Parasitic Specialization of *Puccinia graminis* f. sp. *avenae* in Israel During 1971–1977

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ABSTRACT

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The oat stem rust disease caused by *Puccinia graminis* f. sp. *avenae* occurs annually in Israel on the wild species *Avena sterilis*, the putative progenitor of cultivated oats, which is of countrywide distribution. The disease also attacks cultivated oats grown on a limited acreage. A total of 895 fungal isolates collected mainly from *A. sterilis* in 16 regions during seven successive years were studied for racial identity. The oat rust populations consisted of 12 physiologic races. Races 72 and 8 were the most common. Some shift over the years in the relative prevalence of race 8 that could not be ascribed to changes in oat cultivation was recorded. Two new races, never reported previously, were identified. One race is virulent only on the cultivar Saia and was tentatively designated as 1S. The other one is virulent on the hosts with *Pg*1, *Pg*3, and *Pg*9, resembling races 61 and 80 and designated here as 7HF.

An international survey for the oat stem rust disease revealed the presence of its causal agent, *Puccinia graminis* Pers. f. sp. *avenae* Eriks. & E. Henn., in Africa, Europe, South Pacific, North America, and South America (8). The disease has also been reported in Asia (19). In some years, oat stem rust caused severe losses in certain countries. In the United States, serious epidemics occurred in the major oat-producing areas, which include Iowa, Minnesota, North Dakota, South Dakota, and Wisconsin, reaching devastating proportions in 1953 (10–12). Oat stem rust occurs in most of Canada. In 1977, the disease caused crop losses estimated at 385,000 t of grain (7). In Europe, oat stem rust is very common in Italy (13) on cultivated and wild oats, Sweden (6), Yugoslavia (5), and other countries (8).

Race populations of *P. g.* f. sp. *avenae* have been studied by a number of investigators primarily to improve the efficiency of breeding programs concentrating on disease resistance (7) and to gain better understanding of the nature and variability of the pathogen. Differential cultivars endowed with specific genes for resistance have been employed in such studies.

In Israel, oats are cultivated on a very small acreage, but the indigenous hexaploid *Avena sterilis* L., the natural progenitor of cultivated oats, and other wild oat species serve as natural hosts of *P. g.* f. sp. *avenae*. Seventy-one species of grasses belonging to 35 genera also serve as hosts (21). Isolates from all species are cross-compatible, but the alternate host of the pathogen has never been found in the country.

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In 1926, studies on the parasitic specialization of the fungus were initiated in Israel, and during 1926–1934, the racial identity of a limited number of uredial samples was determined in the Federal Cereal Rust Research Laboratory in Minnesota. These studies were resumed in 1950 by Wahl and associates and continued for a number of years (2,21). Our investigation was undertaken to explore the race composition of *P. g.* f. sp. *avenae* samples collected in Israel from 1971 through 1977.

MATERIALS AND METHODS

Differential hosts. Seedlings of differential oat cultivars with specific genes for resistance were inoculated in the first-leaf stage. The cultivars used and their resistance genes are as follows (15): Minrus (*Pg*1, CI 2144), Richland (*Pg*2, CI 787), Jostrain (*Pg*3, CI 2660), Rodney (*Pg*4, CI 6661), Eagle[2] × CI 4023 (*Pg*8, CI 8111), Santa Fe Sel. (*Pg*9, CI 5844), Rosen's Mutant (*Pg*9, CI 8159), and Saia (CI 4639). Seed of the differential cultivars were provided by the USDA, ARS, Beltsville, MD.

Inoculum. Most of the 895 uredial samples collected from 1971 through 1977 for race identification were isolated mainly from *A. sterilis* randomly sampled throughout the season in the regions of Golan Heights, the Upper Galilee, Western Galilee, Lower Galilee, Sea of Galilee, Hula Valley, Valley of Esdraelon, Plateau of Menashe, Mount Carmel, Samaria, Judean Foothills, Judean Mountains, Central Coastal Plain, Southern Coastal Plain, Northern Negev, and Bet Shean Valley.

Inoculation. Each of the uredial samples was multiplied on seedlings of the susceptible cultivar Fulghum CI 708. Subsequently, urediospores of monouredial origin were individually increased on Fulghum seedlings, and after several inoculation-isolation cycles, a sufficient quantity of urediospores was obtained for differential host inoculation. For each of the previously mentioned differentials, four seedlings were inoculated by gently rubbing the leaves with cotton swabs moistened in a water suspension of urediospores. Before inoculation, seedlings were sprayed with 0.05% water solution of Tween 20 (polyoxyethylene sorbitan monolaurate). The inoculated seedlings were maintained for 24 hr in a water-saturated atmosphere in growth chambers maintained at 19–21 C, then placed on greenhouse benches at the same temperature.

Development of uredia was recorded at 12- and 14-day intervals after inoculation. The reactions were classified according to Stakman et al (14). Infection types ranging from 0; to 2+ were placed in the resistant class; infection types varying from 3− to 4 were placed in the susceptible class. Races were identified according to Stewart and Roberts (15). Virulence was expressed in terms of a formula suggested by Martens et al (9) that comprises numbers corresponding to those assigned to the *Pg* resistance genes. Numbers designating the genes that condition resistance to the specific culture were written first followed by a slash, and then by the numbers designating the ineffective resistance gene.

RESULTS

The 895 samples of *P. g.* f. sp. *avenae* collected across the country consisted of 13 races, viz., 1, 2, 3, 8, 36, 40, 60, 61, 72,

Table 1. Avirulence/virulence combinations of *Puccinia graminis* f. sp. *avenae* races identified in Israel during 1971–1977

Race designation[a]	Avirulence/virulence combinations on *Pg* genes for resistance
1	1, 2, 3, 4, 8, 9 Sa/
2	1, 2, 4, 8, Sa/3, 9
3	2, 3, 4, 8, 9, Sa/1
8	1, 4, 8, 9, Sa/2, 3
36	1, 2, 4, 8/3, 9, Sa
40	4, 9/1, 2, 3, 8, Sa
60	2, 4, 8, 9, Sa/1, 3
61	2, 4, 9, Sa/1, 3, 8
72	4, 9, Sa/1, 2, 3, 8
80	2, 4, 8, Sa/1, 3, 9
7HF	2, 4, Sa/1, 3, 8, 9
1S	1, 2, 3, 4, 8, 9/Sa

[a] Race designation after Stewart and Roberts (15).

Table 2. Frequency of annual occurrence of *Puccinia graminis* f. sp. *avenae* physiologic races in Israel during 1971–1977

Year	No. of isolates	Frequency of a given physiologic race (% within each year)											
		1	2	3	8	36	40	60	61	72	80	7HF	1S
1971	63	3.2	14.3	4.8	19.0	···	7.9	3.2	4.8	36.5	4.8	···	···
1972	118	1.7	19.5	0.8	32.2	3.4	9.3	14.4	···	15.3	0.8	···	3.4
1973	144	3.5	4.9	4.2	41.0	1.4	6.3	6.3	···	25.7	4.9	···	2.1
1974	131	1.5	12.2	11.4	28.2	7.6	0.8	9.2	···	20.6	3.0	···	5.3
1975	138	9.4	12.3	4.3	26.1	1.4	···	20.3	···	23.2	2.2	···	0.7
1976	196	1.5	21.9	2.0	12.8	···	1.0	1.5	9.2	14.8	7.1	28.1	···
1977	105	5.7	30.5	7.6	5.7	···	···	8.6	19.0	5.7	13.3	3.8	···
Mean %		3.79	16.54	5.01	23.63	1.97	3.64	9.07	4.71	20.34	5.16	4.56	1.51

Table 3. Frequency of occurrence (%) and geographic distribution of physiologic races of *Puccinia graminis* f. sp. *avenae* in Israel during 1971–1977

Geographic region	No. of isolates	Frequency of occurrence of a given race within a region											
		1	2	3	8	36	40	60	61	72	80	7HF	1S
Golan Heights	133	9.8	39.1	3.8	6.0	2.3	3.0	6.0	3.8	10.5	5.3	8.3	2.3
Hula Valley	26	7.7	34.6	3.8	3.8	···	11.5	···	3.8	15.4	11.5	7.7	···
Upper Galilee	53	3.8	34.0	1.9	9.4	7.5	···	15.2	7.5	9.4	3.8	7.5	···
Western Galilee	16	18.8	25.0	···	···	···	···	18.8	12.5	18.8	6.3	···	···
Lower Galilee	80	2.5	15.5	13.8	6.3	1.3	2.5	20.0	5.0	27.5	6.3	2.5	···
Sea of Galilee	27	7.4	22.2	18.5	···	···	···	···	7.4	18.5	11.1	14.8	···
Bet Shean Valley	20	···	20.0	···	35.0	···	···	···	10.0	20.0	5.0	10.0	···
Valley of Esdraelon	70	···	14.3	5.7	11.4	7.1	1.4	10.0	14.3	14.3	7.1	10.0	4.3
Plateau of Menashe	32	3.2	40.6	6.3	9.4	···	6.3	6.3	3.1	9.4	12.5	3.1	···
Carmel Mountain	13	···	···	30.8	23.0	···	···	7.7	···	30.8	7.7	···	···
Samaria	21	4.8	23.8	9.5	19.0	4.8	···	4.8	9.5	9.5	4.8	9.5	···
Judean Foothills	87	1.1	5.7	···	60.9	···	1.1	5.7	1.1	13.8	2.3	8.0	···
Judean Mountains	17	···	5.9	···	64.7	17.6	···	···	···	11.8	···	···	···
Central Coastal Plain	138	2.9	5.8	2.9	31.9	0.7	3.6	10.1	2.2	25.4	5.1	3.6	5.8
Southern Coastal Plain	122	1.6	1.6	3.3	36.9	···	4.9	9.8	3.3	25.4	3.3	9.8	···
Northern Negev	40	···	···	2.5	40.0	···	10.0	7.5	···	40.0	···	···	···

80, 7HF, and 1S. Avirulence/virulence combinations characterizing the listed races are shown in Table 1. Some of the races were reported earlier, but races 36, 61, and 80 were not identified in Israel previously, and races tentatively designated 1S and 7HF, to our knowledge, have never been reported before. Races 2, 8, and 72 predominated during 1971–1977, but the order of annual frequency showed some fluctuations (Table 2). Race 72 prevailed in 1971, race 8 in 1973, and race 2 became more common in 1976 and 1977. Race 72 was distributed in all geographic regions surveyed (Table 3), whereas races 1, 2, 1S, and 36 were absent in the arid Northern Negev and Mount Carmel. Races 3, 40, 60, 61, 80, 7HF, and 1S were not found in the Judean Mountains, and races 8, 40, 1S, and 36 were not identified at the Sea of Galilee, which is below sea level.

Gene *Pg*4 and gene(s) in Saia have provided broad overall protection for many years. Notably, cultivars with gene *Pg*4 have shown satisfactory resistance under field conditions for several decades, and selections of Saia cultivated in Israel possess resistance to most fungus isolates occurring in nature. Some isolates, however, were virulent on Saia in seedling tests as well as under field conditions (Table 4).

DISCUSSION

Adequate information on the race composition of *P. g.* f. sp. *avenae* is a prerequisite for successful breeding for disease resistance. Notably, the differential cultivars for race identification of isolates of *P. g.* f. sp. *avenae* are well suited for this purpose because they carry single genes for resistance much like Flor's well-known flax rust differentials (3).

Two concepts have been presented to explain the predominance of certain races and shifts in race populations. One concept postulates that changes in composition of genes for stem rust resistance in the cultivated crops affect the composition of race populations (3). The other theory (1) maintains that, in some cases, factors besides host resistance affect selection of rust biotypes. For example, the predominance in Sweden of races with a wide range of virulence genes could not be attributed to host resistance (4).

A unique opportunity for studying the evolution of *P. g.* f. sp. *avenae* race populations exists in Israel. The limited acreage of cultivated oats in the country is apparently of no consequence in the evolution of the oat stem rust organism, which is common on the indigenous *A. sterilis*, the putative progenitor of cultivated oats. The *A. sterilis-P. g.* f. sp. *avenae* association has coevolved in Israel from remote antiquity, partially undisturbed by man's interference.

Studies of many years failed to find race-specific resistance in *A. sterilis* of either the conventional, low-reaction type or the slow-rusting type (16,20).

Table 4. Frequency of virulence on oat seedlings with specified genes (*Pg*) for resistance to *Puccinia graminis* f. sp. *avenae* populations sampled during 1971–1977

Resistance gene	Number of virulent isolates	Percentage of virulent isolates
*Pg*1	469	52.4
*Pg*2	413	46.1
*Pg*3	804	89.8
*Pg*4	0	0.0
*Pg*8	300	33.5
*Pg*9	268	29.9
Saia	60	6.7

Races 8 and 72 of the parasite, embracing a wide range of virugenes, have prevailed on *A. sterilis* for many years, and their prominence should be ascribed to factors other than host reaction. Shifts in race populations cannot be attributed to changes in host resistance. Obviously, this predominance of races having a broad virulence spectrum does not support Vanderplank's (18) axiom that "unnecessary" genes for virulence impair the parasitic fitness of the fungus.

The recently discovered races 1S and 36 differ from the well-known races 1 and 2, respectively, by virulence on Saia. Presumably, both have originated by stepwise mutations in the same fashion as race 40 did from race 6 in past years (17).

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