

Oat Stem Rust

J. W. Martens

*Agriculture Canada Research Station,
Winnipeg, Manitoba, Canada*

I. Introduction	103
II. Distribution and Importance	104
III. Pathogenic Specialization and Virulence Dynamics	105
A. Early Investigations	107
B. North America	107
C. Eurasia	110
D. The Middle East and East Africa	112
E. Australasia and South America	112
F. Virulence and Competitive Ability	113
IV. Environmental Factors Affecting the Host-Parasite Interaction	114
V. Genetics and Cytology of the Pathogen	115
A. Inheritance of Virulence and Spore Color	115
B. Mutability	116
C. Crosses between <i>Puccinia graminis tritici</i> and <i>Puccinia graminis avenae</i>	117
D. Cytology	117
VI. Host Resistance and Control Strategies	118
A. Origin and Nature of the <i>Pg</i> Genes	118
B. Breeding for Enduring Stem Rust Resistance	123
VII. Conclusions	124
References	124

I. Introduction

Oats, *Avena* spp. L., and its parasite, *Puccinia graminis* Pers. f. sp. *avenae* Eriks. and E. Henn., are first known to have appeared in the literature almost 2400 years ago. Oats were referred to by the Greek writers Dieuches (400 B.C.) and Theophrastus (371–286 B.C.), and by the Romans Cato the Elder (234–149 B.C.), Cicero (106–43 B.C.), and

Pliny the Elder (23–79 A.D.), among others (Coffman, 1961). Numerous writers have interpreted biblical references to blight, blasting, and mildew of grain to mean the rusts and the smuts, but there is probably merit in Arthur's (1929) suggestion that these pathogens were only a few of many factors that could have caused the problem. Aristotle the Greek (384–322 B.C.) writes of rust being produced by warm vapor, of devastating rust, and of rust years. His pupil Theophrastus noted that cereals are more affected by rust than are legumes (Arthur, 1929). The prayer of the officiating priest at a Robigalia ceremony, as given by the Roman poet, Ovid (43 B.C.–17 A.D.), "Stern Robigo, spare the herbage of the cereals, . . . withhold we pray, thy roughening hand," could scarcely be more explicit. It leaves little to the imagination of anyone who has read a rust nursery. Pliny referred to rust as the greatest pest of the crops.

However, a detailed account of the stem rust condition, almost certainly including oat stem rust, had to wait almost 18 centuries, when the Italians Tozzetti and Fontana independently described it in 1767 (see Fontana, 1932; Tozzetti, 1952). The quality of their descriptions is most remarkable.

For almost a century after Persoon first named the organism *P. graminis* in 1797 (Arthur, 1929), it was regarded as a single species capable of attacking all the common cereals and grasses. It was not until 1894 that Eriksson in Sweden recognized it as a distinct biologic race and named it *P. graminis* f. *avenae* (Eriksson, 1894). Thirty years later, Stakman *et al.* (1923) first reported specialization within *P. graminis avenae*.

It is not practical to list all of the investigators who have contributed to our knowledge and understanding of the organism, but some of the pioneers deserve special mention. These include Eriksson and Henning in Sweden, who laid the foundation for pathogenic specialization studies, Stakman and Levine in the United States, Waterhouse and Watson in Australia, and Bailey, Gordon, and Johnson in Canada.

Stakman, Levine, and Bailey (1923) first reported specialization within *P. graminis avenae* describing four avirulence/virulence combinations based on a study of over 100 collections from five countries using two differentials that carried genes *Pg-1* and *-2* and a universally susceptible cultivar, Victory.

II. Distribution and Importance

Stem rust of oats occurs almost everywhere that oats are grown and periodically has caused severe crop losses. The ancients recognized

oats and also wrote of "rust years" earlier than 300 B.C. (Arthur, 1929; Chester, 1946). Eriksson and Henning in 1896 (Chester, 1946) described the occurrence of rust years on a worldwide basis during the period 1660–1892. In 1889 oat rust was so destructive in Sweden that the royal government funded research on rust prevention, which led to the historic and fundamental work of Eriksson and Henning.

In North America there were severe epidemics in 1904, 1916, 1923, 1927, 1935, 1938, 1943, 1949, 1953, 1955, and 1977 (Craigie, 1957; Roelfs and Long, 1980). Roelfs (1978) and Roelfs and Long (1980) report that during the 59-year period from 1918 to 1977, oat stem rust epidemics causing yield losses of over 5% occurred in 8 years in Minnesota and North Dakota, 7 years in South Dakota, 5 years in Texas, 4 years in Iowa and Michigan, 3 years in Illinois, Kansas, and Nebraska, 2 years in Wisconsin, and 1 year in Oklahoma and Pennsylvania. The greatest nationwide losses were sustained in 1953, when they were estimated at 25, 10, 7, 5, and 4% in Minnesota, Iowa, Wisconsin, North Dakota, and South Dakota, respectively. Total United States losses for that year were 947,450 tonnes. In western Canada, Greaney (1936) estimated the average annual losses for Manitoba and Saskatchewan from 1929 to 1934 at 128,581 tonnes, with losses of 463,767 tonnes sustained in 1930. There were also important epidemics in Canada in 1944, 1945, 1947, and 1950 (Green *et al.*, 1961) but loss data are not available. The 1970 epidemic caused losses estimated at 92,572 tonnes in Manitoba (Martens, 1971), and the 1977 epidemic, the most severe in decades, caused losses of about 35% or 385,000 tonnes in Manitoba and eastern Saskatchewan (Martens, 1978). There were moderate losses in Manitoba and Saskatchewan again in 1981 (J. W. Martens, unpublished).

III. Pathogenic Specialization and Virulence Dynamics

Fortunately, the results of almost all of the investigations since Stakman, Levine, and Bailey first described specialization in oat stem rust can be interpreted in genetic terms because all of the differential lines used were later shown to have single genes for resistance. Since that time, a number of different systems of nomenclature have been used to describe the virulence characteristics of races of the organism (Stewart and Roberts, 1970). In this chapter, the results obtained since specialization was discovered will be described in terms of contemporary nomenclature (Martens *et al.*, 1979). This method describes the avir-

Table I

Avirulence–Virulence Combinations of *Puccinia graminis* f. sp. *avenae* Known to Occur in North America^a

North American numbers	Avirulence–virulence formula (<i>Pg</i> genes)	North American numbers	Avirulence–virulence formula (<i>Pg</i> genes)
NA 1	1,2,3,4,8,9,13,16,a/15	NA 27	9,13,15,16,a/1,2,3,4,8
NA 2	1,2,3,4,8,13,16,a/9,15	NA 28	9,13,15,16/1,2,3,4,8,a
NA 3	1,2,3,4,8,16,a/9,13,15	NA 29	9,13,16,a/1,2,3,4,8,15
NA 4	1,2,3,8,16,a/4,9,13,15	NA 30	13,16,a/1,2,3,4,8,9,15
NA 5	1,2,4,8,9,13,16,a/3,15	NA 31	1,3,8,16,a/2,4,9,13,15
NA 6	1,2,4,8,13,16,a/3,9,15	NA 32	1,8,16,a/2,3,4,9,13,15
NA 7	1,2,4,8,16,a/3,9,13,15	NA 33	1,4,8,13,16,a/2,3,9,15
NA 8	1,2,8,16,a/3,4,9,13,15	NA 34	1,3,4,8,16,a/2,9,13,15
NA 9	1,3,8,13,16,a/2,4,9,15	NA 35	2,4,8,13,16,a/1,3,9,15
NA 10	1,4,8,9,13,16,a/2,3,15	NA 36	2,8,13,16,a/1,3,4,9,15
NA 11	1,8,9,13,16,a/2,3,4,15	NA 37	2,8,16,a/1,3,4,9,13,15
NA 12	1,8,13,16,a/2,3,4,9,15	NA 38	1,2,3,4,8,13,15,16,a/9
NA 13	1,13,16,a/2,3,4,8,9,15	NA 39	1,2,4,8,9,13,15,16,a/3
NA 14	2,3,4,9,13,15,16,a/1,8	NA 40	1,3,8,13,15,16,a/2,4,9
NA 15	2,4,8,9,13,15,16,a/1,3	NA 41	1,4,8,13,15,16,a/2,3,9
NA 16	2,4,9,13,15,16,a/1,3,8	NA 42	1,4,8,16,a/2,3,9,13,15
NA 17	2,4,9,13,15,16/1,3,8,a	NA 43	1,8,9,13,15,16,a/2,3,4
NA 18	2,4,9,13,16,a/1,3,8,15	NA 44	2,3,9,13,15,16,a/1,4,8
NA 19	3,8,9,13,16,a/1,2,4,15	NA 45	2,4,8,13,15,16,a/1,3,9
NA 20	3,8,13,16,a/1,2,4,9,15	NA 46	2,4,8,13,15,16/1,3,9,a
NA 21	3,9,13,15,16,a/1,2,4,8	NA 47	2,4,8,16,a/1,3,9,13,15
NA 22	4,8,9,13,16,a/1,2,3,15	NA 48	2,4,13,15,16,a/1,3,8,9
NA 23	4,9,13,15,16,a/1,2,3,8	NA 49	2,8,13,15,16,a/1,3,4,9
NA 24	8,9,13,16,a/1,2,3,4,15	NA 50	3,4,9,13,15,16,a/1,2,8
NA 25	8,13,16,a/1,2,3,4,9,15	NA 51	3,13,15,16,a/1,2,4,8,9
NA 26	8,16,a/1,2,3,4,9,13,15	NA 52	8,13,15,16,a/1,2,3,4,9

^aRevised by Roelfs and Martens from Martens *et al.* (1979).

ulence–virulence of any culture in terms of a simple formula (Table I). The formula consists of numbers corresponding to those assigned the specific genes for resistance in the differential lines. Numbers designating the genes that condition resistance to the culture are written first, followed by a slash line, and the numbers designating ineffective genes. Infection types 0 to 2+ are considered to indicate a resistant or incompatible host response; infection types 3 and 4 are considered to indicate a susceptible or compatible host response (Stakman *et al.*, 1962). The designation of the mesothetic reaction, especially from the early published reports, becomes a matter of judgement. If what is expressed is potentially effective resistance in terms of disease control,

X- or better, then the reaction is considered resistant; an X+ is considered susceptible.

The terminology for describing host-parasite interactions presents certain problems since there is a lack of convention or agreement among pathologists and breeders. For the purposes of this chapter, pathogenicity is taken to mean the ability of the pathogen to cause the stem rust condition; avirulence or virulence qualitatively describes the interaction; and aggressiveness refers to competitive ability. While it would be more accurate to refer to avirulence or virulence of a specific pathogen race on plants, cultivars, or lines with specific genes for resistance, for the sake of conciseness "virulence on *Pg-X*" will be permitted. Similarly, avirulence-virulence combination is more accurate and precise than the term "race," but the latter will often be used because it is more concise and very widely understood.

It should also be noted that the progressive increase in detected variability and virulence in the pathogen population is, in part, related to the availability of the corresponding host gene differentials. The specialization studies began with genes *Pg-1*, -2, and -3; *Pg-4* was added in 1950; *Pg-8* in 1961; *Pg-9* in 1964; *Pg-13* in 1969; and *Pg-15*, -16, and -a in 1978.

A. EARLY INVESTIGATIONS

Following the pioneering work of Stakman *et al.* described earlier, Bailey (1925) published a race identification key based on three cultivars, later shown to possess single genes *Pg-1*, -2, and -3, and on the use of three reaction categories, resistant, mesothetic, and susceptible. In contemporary terms, four races were described: 1,2,3/ and 1,2/3 from North America; 2,3/1 from South Africa and Sweden; and 3/1,2 from Sweden only. By 1933, Gordon and Bailey (1928), and Gordon (1933) reported the occurrence of all of these races plus 1,2,3 and 1/2,3 from Canada. Waterhouse (1929) found all of the described races, except 3/1,2, and a new race 2/1,3 in Australia.

In 1930 Tedon reported five races from Sweden (Gordon, 1933). Thus from early specialization studies, it is evident that many of the possible avirulence-virulence combinations, based on the differentials then available, occurred in widely separated parts of the world.

B. NORTH AMERICA

North America is considered as one epidemiological unit with three subregions: the Great Plains region, where *Berberis* L. is not a signifi-

cant factor, stretches from the Gulf of Mexico some 4000 km to the northern limits of oat production; the eastern region, with a different pathogen population, where *Berberis* is a factor; and the much smaller western region, where the disease is less important and the population unlike that of the rest of the continent. The southern part of the continent may be further divided into additional ecological subregions (Roelfs *et al.*, 1982). The full range of variability of the North American population can usually be found in Canada, where the last generations of the annual cycles are produced. The graphic presentation of the succession of races in Canada and the performance of the pathogen population since 1921 (Fig. 1) is fairly representative of what is known about this aspect of the pathogen. From 1921 to 1943, the pathogen population in North America was relatively stable (Levine and Smith, 1937; Newton and Johnson, 1944; Green *et al.*, 1961; Stewart and Roberts, 1970). Although most of the possible avirulence-virulence combinations on the differentials then available were observed in nature, the one-gene virulence race, 1,2/3 predominated until 1943 when the first of a series of shifts occurred, largely in response to resistance genes in the host population on the continent. The distribution of resistance genes in the host population on the continent may be considered to have had three main phases: from about 1942 to 1955, when genes *Pg-1* and -2 were important in breeding programs; from 1956 to 1978, when *Pg-4*, singly and in combination with *Pg-2*, represented the main genes for resistance; and the present phase with the addition gene combinations *Pg-2*, -4, -9 (McKenzie *et al.*, 1976), *Pg-1*, -9, -13 (McKenzie *et al.*, 1981), and *Pg-2*, -9, -13 (cultivar Dumont; McKenzie *et al.*, 1984). These gene combinations have yet to influence the pathogen population in a significant way.

Although the old races were still important in the late 1940s, races with a wider virulence range increased sharply in both the eastern and Great Plains regions, but not the western region of the continent. By 1950, increasing differences between the eastern and Great Plains regions not attributable to host gene patterns became apparent. The one-gene-virulence races were disappearing, and races 3/1,2 and /1,2,3 were becoming important components in the eastern population, an event that did not occur in the Great Plains regions until 1961. As new genes for resistance were discovered, virulence on them, in various combinations, was usually also found. In most cases (e.g., *Pg-4*, -9, -13, and -a), virulence was present in the pathogen population before cultivars with these genes were grown over a significant area. Thus, the resistant cultivars simply caused selective advantage shifts in the pathogen population. However, changes in the host population do not account for all

the pathogen changes that occurred. Subsequent to 1956–1957, the eastern and Great Plains populations became quite distinct. In the east, virulence on *Pg-2* resistance was very common, but in the Great Plains region it was rare until 1961. By the time *Pg-8* and *-9* became available as differentials in 1960 and 1963, respectively, races 1,8/2,3,4,9 and 2,9/1,3,4,8 were the most common in the east and the Great Plains, respectively. In the western region, races 1,2,3,4,8/9 and 1,2,4,8/3,9 continued to persist. From 1965 to 1981, the eastern population was characterized by combined virulence on all of the available genes except *Pg-8*, *-13*, *-16*, and *-a* and in the Great Plains population on all genes except *Pg-9*, *-13*, *-16*, and *-a* (Fig. 1; Martens and McKenzie, 1979; Roelfs *et al.*, 1978, 1982; Martens, 1981). Virulence on the differentials with *Pg-13*, *-16*, and *-a* is very rare or nonexistent on the continent. The appearance and persistence of race 2,4,9,13/1,3,8 at significant levels throughout most of the period since 1969, despite the fact that most of the commercial cultivars in the Great Plains region are resistant to it, clearly indicates that factors other than virulence are determinants of the pathogen population composition. None of the virulence carried by this race, except that on *Pg-1*, is helpful to survival anywhere on the continent. In the western region, Mexico, and the southern United States, races with virulence on only *Pg-15*, or on *Pg-3* and *-15* continue to persist (Roelfs *et al.*, 1980; Martens, 1978, 1981). All of these results are based on field isolates, collected from commercially grown *Avena sativa* L., which may have either no known resistance genes, or genes *Pg-1*, *-2*, *-4* singly and *Pg-2* in combination with *Pg-4*, or from wild oats (*A. fatua* L.) that is not known to have any genes for stem rust resistance. However, there is recent evidence to suggest that such survey techniques do not detect all of the variability present in the pathogen population on the continent, even though many of the host plants have no resistance. When trap nurseries with various host genotypes, but especially lines with gene *Pg-15*, were planted and sampled extensively, races not found in the field isolates, however large the field samplings, were detected (Martens and McKenzie, 1979). In 1980, a total of 21 identified races, on 10 differentials, were isolated from field-survey and trap-nursery isolates, thus representing the greatest pathogen variability ever reported for one season in the 60-year history of specialization studies in oat stem rust in Canada (Martens, 1981).

C. EURASIA

The Scandinavian pathogen population is characterized by a great deal of variability with at least 16 races including all possible combina-

tions in the four-gene system used (Leijerstam, 1966; Mac Key and Mattsson, 1972), a phenomenon that has prevailed since studies began. The most virulent race reported by Stakman *et al.* (1923) was an isolate from Uppsala, Sweden. Gordon (1933) also noted the relatively wide virulence range of Swedish isolates. The most common races also include those with the most genes for virulence, even though the genes are not necessary for survival in Scandinavia. *Berberis vulgaris* occurs in Scandinavia and may be a factor in the life cycle of the pathogen (Leijerstam, 1966). From 1961 to 1966, four races, including 4,1/2,3,8, were identified in Italy. In 1974–1975, 10 races were identified, including the widely virulent race /1,2,3,4,8,9 (Paradies *et al.*, 1976), indicating a wide range of virulence that is apparently not related to resistance genes in the host.

Kostic (1966) reported that oat stem rust is a serious problem in Yugoslavia in some years. Races 1,2,4/3 and 4/1,2,3 were the most common, but four other races were also isolated.

Sebesta (1973a) isolated six races in Czechoslovakia in 1967–1968, with the most widely virulent race comprising 64% of all isolates identified, but found no virulence on *Pg-4* resistance. For the 11-year period 1965–1975, 10 avirulence–virulence combinations were identified in Czechoslovakia, Austria, and Romania. Race 4,9/1,2,3,8 was most common during this period (Sebesta and Zwatz, 1980). Genes *Pg-13*, -4, and the adult plant gene *Pg-11* were the most effective against this central European pathogen population.

Suzdalskaya *et al.* (1978) have recently published an account of the dynamics of this pathogen in the European and Transcaucasian (Georgian) regions of the U.S.S.R. They report that races 3,4/1,2, 4/1,2,3, 3/1,2,4, and /1,2,3,4 prevailed in all of the regions studied 1959 to 1963. From 1964 to 1968, 2,9/1,3,4,7,Sa and 2,3,9/1,8,Sa (Sa = Saia) were common; and from 1970 to 1975, 1,2,8,9,Sa/3,9 and 3,4,Sa/1,2,8,9 prevailed. In 1971 and 1974, race /1,2,3,4,8,9 was also observed. For the 6-year period ending in 1975, the following average annual frequency of virulence was observed in the isolates studied: *Pg-1* = 59%; *Pg-2* = 62%; *Pg-4* = 21%; *Pg-8* = 56%; *Pg-9* = 75%; and *Saia* = 15%. Virulence on *Saia* was found most commonly in isolates from the Volga–Vyatka region, only rarely from the central region, Byelorussia and the Ukraine, and never from the north western region, the Baltic Sea area or Georgia. The authors conclude that the high frequency of virulence on the specified types of resistance in the pathogen population of the U.S.S.R. cannot be explained by the selective effect of resistance genes in the host population, since most of the oat cultivars grown are susceptible to stem rust and have no genes that could produce a noticeable

selective effect on the pathogen population. Shikina (1974) reported 15 races occurring in the northern Caucasus with six races predominating, suggesting considerable variability in the populations.

Mehta (1940) observed stem rust of oats in the Nilgiri Hills of southern India at altitudes of over 2000 meters. Although the sample size was small, he identified four races, 2,3/1, 2/1,3, 3/1,2, and /1,2,3, indicating considerable variability and a wide range of virulence.

D. THE MIDDLE EAST AND EAST AFRICA

Variability and apparently unnecessary virulence characterize the pathogen populations of these regions. From 1926 to 1938, five races were identified from the Middle East, including 1,2,3/, 1,2/3, 1/2,3, 2/1,3, and /1,2,3. The latter was the most widely virulent race, and comprised 30% of all isolates identified during that period (Wahl *et al.*, 1966). This pattern has continued in Israel (Sztejnberg and Wahl, 1967, 1976), with race 4,9/1,2,3,8 being the dominant race for many years.

Limited data from East Africa indicate a similar pattern (Green *et al.*, 1970; Martens *et al.*, 1976). Races 3,8/1,2,4 and 8/1,2,3,4 were prevalent from 1960 to 1964. In 1968, races 9/1,2,3,4,8, 3,9/1,2,4,8, and /1,2,3,4,8,9 were identified with the first comprising 75% of the isolates. In 1971 races 3,9,13/1,2,4,8, 9,13/1,2,3,4,8, 13/1,2,3,4,8,9, and /1,2,3,4,8,9,13 were identified in collections from Ethiopia and Kenya. The genes for resistance in the commercial oat population in this region do not provide an obvious reason for this sustained wide range of virulence.

E. AUSTRALASIA AND SOUTH AMERICA

From 1928 to 1938, races 1,2,3/ and 1,2/3 comprised over 85% of over 700 isolates identified in Australia. Races 2,3/1, 2/1,3, and /1,2,3 were also found (Waterhouse, 1952; Luig and Baker, 1973). The dominant races continued to prevail from 1939 to 1951 with 57% of all isolates identified; with races 2,3/1, 2/1,3, and 1/2,3 comprising the balance. From 1970 to 1972, Luig and Baker (1973) identified 15 races (22 strains) in eastern Australia. There was considerable virulence in the population on Pg-1, -2, -3, -4, less on Pg-9, and none was observed on Pg-8 resistance. In contrast to the Australian race spectrum, only three races with relatively narrow virulence range, 1,2,3,4,8,9,13/, 1,2,3,4,8,13/9, and 1,2,4,8,13/3,9 were identified in New Zealand from over 60 isolates in 1975-1976 (Martens *et al.*, 1977).

Although only limited data are available on the South American rust population, it appears genetically variable with many genes for virulence (Orjuela *et al.*, 1962; Martens *et al.*, 1976). In Colombia, stem rust occurs wherever oats are grown and is a limiting factor in its production. Orjuela *et al.* reported 22 different avirulence-virulence combinations, including some not found elsewhere, and some that were virulent on all of the resistance genes used. They concluded that the extreme physiologic variation of *P. graminis avenae* in that country was surprising. *Berberis* spp. are known to occur in Columbia, but it is not known if they are a factor in the life cycle of the rust there. Coelho (1976) reported 10 races from Brazil but noted that none of them were virulent on Pg-8 resistance.

F. VIRULENCE AND COMPETITIVE ABILITY

Leonard (1969) cultured a heterogeneous population of the pathogen for eight generations in the greenhouse and found that the races with unnecessary virulence genes had survival values of 14–46% lower than races without them. He concluded that multiline cultivars could be used to stabilize pathogen populations. Martens (1973) cultured mixtures of races of differing virulence range on susceptible plants in growth chambers for five uredial generations at various temperatures, and in the field. He found that the races with fewest genes for virulence maintained or increased their levels in the growth chambers in all cases, but were consistently out-performed by races with more genes for virulence under field conditions. He concluded that the number of genes for virulence carried by a given race, other than those required for successful parasitism, was probably not the key determinant affecting the frequency of races in nature. Sebesta (1973b) found that the most widely virulent race was also the most aggressive. Suzdalskaya *et al.* (1978) concluded that the very frequent occurrence of virulent races in the U.S.S.R. cannot be explained by the selective effect of the resistance genes of the host plant, since the majority of oat cultivars grown are susceptible to stem rust and could not produce a noticeable selection effect on the pathogen population.

Sztejnberg and Wahl (1976) noted that race 4,9/1,2,3,8 has been prevalent in Israel for many years. They concluded that its prevalence cannot be ascribed to the preferential selection pressure of the host, and "this shows that a race with a wide range of virulence is not inferior in fitness even when the virulence is not necessary for survival."

Mac Key and Mattsson (1972) found isolates with every possible

avirulence–virulence combination for the system they were using in Sweden. They noted that it is disturbing that a pathogen population like that in Scandinavia maintains so many genes for virulence. The population is fairly well isolated geographically under rather extreme ecological conditions and apparently has not been subjected to the selective effects of specific genes for resistance in the host. Similar evidence from Australia (Luig and Baker, 1973) and East Africa (Green *et al.*, 1970; Martens *et al.*, 1976) leaves no doubt that stabilizing selection is not a key determinant affecting pathogen population distribution in those parts of the world where it has been studied.

Why does this organism carry so many genes for virulence, and how do they arise? It is possible that in some cases the genes for resistance are more widely distributed in the host population than is recognized. Genes *Pg-3* and *-9* could be cases in point. However, with the genes from *A. sterilis* L., *Pg-13* and *-15*, it is most unlikely that they ever occurred in North America before they were intentionally introduced and identified. But the corresponding virulence genes were already present before the pathogen had an opportunity to “recognize” these genes for resistance. Perhaps these genes impart selective advantage other than virulence on *Avena* spp. Possibly the pathogen parasitizes hosts other than *Avena* where these “unnecessary” genes are in fact useful, if not necessary.

IV. Environmental Factors Affecting the Host–Parasite Interaction

The presence or absence of the gene(s) for virulence in the pathogen and resistance in the host are only two of the determinants affecting the course of the interaction. Sensitivity to temperature in some interactions has been recognized almost since the beginning of race studies. Gordon (1933) demonstrated the thermostability of host genes *Pg-1* and *-2* and the sensitivity of *Pg-3*. He also noted that telia were formed more rapidly at 24°–28°C than at 12°–16°C. Roberts (1962) examined the phenomenon of temperature sensitivity and found that the expression of gene *Pg-4* was dependent on a certain temperature during a critical period between the inoculation and the fleck stage.

Plants kept at 22°C after inoculation would not express resistance if grown at 30°C for at least 3 days prior to the fleck stage. Plants kept at 30°C after inoculation required at least 4 days at 22°C before flecking to completely express the characteristic resistance. Preinoculation

temperatures had no effect on the expression of resistance. The response was shown to be quantitative in the case of partial treatment, and it was localized rather than systemic. Martens *et al.* (1976) showed that the temperature sensitivity of *Pg-4* and other genes was independent of the host genotype, but that there was a race effect. High-temperature breakdown of *Pg-4* resistance occurred at a higher temperature with some races than with others, but at 30°C breakdown was complete for all races. They found the optimal temperature for rust development to be 20°–25°C and confirmed the observations of Gordon regarding telial development.

Genes *Pg-1* and *-2* are temperature-insensitive; genes *Pg-9*, *-8*, *-13*, *-15*, *-16*, *-4*, and *-3* are temperature-sensitive in order of increasing sensitivity (Martens *et al.*, 1967, 1979).

The effect of temperature, light, and host genotype on prepenetration development of *P. graminis avenae* was studied by Kochman and Brown (1976a). Germination of uredospores occurred at the relatively wide range of 10°–30°C, but optimum conditions for germ-tube growth and formation of appressoria was 20°C in darkness. Germination was inhibited at 35°C. Under optimum conditions, maximum germination and formation of appressoria was attained within 4 hr after inoculation. No host effect on germination or prepenetration development was detected among the six wild and cultivated lines, including one with genes *Pg-2* and *-4*. This observation suggests the absence of any prepenetration defence mechanisms. However, optimum development conditions were different for the penetration phase (Kochman and Brown, 1976b). Penetration was greatest at 30°–35°C and at light intensities of 5625 lux or above. Maximal penetration was achieved in a dew period of 16 hr.

V. Genetics and Cytology of the Pathogen

A. INHERITANCE OF VIRULENCE AND SPORE COLOR

Gordon and Welsh (1932) appear to be the first investigators to have studied the inheritance of virulence in *P. graminis avenae*. They selfed race 1/2,3 and from 20 aecial cups they isolated races 1/2,3 (15 times), /1,2,3 (seven times), and 2/1,3 (once), demonstrating that the isolate was heterozygous at two of the three loci studied. Johnson and Newton (1940) selfed a series of races and found two different cultures of race 1,2/3 to be homozygous and one culture to be heterozygous for vir-

ulence on *Pg-3* resistance; race 2,3/1 was heterozygous for virulence on *Pg-2* resistance; two different cultures of race 1/2,3 were heterozygous for virulence on *Pg-1* resistance; and races 2/1,3 and 1,3/2 were homozygous.

In crosses between races, they generally found avirulence to be dominant. In reciprocal crosses, they noted evidence of cytoplasmic inheritance of virulence on *Pg-3* resistance. In almost all crosses involving virulence on *Pg-3*, the hybrid was similar to the maternal parent. In crosses between races with normal brick-red- and orange-colored uredia, they found red to be dominant. In further studies, with hybrids of races 1,3/2 and 2/1,3, Johnson (1949) showed avirulence on *Pg-1* and *Pg-2* resistance to be dominant, and he concluded that they were governed by two pairs of complementary genes, while he confirmed that avirulence on *Pg-3* resistance was maternally inherited. However, Green (1965), working with selfs and crosses of 10 different cultures, concluded that avirulence on resistance conferred by genes *Pg-1* and *Pg-2* is governed by single dominant genes and not two complementary genes each as previously indicated. Green also confirmed the extra-chromosomal nature of the inheritance of avirulence on *Pg-3* resistance. All the cultures used in the study were homozygous for virulence or avirulence on resistance conferred by gene *Pg-4*, and in all crosses between avirulent and virulent races, avirulence was dominant in the F_1 . All the progenies of all the selfing and crossings were avirulent on resistance conferred by gene *Pg-8*. In further studies, Green and McKenzie (1967) confirmed that avirulence on *Pg-1*, *Pg-2*, and *Pg-4* resistance was controlled by single dominant genes. However, in some crosses the action of the dominant avirulence gene on *Pg-2* resistance was modified by a recessive gene and type 2+ rather than type 1 infections were produced. Avirulence on plants with *Pg-9* resistance also appeared to be controlled by a single gene. Although the results were not conclusive, evidence suggested that avirulence on cultivars with *Pg-8* resistance is recessive and that on hosts with *Pg-13* resistance avirulence is dominant (Martens *et al.*, 1970). There is no evidence for linkage between virulence genes, and generally gene-for-gene relationships occur within this host-parasite system (Martens *et al.*, 1970; Mac Key and Mattsson, 1972).

B. MUTABILITY

Puccinia graminis avenae can be readily mutated in the uredial stage by means of chemical treatment (Teo and Baker, 1975). Ethyl methane-

sulfonate (EMS) induced variability in virulence, uredospore color, and the rate of telial development. Within the ranges of $9.8\text{--}19.6 \times 10^{-2}$ M EMS, $20^{\circ}\text{--}30^{\circ}\text{C}$, and 2.0–5.4 hr treatment duration, increases in any of the treatment variables increased mutation rates. However, with increasing severity of treatment, spore viability decreased from 96 to 19%. Recurrent mutagen treatment of successive uredial generations was shown to produce variability not induced by the first treatment.

C. CROSSES BETWEEN *PUCCINIA GRAMINIS TRITICI* AND *PUCCINIA GRAMINIS AVENAE*

Johnson and Newton (1933) were successful in hybridizing these two *formae specialis* in one of 32 attempts. They crossed a race of *Puccinia graminis* f. sp. *tritici* that was homozygous for avirulence on all the oat hosts used, *Dactylis glomerata* L. and *Phalaris canariensis* L., with race /1,2,3 of *P. graminis* f. sp. *avenae*. Selfed progeny resulted in segregates of races /1,2,3, 2/1,3, and 3/1,2 and segregates virulent on wheat, which indicated a successful hybrid. The hybrids were virulent on oat cultivars with no resistance genes, genes *Pg-1* and *Pg-2*, the wheat cultivars Little Club and Liguleless, *D. glomerata* and *P. canariensis*, and some barley cultivars, but they were avirulent on oats with gene *Pg-3*, and on nine wheat cultivars that the *tritici* parent could attack.

D. CYTOLOGY

McGinnis (1953) examined germinating sporidia of *P. graminis* during nuclear division and found at metaphase a haploid number of six chromosomes. Prophase chromosomes were observed to be united to form a continuous chain. Discrete individual chromosomes could not be seen at this stage. At metaphase, chromosomes appeared loosely paired with residual terminal attractions, suggesting that the basic haploid chromosome number could be three.

Craigie and Burrows (1967) noted departures from the usual binucleate condition in uredial mycelium, urediospores and germ tubes in race /1,2,3 of *P. graminis* f. sp. *avenae*. They observed occasional trinucleate and tetranucleate cells, which reproduced in kind. The trinucleate condition developed when one nucleus of a binucleate cell divided and the other did not, or by the migration of one nucleus from a tetranucleate cell. Trinucleate cells occurred mainly in younger mycelium, and their frequency varied widely from one infection site to another. These

cells tended to revert to the binucleate condition either by dissolution of the extra nuclei or by their migration from the cells concerned.

VI. Host Resistance and Control Strategies

A. ORIGIN AND NATURE OF THE *Pg* GENES

The *Pg* genes that have been important historically, that have influenced pathogen dynamics, or that are presently potentially important in terms of breeding for resistance are all included in the international differential set (Table I). The genes that are absent from Table I are described by Simons *et al.* (1978). All of these genes are available as single-gene lines in the Rodney O background, which are the basis for the international differential set (Martens *et al.*, 1979).

Gene *Pg-1* was introduced to the United States, presumably from northern or central Russia, in about 1850 in the cultivar White Russian, which became one of the progenitors of some 15 cultivars on the continent (Coffman, 1977). The dominant thermostable gene was used extensively in the north central states of the United States for many years (Stewart and Roberts, 1970), but not in Canada.

Gene *Pg-2*, also dominant and thermostable, appears to have been introduced to the United States from the U.S.S.R. at least three different times. The first introduction involved the cultivar Green Russian, which, according to Coffman (1977), was brought into North Dakota by settlers from the U.S.S.R. in about 1870. The exact date and where the settlers came from is not clear (Coffman, 1977), but other sources (Quisenberry and Reitz, 1974) indicate a migration from the Ukraine to Dakota in the early 1870s, suggesting the Ukraine as a source of Green Russian. The second source of the gene was the cultivar Kherson, introduced by F. W. Taylor of Nebraska. It was named after the area of origin near Odessa on the Black Sea. The third was with the introduction of the cultivar Sixty-Day, believed to be very similar to Kherson, which was sent to the United States Department of Agriculture from Southern Podolia, also in the Ukraine (Coffman, 1977). Thus, *Pg-2* appears to have been fairly common in the host population of the southern Ukraine during the latter part of the nineteenth century. Green Russian and Kherson became progenitors of some 130 cultivars in North America (Coffman, 1977; Coffman *et al.*, 1961), indicating how widespread and important *Pg-2* became.

Gene *Pg-3*, the last of the "pre-1950" genes, is also dominant. It was introduced to Canada from France in 1888–1889 in the cultivar Joannette. Its importance is more historical than practical, since neither the introduction nor gene *Pg-3* were used extensively in oat breeding. *Pg-3* has been in the international differential set almost since the beginning of race studies. In many respects the rust reaction it confers is unlike that of the genes previously described. It confers a mesothetic reaction to some races, a highly resistant one to avirulent races at low temperatures, and it is thermolabile (Roberts, 1962; Martens *et al.*, 1967). It is either closely linked to a gene conferring crown rust resistance, or itself confers resistance to both rusts (McKenzie *et al.*, 1968). Moreover, virulence on this type of resistance is inherited extra-chromosomally (Green and McKenzie, 1967).

Gene *Pg-4* is a dominant, thermolabile gene that was introduced to the United States via South Africa in the cultivar Hajira in 1919 (Welsh and Johnson, 1951). Authors agree on a North African origin, but Welsh and Johnson (1951) thought that it originally came from Egypt in 1904, while Coffman (1977) gives Algeria as the probable source. However, Hajira was not important as a cultivar or as germ plasm until the discovery of *Pg-4*, which was present in about 10% of the plants in the cultivar (Welsh and Johnson, 1951). This source of resistance has been used very widely, and together with *Pg-1* and *Pg-2* has in the past been the basis for breeding for stem rust resistance wherever there were breeding programs with that objective.

Gene *Pg-5* is probably the same as *Pg-4* (Welsh and Johnson, 1951).

Genes *Pg-6* and *Pg-7* are dominant genes in the diploid species *A. strigosa* Schreb., C.D. 3820, conditioning resistance to a wide range of races (Murphy *et al.*, 1958; Dyck and Zillinsky, 1962). However, it has not yet been possible to transfer this resistance to the hexaploid level. The two genes may be the same (Dyck, 1966).

The origin of *Pg-8*, a thermolabile, recessive gene, is somewhat difficult to trace. Browning and Frey (1959) identified the source as a hull-less cultivar from Africa (C.I. 3030 and C.I. 3031), but it was also present in C.I. 2710, an unnamed Chinese cultivar. Welsh *et al.* (1961) isolated the gene from R.L. 524.1, which is a Hajira × Banner hybrid, and the resistance most likely came from Hajira. Thus, wherever else it may have occurred, it appears to have been present in North Africa. As far as is known, *Pg-8* has not been used in breeding programs. It appears to be effective against eastern Australian races (Luig and Baker, 1973). It is also effective against races found in the *Berberis* areas of eastern North America, and it is effective in parts of South America

(Coelho, 1976) and parts of Russia (Suzdalskaya *et al.*, 1978). In the Great Plains region of the continent, over 90% of all field isolates are virulent on this type of resistance.

Pg-9, a thermolabile, recessive gene, probably originated from the U.S.S.R. It was introduced to the United States in 1930 (Coffman, 1977) in the cultivar Ukraine. Since its discovery it has been identified in lines from numerous breeding programs (McKenzie and Green, 1965), and in the introduction Kerkhiachskii 41 from the U.S.S.R. and in Sante Fe introduced from Argentina in 1945 (Coffman, 1977). As far as is known it has not been used in breeding programs until recently (McKenzie *et al.*, 1976). Like gene *Pg-3*, *Pg-9* is closely associated with a gene for resistance to *P. coronata* (McKenzie *et al.*, 1965); because of this association, it may be present in some North American cultivars. *Pg-9* is effective against the most common and widely virulent races of the Great Plains region, but not of the eastern region of North America.

Gene *Pg-10* is a dominant or partially dominant gene identified in the hexaploid hull-less lines C.I. 1575, C.I. 2641, and C.I. 2824 and conditions a moderately susceptible infection type to race NA 25 (Pavek and Myers, 1965). Because of the infection type it confers, this gene has not been used in differential sets.

Gene *Pg-11* is unique among the known genes for stem rust resistance in oats in that it is expressed only in the adult plant stage. Adult plants with *Pg-11* are resistant to all races that have been tested. It is an incompletely recessive gene that was isolated from C.I. 3034, a line that also carried gene *Pg-1* (McKenzie and Martens, 1968). C.I. 3034 originated from Rhodesia in about 1926, as a rust-resistant selection from a badly rusted field of the cultivar Burt. Burt is a selection from cultivar Red Rustproof, which is a Mexican oat that was brought back from Mexico to South Carolina, United States, by a soldier in 1848 or 1849 (Coffman, 1977). If the progenitor of C.I. 3034 was really Burt via Red Rustproof, we must try to explain the presence of *Pg-1*, which is not present in Burt and is widely believed to be of U.S.S.R. origin, occurring in White Russian and its derivatives. This cultivar was introduced to the United States in about 1850, the same time as Red Rustproof; since both have been widely used in breeding programs, natural outcrossing and/or admixtures might have occurred. Thus there are at least three choices for the putative origin for gene *Pg-11*: a *de novo* mutant from southern Africa; a western Black Sea origin along with *Pg-1*, -2, -9, and -15; and an Iberian origin via Burt, Red Rustproof, and Mexico.

There appears to be an association between the resistance and yellow plant color, weak straw, and somewhat reduced yield in the absence of rust. The seedlings with *Pg-11* are susceptible and have near-normal chlorophyll and carotenoid levels, but with increasing age and the onset of adult plant resistance the pigment content decreases more rapidly than in plants without the gene (Harder *et al.*, 1971). No significant change in the ratios of chlorophyll to carotenoids occurred during the shift from susceptibility to resistance. A gene affecting chlorophyll levels may be tightly linked with *Pg-11*. Alternately, 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 rust resistance. This type of resistance has been used successfully in cultivar improvement in Mexico (A. P. Roelfs, personal communication).

The *Pg-a* complex (Martens *et al.*, 1981) consists of gene *Pg-12* and a complementary or interacting gene(s). *Pg-12* is a recessive gene that was isolated from Kyto, a cultivar introduced from Yugoslavia via Finland by the United States Department of Agriculture in 1939 (Martens *et al.*, 1968). The cultivar Osmo expresses a rust reaction similar to that of Kyto (Green and McKenzie, 1964).

The pedigrees of Kyto and Osmo (Baum, 1972) indicate that they have the Swedish cultivar Victory = Milton = Probsteyer and unknown local Finnish cultivars, one of them from the Kuopio region of south central Finland, in common. Since Victory is the universally susceptible host for oat stem rust, Kuopio, Finland, becomes the prime candidate for the origin of *Pg-12*, as far as it can be traced. In the seedling stage, Kyto and Osmo are resistant to all races that have been tested. The infection type ranges from (,;) to (2) and is associated with severe necrosis but no chlorosis of the surrounding leaf tissue. Resistance diminishes as the plants develop, and while some resistant reactions can still be observed in adult plants, they appear moderately susceptible (Martens *et al.*, 1968). In interaction with another gene(s), *Pg-12* provides highly effective resistance against all but two of the oat stem rust races known to occur in North America (Martens *et al.*, 1981).

Gene *Pg-13* is a recessive gene isolated from *A. sterilis* collected near Tunis, Tunisia (McKenzie *et al.*, 1970). It is the first stem-rust-resistance gene to have been found in this species and is also one of the most effective genes available to breeders (Roelfs *et al.*, 1982; Martens, 1981; Table I). Although races with virulence on *Pg-13* have been observed, it has been found effective in most of the countries where it has

been tested (Martens *et al.*, 1976). Fidler and Dumont, cultivars with *Pg-13* in combination with several other genes, have recently been released (McKenzie *et al.*, 1981; McKenzie *et al.*, 1984).

Gene *Pg-14* is a partially dominant gene isolated by Mac Key and Mattsson (1972) from Milford (C.I. 5039), Winter Turf (C.I. 1570) and other lines. Milford is a Welsh cultivar, released in 1947, with both the Swedish Milton = Victory = Probsteyer line and Winter Turf = Grey Winter line prominent in its pedigree. Winter Turf is a very old cultivar that was introduced to the United States from England by George Washington in about 1764 (Coffman, 1977). It was apparently not noted for its stem rust resistance. From this evidence it becomes difficult to identify a putative origin for this gene.

Gene *Pg-15* is a partially dominant gene isolated from *A. sterilis* (Martens *et al.*, 1980) collected east of Uskudar on the Black Sea near Istanbul, Turkey. Races avirulent on *Pg-9* are usually also avirulent on *Pg-15* in the Great Plains region (Roelfs *et al.*, 1980; Martens, 1981). Lines with this gene have been shown to be highly effective in trap nurseries for detecting variability in the pathogen population (Martens and McKenzie, 1979). This gene has not yet been used in commercial cultivars.

Resistance to oat stem rust is known to occur in the diploid species *A. strigosa* Schreb., from which Murphy *et al.* (1958) and Dyck (1966) isolated a dominant gene. In the diploid background, this resistance is highly effective in many parts of the world. Stem rust resistance has also been found in collections of other diploid species, including *A. longiglumis* Durieu. from Mamura-Tiflet and Rabat-Meknes regions of Morocco (J. W. Martens, T. Rajhathy, and R. I. H. McKenzie, unpublished). Resistance also occurs in the tetraploid species *A. barbata* Pott *ex* Link. in the Middle East (Dinoor and Wahl, 1963), North Africa (J. W. Martens, unpublished) and Turkey (Martens *et al.*, 1980).

Repeated attempts to transfer resistance from the lower ploidy levels to the hexaploid *A. sativa* have been largely unsuccessful (Mac Key and Mattsson, 1972; Dyck, 1966; Rajhathy and Thomas, 1974). *Pg-16* (Martens *et al.*, 1979) is a highly effective gene from *A. barbata* that may be successfully transferred to the hexaploid level.

Thus, the best available evidence suggests that most of the known genes for stem rust resistance in hexaploid oats originate from two relatively small geographic areas: the region around the western Black Sea (Russian-Turkish) yielded *Pg-1*, -2, -9, -15, and possibly *Pg-11*, and the North African region yielded *Pg-4*, -8, -13, and possibly *Pg-3* via France. Further plant-collecting expeditions in quest of new genes for resistance should include these regions and should sample the indige-

nous host populations extensively. Finland and England are putative origins for genes *Pg-12* and *Pg-14*, respectively, as far as has been possible to trace them.

B. BREEDING FOR ENDURING STEM RUST RESISTANCE

In view of the variability and the wide virulence range of the pathogen throughout the world, and the limited numbers of available resistance genes, breeding for enduring disease control is a major challenge. How can it best be done?

The best intermediate-term prospect for the efficient control of stem rust in oats lies in the synthesis of cultivars carrying several effective genes for resistance with the germ plasm presently available. The basis for optimism regarding the effectiveness of this approach is the wheat stem rust example in North America (Roelfs, 1978; Green and Campbell, 1979) and Australia (Watson, 1981). On the Great Plains of North America, there have been virtually no losses due to stem rust for over 30 years, despite very large areas of near monoculture wheat, because of the use of complex resistance. The presently available genes for resistance offer considerable scope for complex resistance breeding, despite the problem of allelism. Seven of the seedling resistance genes in the hexaploid host occur in three independent linkage groups: genes *Pg-1*, -2, and -8 in one group; genes *Pg-4* and -13 in another; and genes *Pg-3* and -9 in a third group. Genes *Pg-12* and -15 occur independently of these groups and of each other (McKenzie *et al.*, 1970; Martens *et al.*, 1980).

Genes such as *Pg-8*, -13, and -16 if it can be transferred, and the *Pg-a* complex, in combination with *Pg-1*, -2, and -4, offer considerable scope for complex resistance breeding. If possible, genes should not be used singly in the production of new cultivars.

The apparent effectiveness of the *Pg-a* complex, which clearly demonstrates transgressive segregation for resistance, indicates that the search for similar additional systems in the existing germ-plasm banks should be pursued.

The search for new sources of resistance in the hexaploid state should be intensified. In view of how little plant collecting has been done in the past, extensive sampling of the populations in regions known to have yielded effective resistance genes in the past should almost certainly result in the discovery of additional genes.

The prospects of success with mutation breeding are not encouraging. Attempts to induce *de novo* resistance by irradiation, chemical

mutagen treatment or low-level chronic irradiation of growing plants using large populations have produced no new sources of useful resistance (McKenzie and Martens, 1974; Harder *et al.*, 1977). The greatest potential for mutation breeding may be in the transfer of resistance from lower ploidy levels to the hexaploid level.

VII. Conclusions

Puccinia graminis avenae is a highly variable pathogen that carries genes for virulence in excess of what is required for its survival in most parts of the world where it has been studied. Host genes for resistance to this pathogen are rare relative to those for *P. coronata* Cda. on the same host, even though both host-parasite systems are known to have coevolved in the same region for a very long time.

Most of the known genes for resistance are believed to originate from two relatively small geographic areas: the region around the western Black Sea, and North Africa. The prospects of controlling the disease by the use of the existing genes for resistance in multigene cultivars are good. The North African gene *Pg-13*, in combination with previously discovered genes, and the *Pg-a* complex are particularly promising. The search for new sources of resistance should be continued and intensified. Efforts to transfer resistance from diploid and tetraploid species of the genus to the hexaploid level should also be pursued.

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