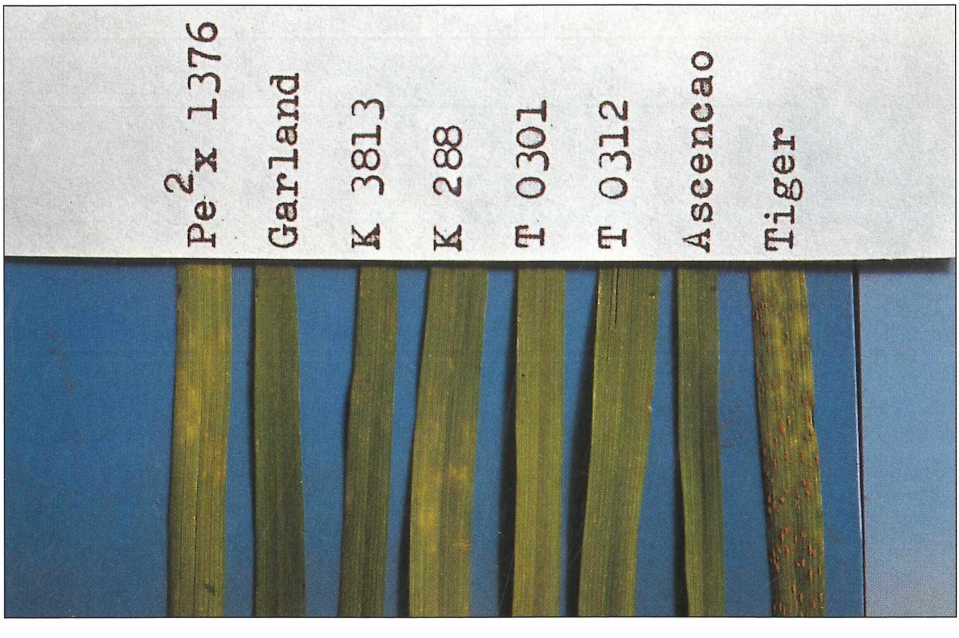


Fig. 15. Tested to the crown rust isolate 29–80/1 from Austria. The first group of genotypes: Pc 38 (0-2;), Pc 39 (0;), Pc 50-2 (0), Pc 50-4 (0), Pc 54 (0), Pc 55 (0;), Pc 56 (0;), Pc 62 (4).

Fig. 16. Tested to the crown rust isolate 29-80/1 from Austria. The second group of genotypes: Pen² x CAV 1376 (0;), Garland (0), KR 3813 (0;), KR 288 (0;), TAM 0301 (Pc 58) (0), TAM 0312 (Pc 59) (0), Ascencao (Pc 14) (0), Tiger (4).



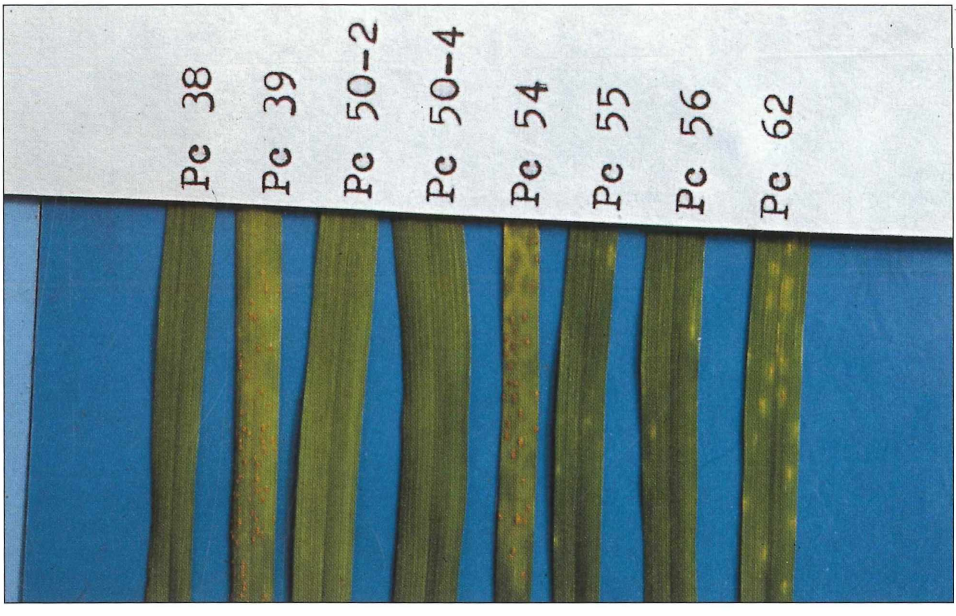
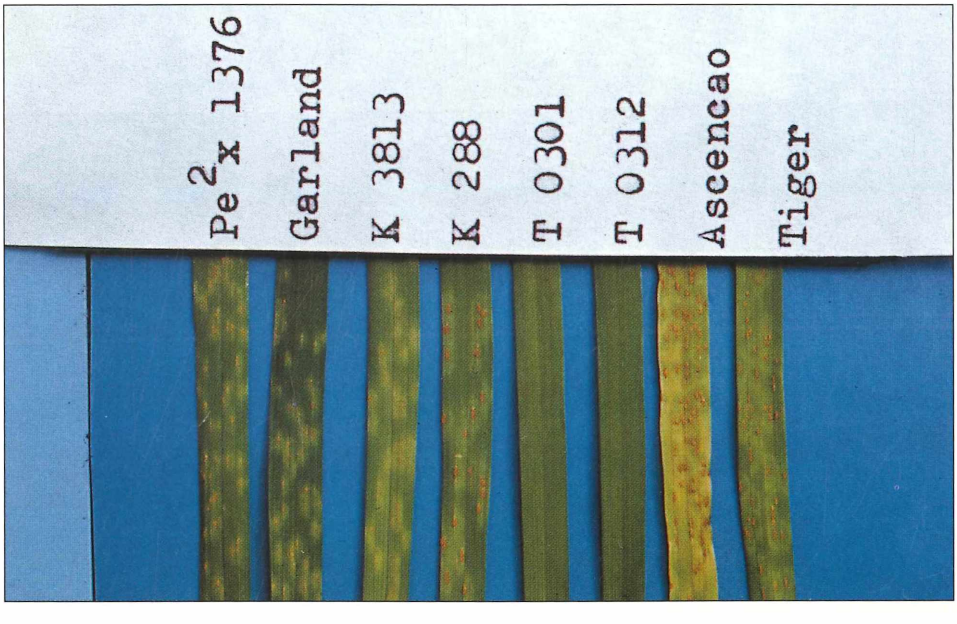


Fig. 17 Tested to the crown rust isolate 30-80/1 from Austria. The first group of genotypes: Pc 38 (0), Pc 39 (3), Pc 50-2 (0), Pc 50-4 (0), Pc 54 (4), Pc 55 (0;), Pc 56 (0;), Pc 62 (0-1;).

Fig. 18. Tested to the crown rust isolate 30–80/1 from Austria.
The second group of genotypes: Pen² x CAV 1376 (1), Garland (1), KR 3813 (1), KR 288 (4), TAM 0301 (Pc 58)(0), TAM 0312 (Pc 59)(0), Ascencao (Pc 14)(4), Tiger (4).



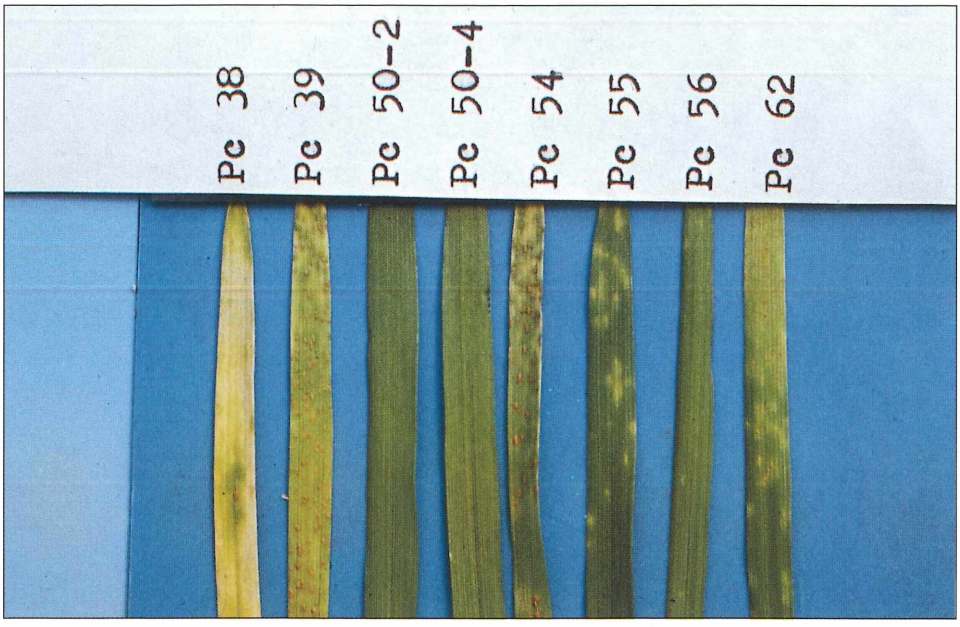


Fig. 19. Tested to the crown rust isolate 62–80 from the Czech Republic.
The first group of genotypes: Pc 38 (0;), Pc 39 (4), Pc 50-2 (0), Pc 50-4 (0), Pc 54 (4), Pc 55 (0;), Pc 56 (0;), Pc 62 (1).

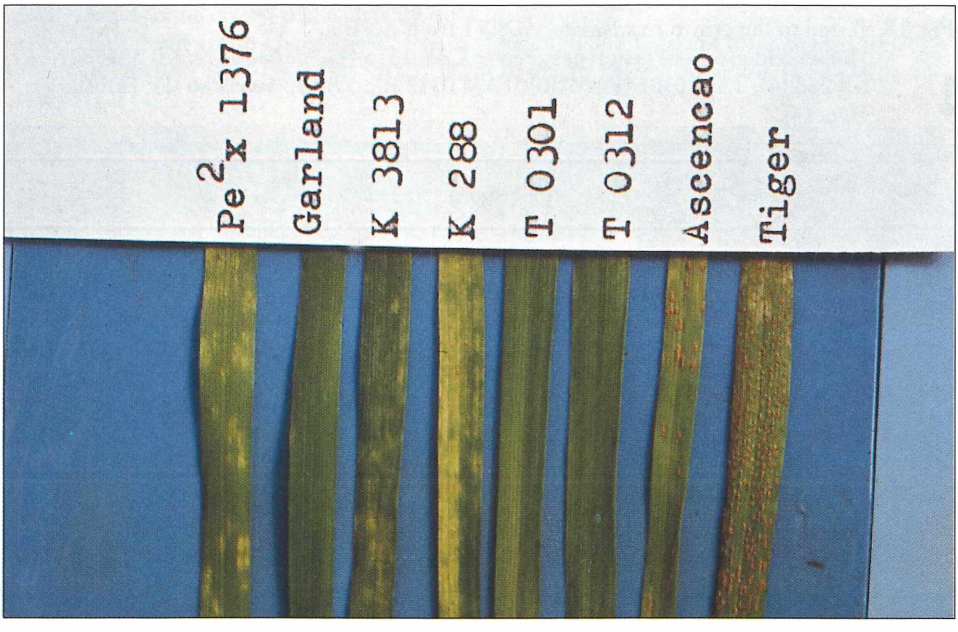


Fig. 20. Tested to the crown rust isolate 62–80 from the Czech Republic.
The second group of genotypes: Pen² x CAV 1376 (1), Garland (0), KR 3813 (0;), KR 288 (0-2;), TAM 0301 (Pc 58)(0;), TAM 0312 (Pc 59)(0), Ascencao (Pc 14)(4), Tiger (4).

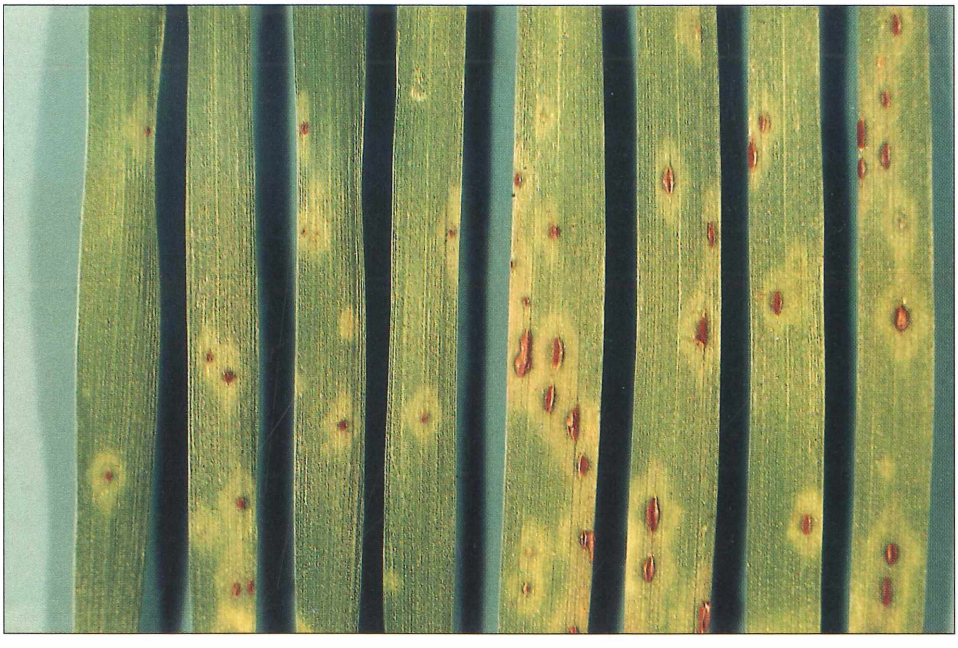
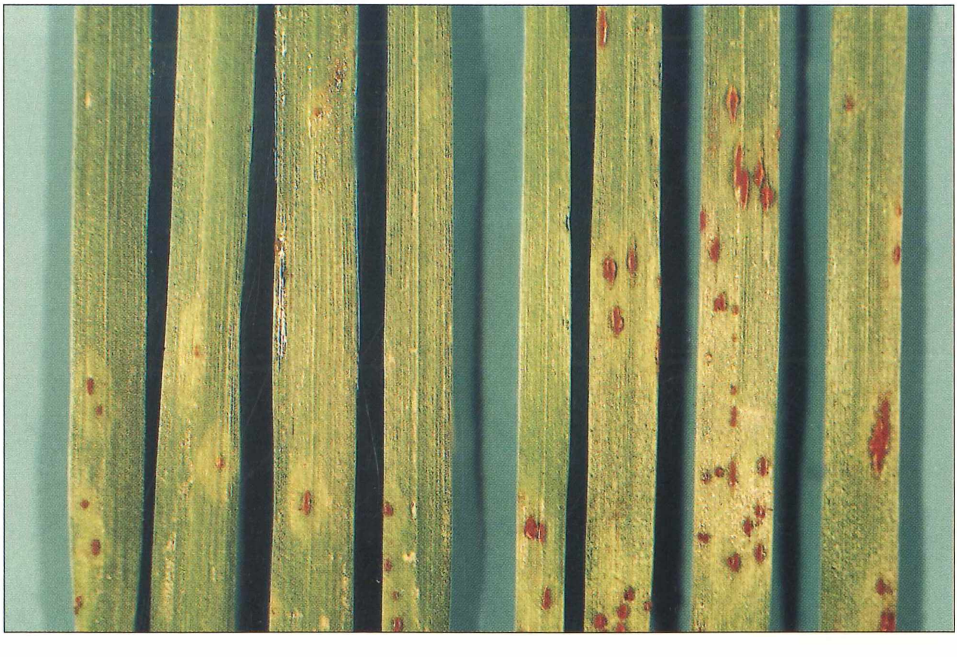


Fig. 21. Resistance of oat to stem rust.
Left: Moderately resistant line Pg 15. Right: Resistant line Pc 54.
Tested to the stem rust isolate 75–84 from the Czech Republic.

Fig. 22. Resistance of oat to stem rust.
Left: Moderately susceptible line Pg 15. Right: Moderately resistant line Pc 54.
Tested to the stem rust isolate 96–84 from Austria.



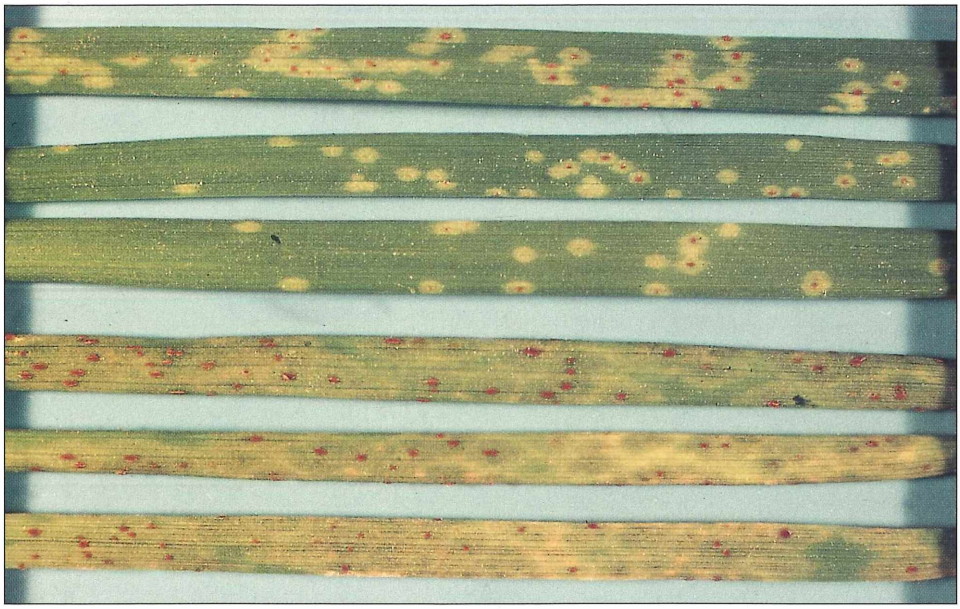
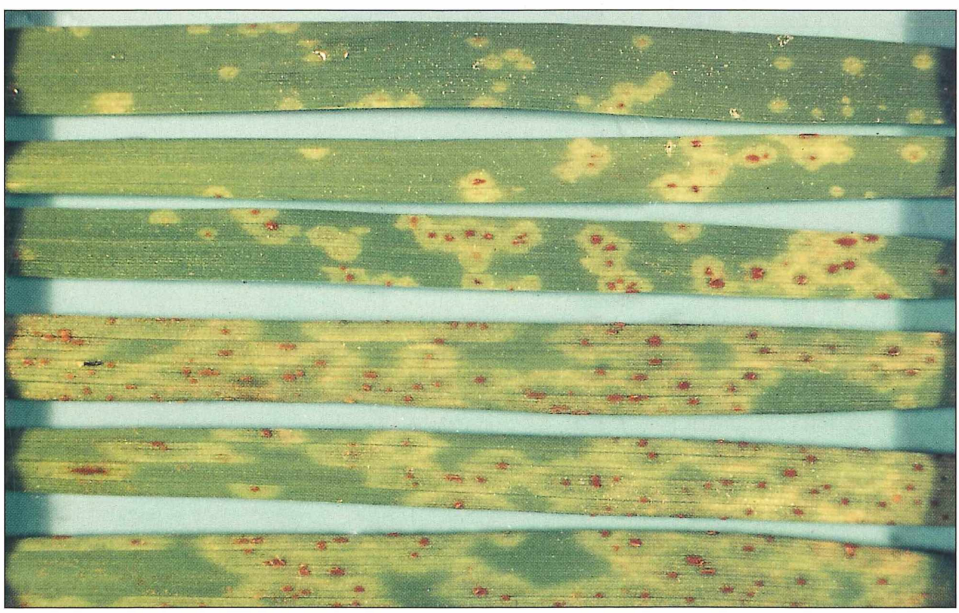


Fig. 23. Resistance of *Avena sterilis* L. accessions WYR 343-1 and WYR 343-2 to stem rust. Left: Resistant reaction of the line WYR 343-1. Right: Moderately resistant reaction of the line WYR 343-2. Tested to the stem rust isolate 39–84 from the Czech Republic.

Fig. 24. Resistance of *Avena sterilis* L. accessions WYR 343-1 and WYR 343-2 to stem rust. Left: Resistant reaction of the line WYR 343-1. Right: Moderately resistant reaction of the line WYR 343-2. Tested to the stem rust isolate 90–84 from the Czech Republic.



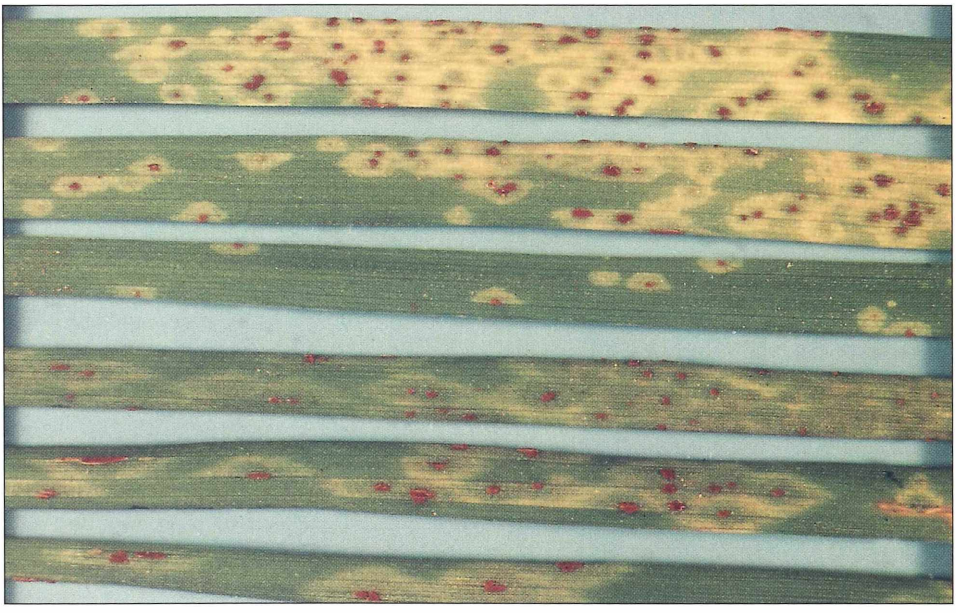
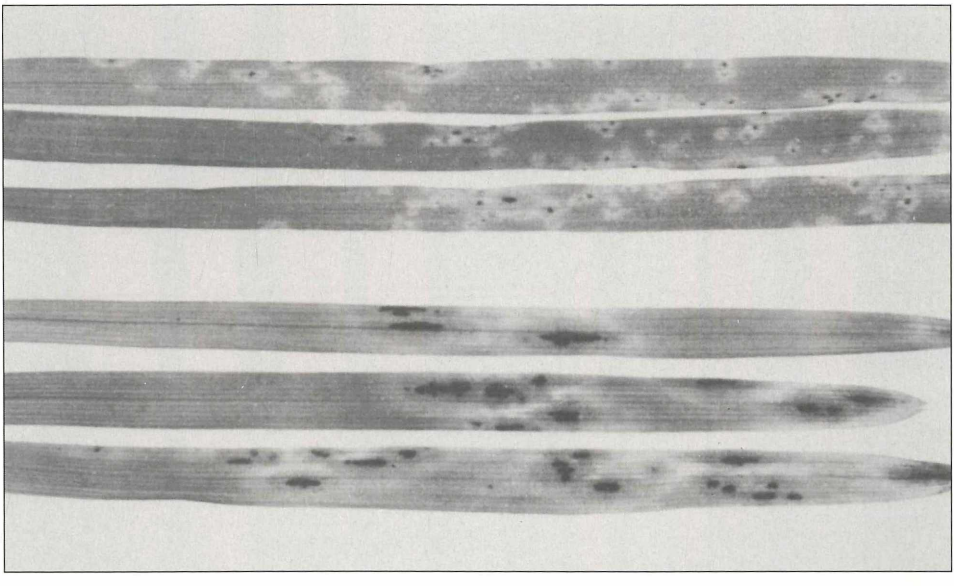


Fig. 25. Resistance of *Avena sterilis* L. accessions WYR 343-1 and WYR 343-2 to stem rust.
Left: Resistant-moderately resistant reaction of the line WYR 343-1.
Right: Uniformly moderately resistant line WYR 343-2.
Tested to the stem rust isolate 91–84 from the Czech Republic.

Fig. 26. Resistance of oat to stem rust.
Left: Resistant reaction of the cv. Minrus.
Right: Susceptible reaction of its derivative, isogenic line Rodney D.
Tested to the oat stem rust race 68 (1), isolate 88-67/3 from the Czech Republic.



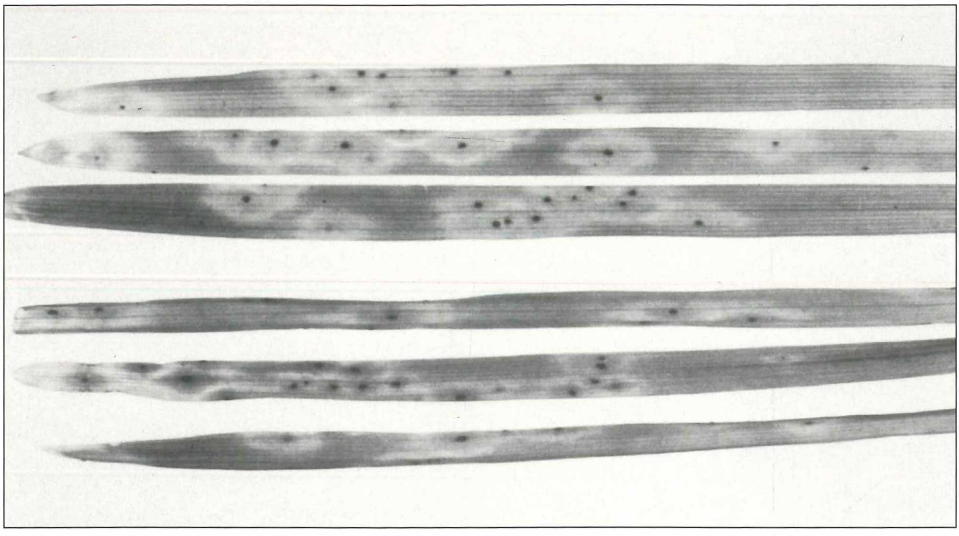
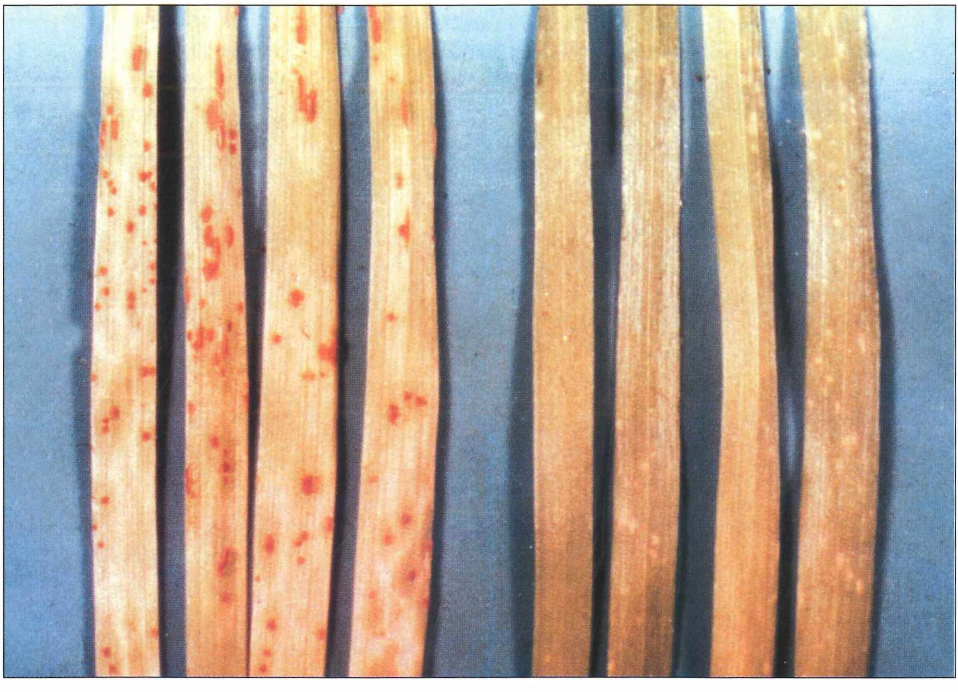


Fig. 27 Reaction of oat to stem rust.
Moderately resistant reaction of the cv. Minrus (Pg 1+) (left) and moderately resistant its derivative isogenic line Rodney D (Pg 1) (right).
Tested to the oat stem rust isolate 55-70/4 (race 76) from Rumania.

Fig. 28. Resistance of oat to stem rust.
Left: Highly resistant reaction of the cv. Minrus.
Right: Susceptible reaction of its derivative isogenic line Rodney D.
Tested to the oat stem rust isolate 116-79 from the Czech Republic.



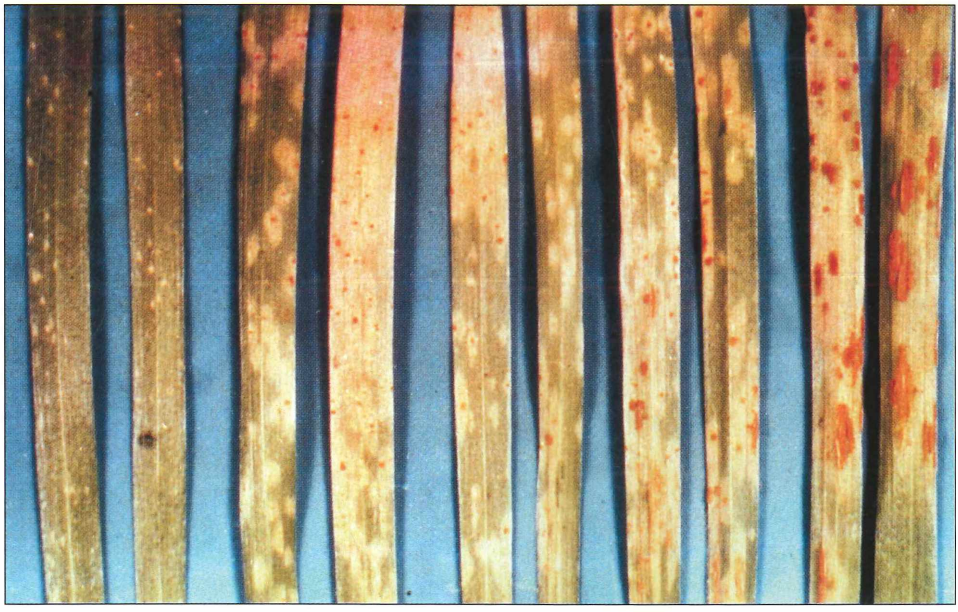


Fig. 29. Resistance of oat to stem rust.
Phenotypes of F_2 from the cross Minrus x Rodney D.
From the left: highly resistant (0;), resistant (1), moderately resistant (0-2;), heterogenic (0–3;) and highly susceptible (4).

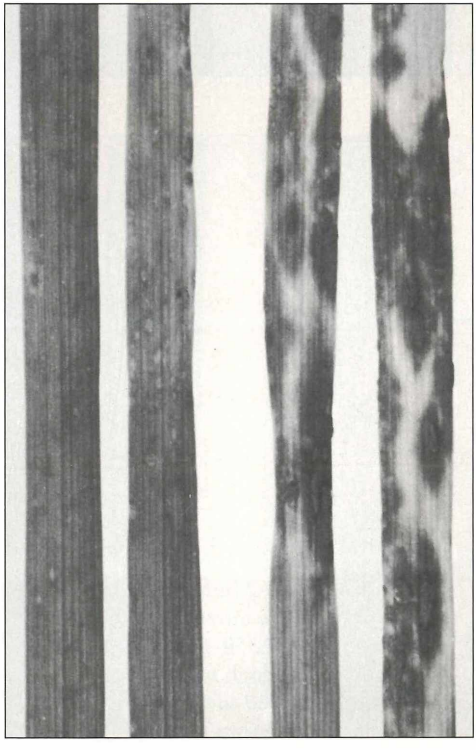


Fig. 30. Resistance of oat to stem rust.
Left resistant cv. Jostrain, right its derivative, susceptible line Rodney E (Pg 3).
Tested to the oat stem rust isolate 21–76/5 from Austria.

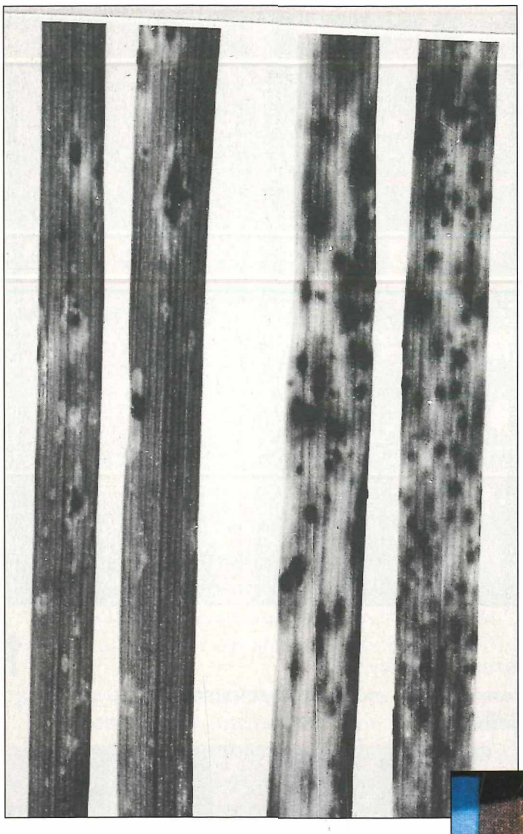


Fig. 31. Resistance of oat to stem rust. Left mesothetic reaction of the cv. Jostrain, right moderately susceptible reaction of its derivative, line Rodney E (Pg 3). Tested to the oat stem rust isolate 23–76 from Austria.

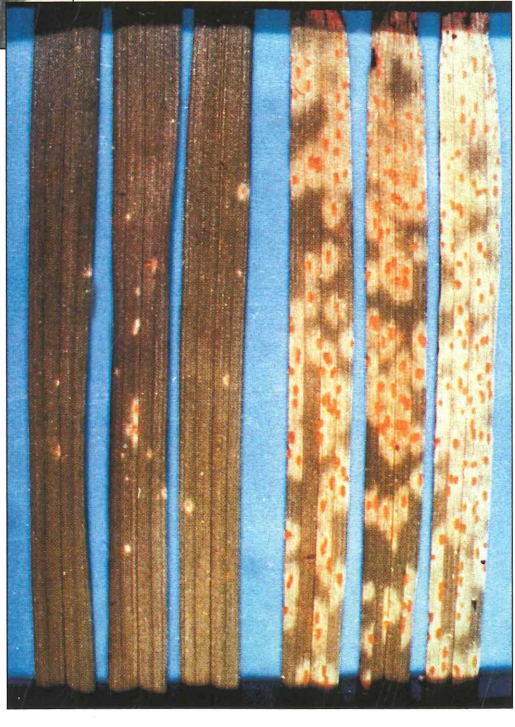


Fig. 32. Reaction to oat crown rust in line Pc 50-2 (infection types 0–2; very low intensity of attack) and in line Pc 50-4 (infection type 4, very high intensity of attack). Tested to the crown rust isolate 7–77 from Poland.

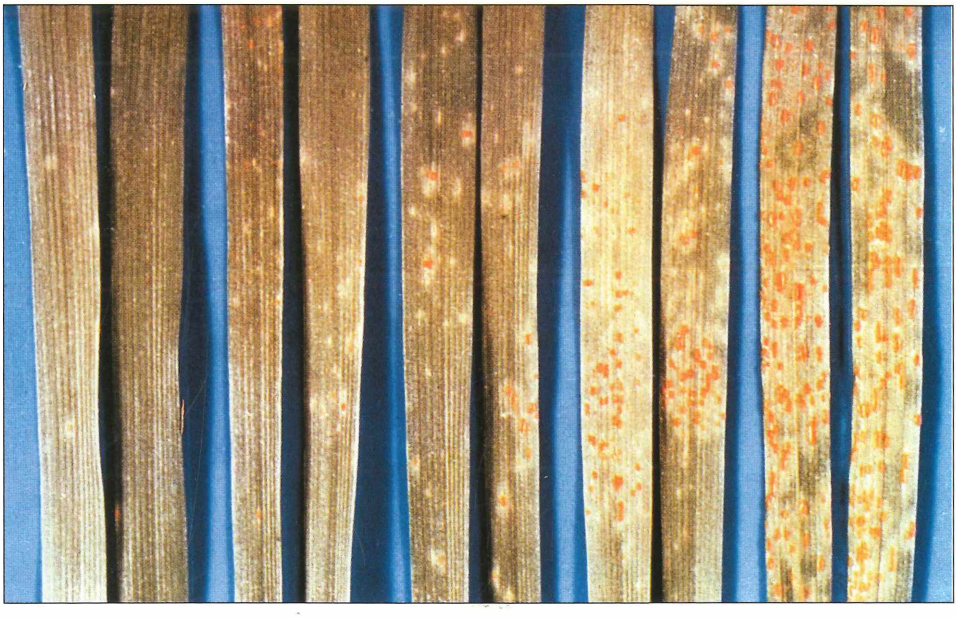
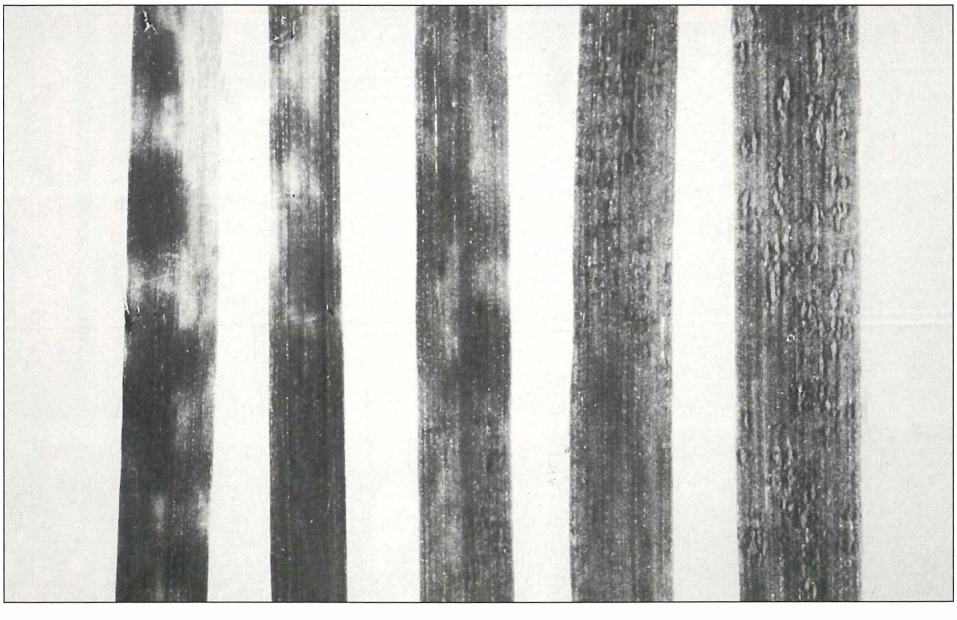


Fig. 33. Phenotypes of the F_2 of the cross Pc 50-2 x Pc 50-4.
Tested to the crown rust isolate 7–77 from Poland.

Fig. 34. Crown rust resistance of *Avena fatua* L. CS1.
From the left: highly resistant *A. fatua* L. CS1 and the highly resistant, resistant and moderately resistant phenotypes of the cross *A. fatua* CS1 x Leanda; right: highly susceptible cv. Leanda.



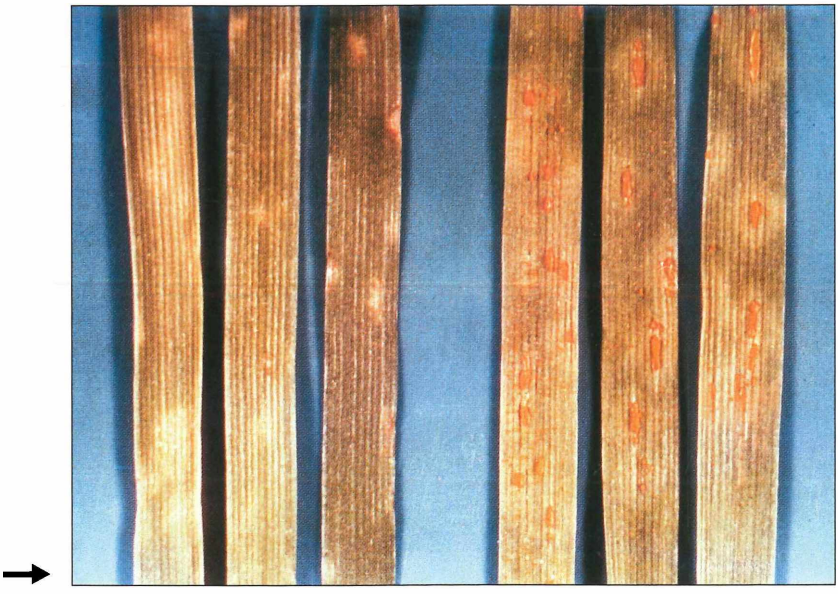
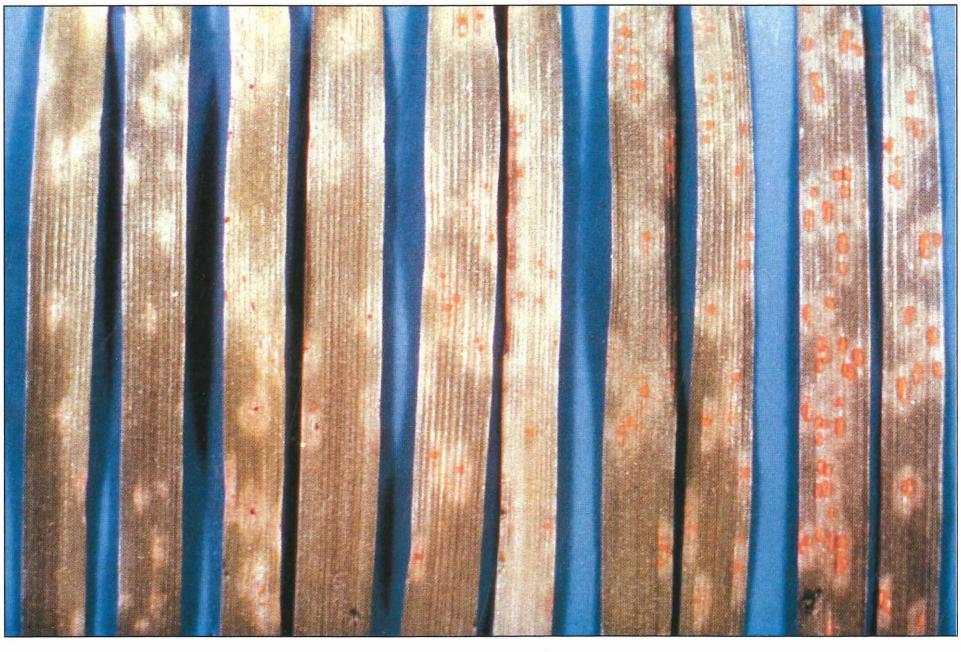


Fig. 35. Resistance of oat to crown rust.
Left: highly susceptible cv. Pan, right: highly resistant line of the accession *Avena sterilis* L., CAV 2648.

Fig. 36. Phenotypes of F_2 of the cross *Avena sterilis* L., CAV 2648 x cv. Pan.
From the left:highly resistant plants (infection type 0;), resistant (type 1), moderately resistant (type 2) and highly susceptible (type 4).
On the second couple of leaves from the left mass production of teliospores is obvious.



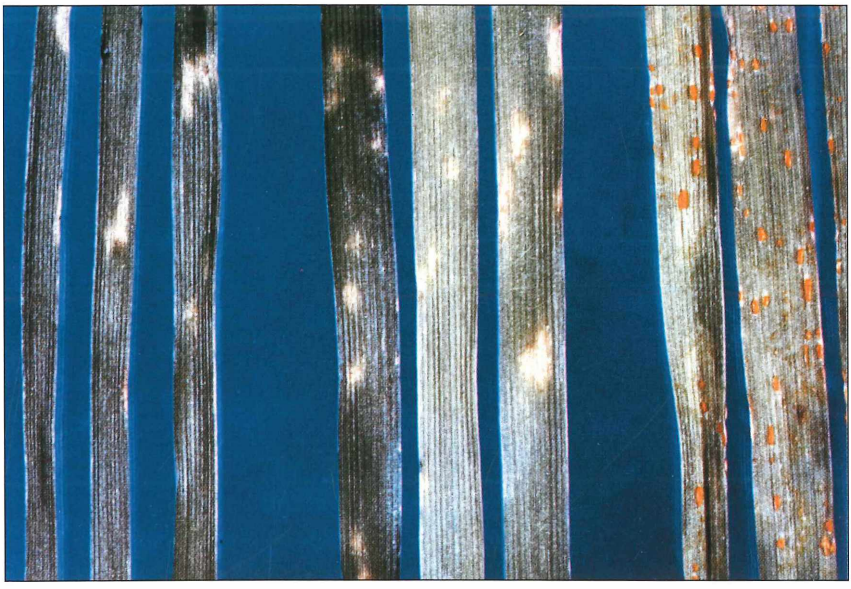


Fig. 37. Derivatives of the cross *Avena sterilis* L., CAV 2648 x Pan with different resistance to crown rust. Tested to crown rust isolate 7–77 P.
From the left donor of crown rust resistance, *A. sterilis* L., CAV 2648, in the centre a line with high resistance, phenotypically identical with the donor of resistance, right the cv. Pan.

Fig. 38. From the left donor of resistance *A. sterilis* L., CAV 2648, in the centre resistant line, right susceptible cv. Pan.

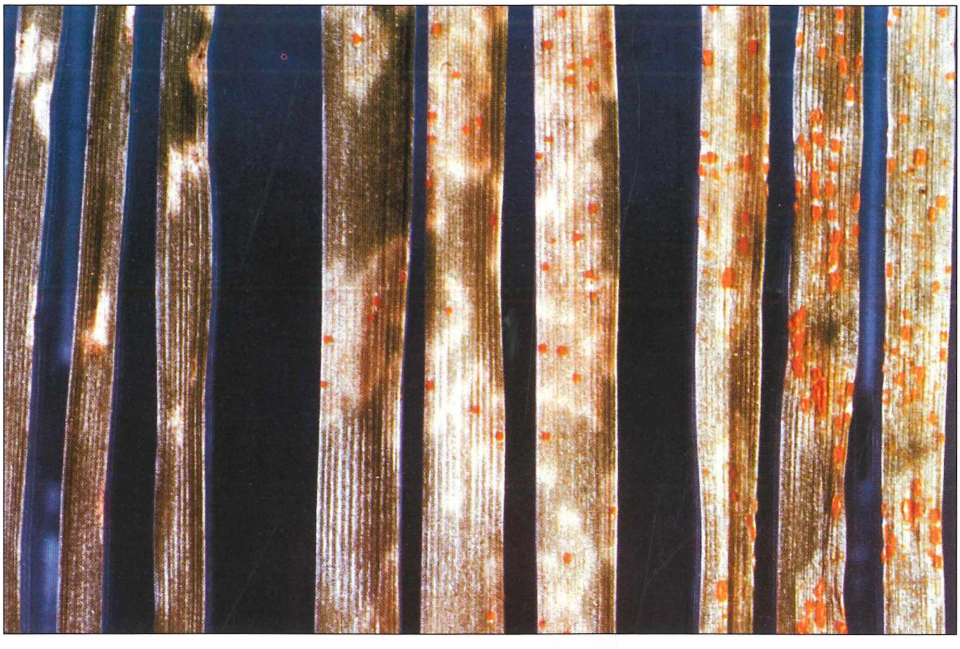


Fig. 39. From the left donor of resistance, *A. sterilis* L., CAV 2648, in the centre a line with moderate resistance. In the seedling stage, crown rust produced only urediospores on it; right highly susceptible cv. Pan.

Fig. 40. From the left donor of resistance, *A. sterilis* L., CAV 2648, in the centre a line with moderate resistance. In the seedling stage, crown rust produced teliospores on it, right highly susceptible cv. Pan.

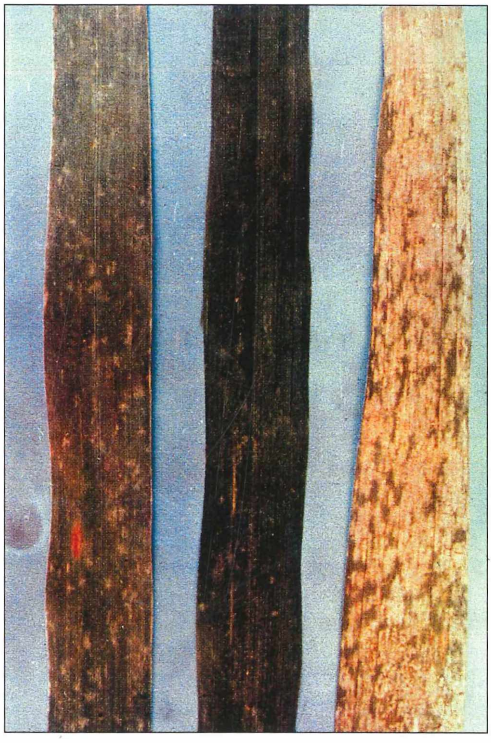


Fig. 41. Sources of specific resistance of oat to powdery mildew. From the left cv. Mostyn (Eg 3), in the centre the translocation line *Avena barbata* Brot, Cc 6490 (Eg-4), right highly susceptible cv. Tiger.

Fig. 42. Sources of adult plant resistance of oat to powdery mildew. From the left cv. Maelor, in the centre cv. Maldwyn, right highly susceptible cv. Tiger.

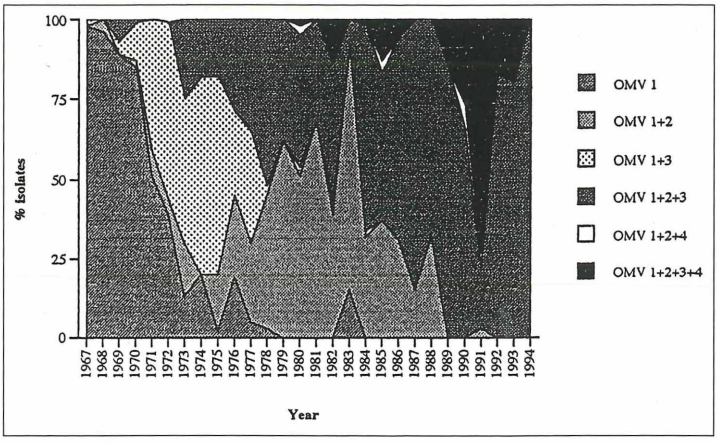
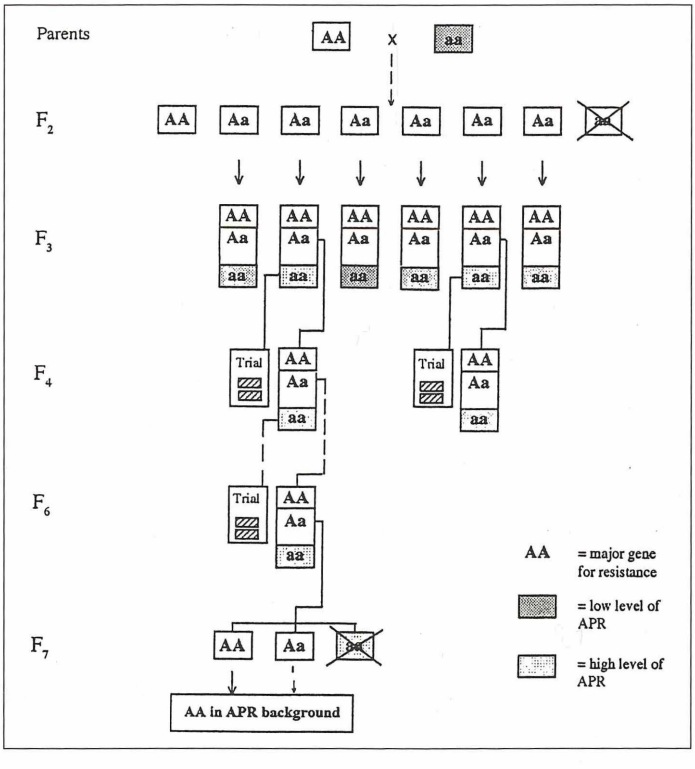


Fig. 43. Percentage of powdery mildew isolates received by the UK Cereal Pathogen Virulence Survey (1967–1994) with different virulence combinations. (ŠEBESTA ET AL. 1997)

Fig. 44. Development of segregates combining major gene and adult plant resistance (APR) (after Jones, 1982). The parent consists of an effective major gene source (AA) and an APR variety with no corresponding major gene (aa). Susceptible F_2 plants are eliminated in glasshouse tests and aproximately 50 seed of the resistant progeny (F_3) planted as spaced plants in the field and exposed to mildew. The heterozygote families are identified and susceptible and resistant plants marked. In the following year the seed from susceptible plants are put out as bulked rows or hills for quantitative assessment and the seed from resistant plants again sown as spaced plants. Following an analysis of disease scores on the replicated clumps ('aa' plants), this enables the identification of the F_2 derived families with the highest levels of APR. This process is continued for several generations, then the final heterozygotes are allowed to segregate and the double dominant 'AA' plants (identified by glasshouse tests), of those families showing the highest levels of APR in the clump tests, identified and seed multiplied.



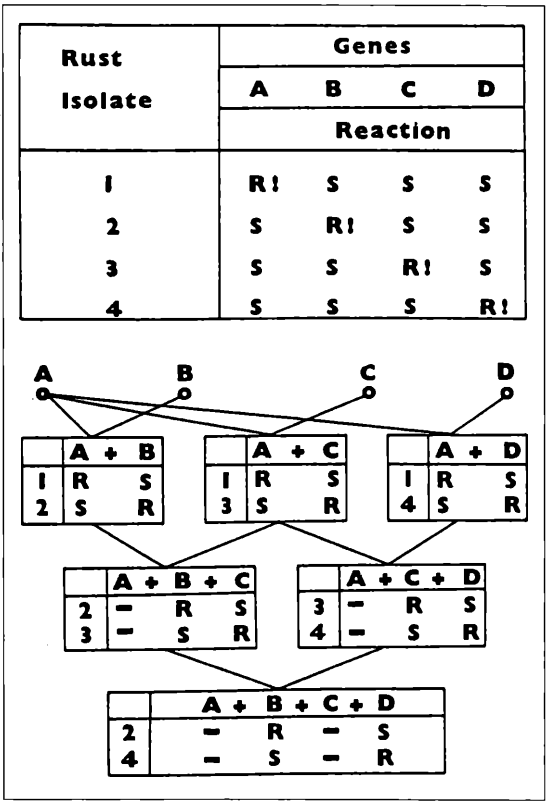
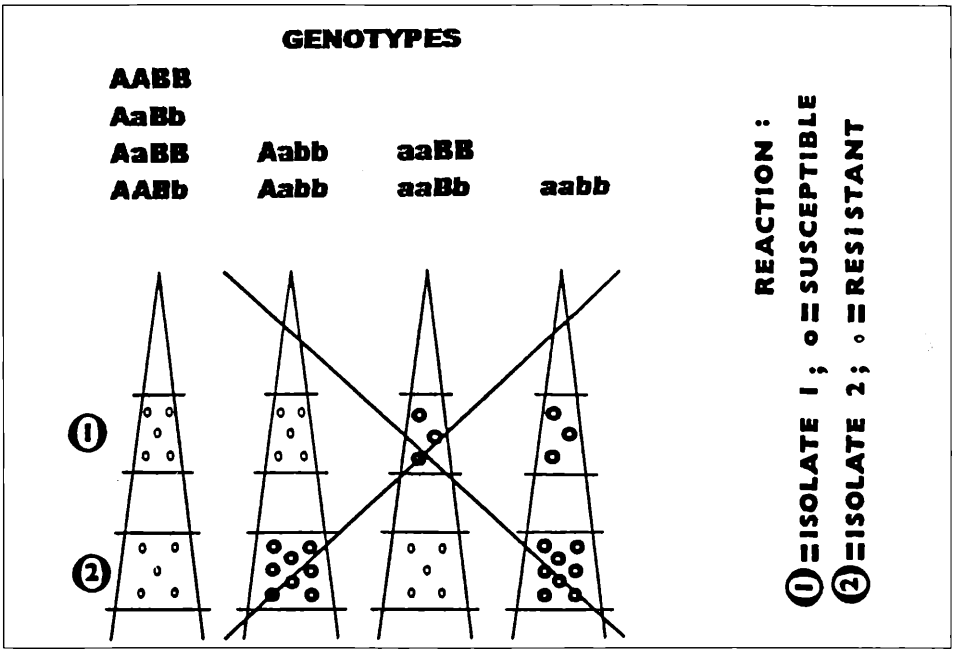


Fig. 45. Successive combining of non-allelic major resistance genes in a multigenic line by means of the pathologic detection. (VETTEL, 1957; RUDORF, 1965; BRUCKNER, 1967; (ŠEBESTA, 1979).

Fig. 46. Pathologic detection of non-allelic resistance genes combined in multigenic line (Seedling test) (ŠEBESTA, 1979).



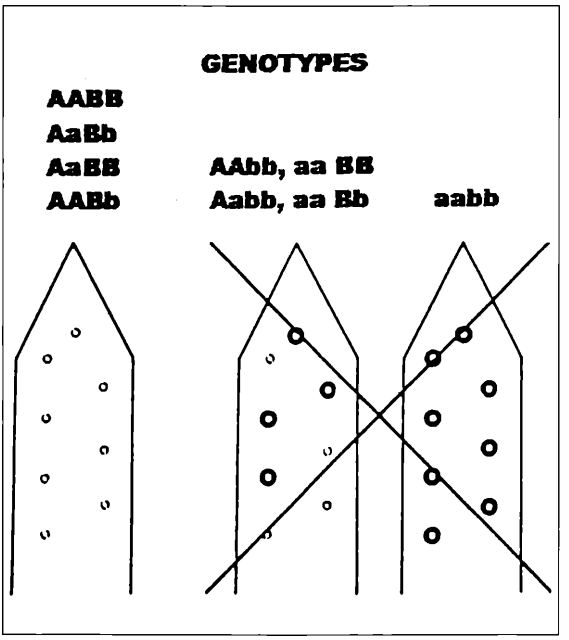


Fig. 47. Pathologic detection of non-allelic resistance genes combined in multigenic line (Adult plant test) (ŠEBESTA, 1979).

Fig. 48. Initiators of the international cooperation in disesase resistance of oat at the EODN-experiment in Petzenkirchen research station in Austria (right J. ŠEBESTA and left B. ZWATZ).

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