Letter to the Editor

Race Dynamics of *Puccinia graminis* f. sp. *tritici* in the USA from 1973 to 1980

S. Nagarajan

Indian Agricultural Research Institute, Regional Station, Flowerdale, Simla 171002, India.
Part of the research reported here was done during the tenure of AvH fellowship with J. Kranz, Giessen, W. Germany.
Comments and suggestions from A. P. Roelfs, United States, and N. I. Luig, Australia, helped to improve the style of the manuscript.
Accepted for publication 15 November 1982.

Pathogen specialization in host-pathogen reaction is important in plant disease control. Different procedures have been suggested for the presentation, analysis, and interpretation of data on specific interactions (1,10). For example, the distribution of races over years can be consolidated and analyzed to gain an understanding of the dynamics of the pathogen population. Data from wheat stem rust (caused by *Puccinia graminis* f. sp. *tritici* Erickss. & Henn.) race monitoring done in the United States are extensive, systematic, readily accessible, and organized according to a race identification procedure that facilitates study of the dynamics of changes in specific virulence frequencies in the population. The numbers of stem rust samples collected each year along a field survey route and from trap plots established for race analysis vary depending on the prevalence of the disease. Areas with heavily infected crops often are over-represented, and the date of sampling and the host growth stage at times of sampling also may vary. All of these variables can affect the race composition spectrum, but there was no major change in host resistance from 1973 to 1980 (8). Therefore, variation in the frequency and occurrence of isolates is due either to environmental factors or fitness parameters. Understanding this, I analyzed the American data for the period 1973–1980 (4–6, 8–12) (Table 1) to determine whether the pathogen population has gained additional virulence for the host genes being evaluated.

Data for races of *P. graminis* f. sp. *tritici* from the United States were decoded for those with >0.6% frequency to determine the number of virulence genes per race (Table 1). The number of races with the same number of detected virulence genes also was determined. From these two parameters, the mean number of detected virulence genes per race in the population, designated as factors of virulence (*FV*) (Eq. 1), and the virulence value (*VV*) (Eq. 2), which represents the sum of the products of the two parameters and reflects the frequency of virulence genes in the pathogen population, were calculated. Comparison of *FV* values over years reveals whether races with greater or less virulence are increasing in the population.

FV is calculated as:

$$FV = \sum_{i=0}^{12} if_i/x \qquad (1)$$

in which i is the number of detected virulence genes in a race (ie, $i = 0, 1, 2, \ldots 12$), f_i is the number of races with i virulence genes, and x is the number of races identified in the population.

Comparison of *VV* values shows whether isolates with more virulence genes dominate the population or whether a certain level of virulence becomes stabilized. *VV* is calculated as:

$$VV = \sum_{i=0}^{12} iP_i \qquad (2)$$

in which P_i is the percentage of isolates of races with i genes for virulence in a population. Isolates of *P. graminis* f. sp. *tritici* are identified based on their reaction on 12 different lines with genes *Sr* 5, *Sr* 6, *Sr* 7b, *Sr* 8, *Sr* 9a, *Sr* 9b, *Sr* 9d, *Sr* 9e, *Sr* 10, *Sr* 11, *Sr* 13, and *Sr* Tt-1 (10). Although there are a number of other genes, the *FV* and *VV* values were calculated from the occurrence of virulence genes corresponding to these 12 resistance genes, because they constitute the differential lines used to identify races.

The *FV* value was 5.9 in 1973; it increased to 6.8 by 1975, stayed high for the next 2 yr, and dropped to 5.5 in 1980 (Fig. 1). During the last 3 yr few samples were obtained due to poor rust development (5,6,8), but the frequency of races with fewer virulence genes increased.

On the basis of 15 yr of data, Luig and Watson (2) showed that in Australia the stem rust pathogen gained additional virulence both in the area of Queensland and New South Wales, and in the area of Victoria, South Australia, and Tasmania. They found a conspicuous increase in the number of factors for virulence in the pathogen population at 5-yr intervals. In contrast, 8 yr of race analysis data from the United States showed that there was no clear increase in the total number of virulence genes of the United States pathogen population. Instead, it remained dynamic, fluctuating within limits that enabled survival of various virulence combinations.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

TABLE 1. Virulence spectra of races of *Puccinia graminis* f. sp. *tritici* collected in the United States between 1973 and 1980[a]

Year	Isolates (no.)	Classification[b] of isolates, number of virulence genes, and percentage frequency																		Remarks on disease spread
		BBC 1	QCB 3	MBC 3	LCL 4	HDL 4	QFB 4	QCC 4	HNL 5	HJC 5	TBM 6	TLM 7	TDM 7	QSH 7	RCR 7	RHR 8	TNM 8	RKQ 8	RPQ 8	RTQ 9	
1973	1,304	···	2	···	1	2	13	···	···	···	···	14	···	8	···	···	51	2	···	2	Dry weather
1974	2,609	···	4	3	···	1	10	2	2	···	1	17	2	3	···	···	52	2	1	1	Favorable
1975	2,418	···	7	···	···	···	1	···	···	···	···	3	5	8	1	···	68	1	1	1	Favorable
1976	1,703	···	3	···	···	···	3	···	···	···	···	7	10	3	···	···	72	1	···	1	Favorable
1977	1,207	···	···	···	5	···	6	···	1	···	···	1	7	17	1	···	53	3	1	3	Favorable weather in north
1978	790	···	2	1	···	1	5	···	···	···	···	···	13	11	···	···	57	2	1	4	Unfavorable
1979	420	···	2	4	···	2	19	···	2	2	···	4	6	9	3	···	38	6	···	2	Unfavorable
1980	143	6	4	2	1	1	26	···	···	1	···	···	8	···	2	1	43	5	···	1	Unfavorable

[a] Based on data from Roelfs and McVey (10–12) and Roelfs et al (4,6,8,9).
[b] Classification based on the system of Roelfs and McVey (10).

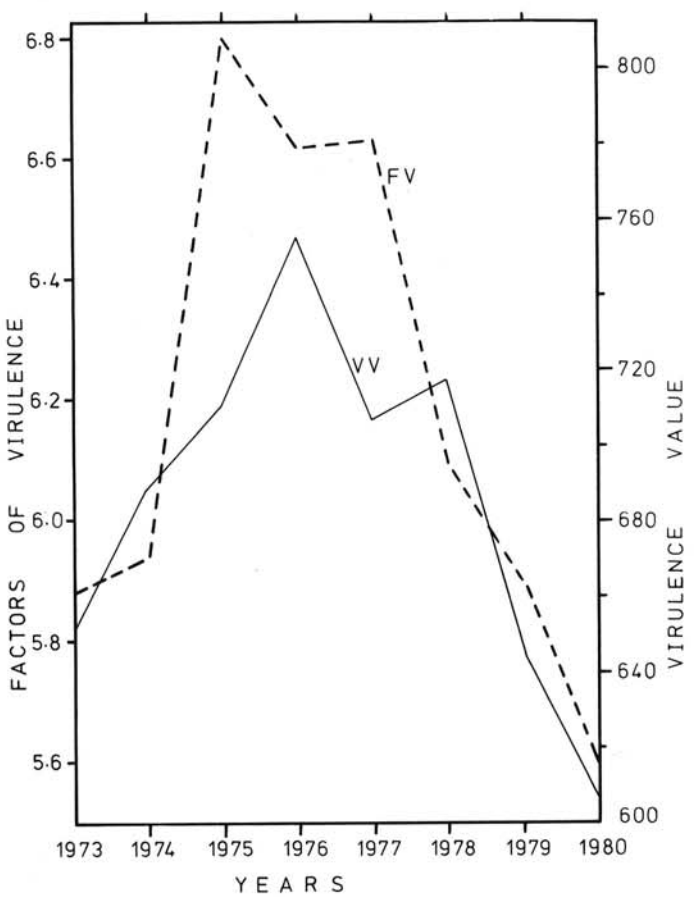

Fig. 1. The virulence value (*VV*) and the factors of virulence (*FV*) for the *Puccinia graminis* f. sp. *tritici* population in the United States during 1973–1980. Note that during the less severe stem rust years (1978 and 1979) both values declined, indicating that races with a few virulence factors dominated. See the text for details on *FV* and *VV* calculation. Graph generated based on the data of Roelfs and McVey (10–12) and Roelfs et al (4,6,8,9).

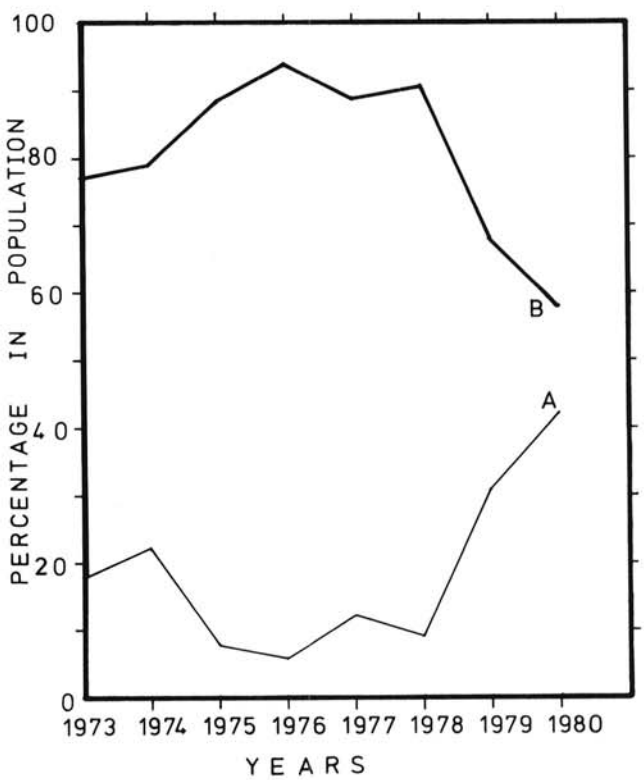

Fig. 2. Frequency of **A,** simple races and **B,** complex races in the population of *Puccinia graminis* f. sp. *tritici* in the United States during 1973–1980. Simple races have five or less identified genes for virulence and complex races have six or more. Note that simple races increased during 1979 and 1980 that were marginal and poor conditions for stem rust increase.

The *VV* increased steeply between 1974 to 1976, but dropped dramatically during the less favorable 1978 to 1980 crop seasons. During years when conditions were more favorable for disease (Fig. 1) complex races (races with virulence for >50% of the 12 differential lines) tended to dominate, while in unfavorable years simple races (with virulence to <50% of the 12 genes) increased both in number and frequency (Fig. 1).

In a sexual population of *P. graminis* f. sp. *tritici* in the United States, races with virulence corresponding to resistance genes at fewer than six loci were absent. Roelfs and Groth (7) attributed the absence to the poor survival of these races in winter and spring wheat grown in the Great Plains and adjoining areas. The present analysis shows that under marginal conditions (6,8) simple races with virulence to five or fewer resistant genes dominated. During the unfavorable years (1979 and 1980) such races constituted 31 and 42% of the pathogen population, while in favorable years (1975 and 1976) their frequency was only 8 and 6%, respectively (Fig. 2). Watson (13) observed that strains of *P. graminis* f. sp. *tritici* with few virulence genes survived better under field conditions and were more competitive in mixtures than those with additional genes for virulence. The present analysis does not substantiate this view; in the United States from 1973 to 1980 survival of races with more genes for virulence was better under a different set of conditions from that favoring races with few genes for virulence.

Vanderplank (14) theorized that additional virulence was gained

at the cost of fitness parameters. Watson (13) believed that fitness and virulence are not necessarily negatively associated and that the genes for virulence are independent of those for survival ability and aggressiveness. Fitness is generally characterized by sporulation and lesion size under ideal conditions (2). From the present analysis it appears that the ability to survive under extreme conditions may be equally important and that races with few virulence genes seem to possess it.

It appears that races with varying number of virulence genes fluctuate both in number and in percentage of the population. This may enable races with various gene combinations to survive, which is necessary to keep the pathogen co-evolving with the host. The 8 years of American data do not show that the population has gained additional virulence although it has been noted to be otherwise in Australia (3). Studies on the possible occurrence of ecological races and on the adaptability and survival of simple races under varying environments would give a new insight into this complex problem of fitness, survival, and evolution of new races.

LITERATURE CITED

1. Browder, L. E., Lyon, F. L., and Eversmeyer, M. G. 1980. Races pathogenicity phenotypes, and types cultures of plant pathogens. Phytopathology 70:581-583.
2. Hamid, A. H., Ayers, J. E., Schein, R. D., and Hill, R. R. 1981. Variation in parasitic fitness among isolates of *Cochliobolus carbonum* race 3. (Abstr.) Phytopathology 71:561.
3. Luig, N. H., and Watson, I. A. 1970. The effect of complex genetic resistance in wheat on the variability of *Puccinia graminis* f. sp. *tritici*. Proc. Linn. Soc. N. S. W. 95:42-45.
4. Roelfs, A. P., Casper, D. H., and Long, D. L. 1978. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1977. Plant Dis. Rep. 62:735-739.
5. Roelfs, A. P., Casper, D. H., and Long, D. L. 1979. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1978. Plant Dis. Rep. 63:701-704.
6. Roelfs, A. P., Casper, D. H., and Long, D. L. 1981. Races of *Puccinia graminis* f. sp. *tritici* in the United States in 1979. Plant Dis. 65:138-143.
7. Roelfs, A. P., and Groth, J. V. 1980. A comparison of virulence phenotypes in wheat rust populations reproducing sexually and asexually. Phytopathology 70:855-862.
8. Roelfs, A. P., Long, D. L., and Casper, D. H. 1982. Races of *Puccinia graminis* f. sp. *tritici* in the United States and Mexico in 1980. Plant Dis. 66:205-207.
9. Roelfs, A. P., Long, D. L., Casper, D. H., and McVey, D. V. 1977. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1976. Plant Dis. Rep. 61:987-991.
10. Roelfs, A. P., and McVey, D. V. 1974. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1973. Plant Dis. Rep. 58:608-611.
11. Roelfs, A. P., and McVey, D. V. 1975. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1974. Plant Dis. Rep. 59:681-685.
12. Roelfs, A. P., and McVey, D. V. 1976. Races of *Puccinia graminis* f. sp. *tritici* in the USA during 1975. Plant Dis. Rep. 60:656-660.
13. Watson, I. A. 1970. Changes in virulence and population shifts in plant pathogens. Annu. Rev. Phytopathol. 8:209-230.
14. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 206 pp.