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DISTRIBUTION OF STRIPE RUST VIRULENCES OF WHEAT AND VARIETAL RESPONSE IN PAKISTAN

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Abstract

The distribution of *Puccinia striiformis tritici* virulences prevalent in nature in the stripe rust zones of the Punjab, North Western Frontier and Baluchistan during the period 1986-88 is described. High virulence was observed in Punjab and NWFP on differentials carrying stripe rust resistance genes *Yr1, Yr2, Yr6, Yr7 and YrA*. Similarly pattern of high virulence was observed on commercial cultivars carrying stripe rust resistance genes *Yr2, Yr6, Yr7* and *YrA*. No virulence was observed in Baluchistan on either the differential hosts or any commercial cultivars except Local White which is not known to carry any resistance to stripe rust. Similarly virulence against host genes *Yr3, Yr4, Yr5, Yr8, Yr9, Yr10, YrSu 92* and *Yr15* was also not found.

Introduction

Wheat cultivated under a wide range of ecological conditions in Pakistan is severly affected by rusts because of the rapidity with which the disease spreads and devastates the crop. Of these the stripe rust, *(Puccinia striiformis* f. sp. *tritici* West) prevalent in the central and northern foot-hill areas of the Punjab, NWFP and uplands of Baluchistan as well as in Gilgit, Skardu and Chitral areas bordering Afghanistan and China is the most important disease which can limit the yields in certain years. During annual wheat rust surveys the first appearance of stripe rust was generally noted in farmer's fields in central Punjab (Faisalabad) around mid February which develops to high intensities in other areas of Punjab on susceptible cultivars by mid March. Similarly it was noted in NWFP (Mardan and Peshawar) during late March and developed to higher intensities by mid/late April. In uplands of Baluchistan, Gilgit, Skardu, Chitral and Kaghan it was wide spread during May-June. The stripe rust thrives well at temperatures ranging from 9 to 25°C with high relative humidity. Low light intensity generally gives susceptible reactions whereas high light intensities produce resistant reaction (Stubbs, 1985).

After the discovery of "Biological forms" (Stakman *et al.*, 1917) in *Puccinia graminis tritici*, Hangerford & Owens (1923), were the first to report that there were also "strains" in *P. glumarum tritici* (= *P. striiformis tritici* West). Johnson *et al.*, (1972) recommended a differential set which is currently being used by the stripe rust research workers for identifying the races. Stubbs (1985) reviewed the presence of stripe rust resistance genes and their distribution in the world. The disease normally spreads through dispersal of urediospores by wind. Unintentional human activity also plays a role in the spread of the disease or introduction of new virulences (Wellings, 1987). A new viru-

lence can be detected only if the corresponding resistance gene is present in a cultivar where it acts as a selecting agent. Thus a particular virulence is found in places where the corresponding gene for resistance either originated or is in wide use. The present report describes the virulence spectrum of stripe rust in the stripe rust zones of Pakistan.

Materials and Methods

Wheat nurseries comprising of 14 differentials known to carry specific genes for stripe rust resistance (Stubbs, 1985) as well as 16 different wheat cultivars commonly grown in the stripe rust zones of Pakistan (Table 1) were planted during 1986-88 at Quetta, Yousafwala (Sahiwal), Faisalabad, Islamabad, Pirsabak (Nowshera), Peshawar and Kaghan. Genes conditioning resistance to stripe rust in these cultivars had been determined (Pervaiz & Johnson, 1986; Hussain *et al.*, 1986 & Kirmani, 1986). Each line was planted in single row of one meter with rows spaced at 30cm. The entire nursery was surrounded by a mixture of susceptible spreaders comprising of cvs Morocco and Local White, the later also included as a "susceptible check" in the nursery. Stripe rust was allowed to develop under natural conditions. Stripe rust severity and reaction on each entry were recorded according to the recommended scale of Mc Neal *et al.*, (1971) at the maximum development of the disease in the adult plant stage at each location.

Results and Discussion

Data of average disease response from Faisalabad, Pirsabak, Kaghan and Quetta was found to represent the virulence spectrum for the respective provinces. The pattern of infection on the differential hosts as well as the commercial cultivars carrying postulated genes for resistance to *P. striiformis* was found to be similar during the three years of trial from 1986-88 (Table 1).

High infection type (HIT) was noted on the differential hosts Chinese 166 *(Yr1)*. Heines Kolben *(Yr6)* and Lee *(Yr7)* and on the supplemental differential hosts Kalyansona *(Yr2)* and Sonalika *(YrA)*. Infection type (IT) on Heines VII *(Yr2)* at Kaghan and Pirsabak was moderately high as compared to IT found at Faisalabad indicating that prevailing virulence was either of different type or could be due to the presence of additional genes in Heines VII. Moreover, high IT was observed on Kalyansona and the widely grown Cv. WL-711 possessing *Yr2* at Faisalabad. Pirsabak and Kaghan. No infection was however observed at Quetta on Heines VII, Kalyansona or on WL-711 indicating that virulence for *Yr2* is absent in Baluchistan. Blue silver *(YrA)*., another commercial cultivar gave HIT (Table 1) at all the locations except at Quetta. The high infection was comparable to the high infection on the supplemental differential host Sonalika *(YrA)* (Table 1). It would therefore, suggest that virulence for *(YrA)* is prevalent in the stripe rust zones of Punjab and NWFP, while it is absent in Baluchistan. Similarly, cultivar LU-26 possessing

Table 1. Adult Response of differential hosts and commercial cultivars against stripe rust of wheat in Pakistan during 1986-88.

	Host genes Yr	Location and province			
		Quetta Baluchistan	Faisalabad Punjab	Pirsabak NWFP	Kaghan NWFP
		Diseases Response*			
Differential Host					
Chinese 166	1	0	30S	30S	50S
Heines VII	2+	0	TMR-MS	10MS	TMSS
Vilmorin 23	3	0	0	0	0
Hybrid	4	0	0	0	0
Trit. Spelta album	5	0	0	0	0
Heines Kolben	6	0	60S	50MSS	40S
Lee	7	0	50S	60S	50S
Compair	8	0	0	0	0
Clement	9	0	0	0	0
Moro	10	0	0	0	0
Su92 x Omar	Su 92	0	0	0	0
V763-254 W6.2	15	0	0	0	0
Kalyansona	2	0	50S	50S	30S
Sonalika	A	0	80S	60S	40S
Commercial cultivar					
Blue silver	A	0	30S	40S	20S
WL-711	2	0	30S	40S	30S
Lu-26	6	0	40S	60S	30S
Lyallpur-73	6	0	30MRMS	10MRMS	10MRMS
Zamindar-80	6	0	15MRMS	10MRMS	20MRMS
Barani-83	7+2	0	5MR	10R	10R
Faisalabad-83	7+2	0	10MR	10MR	5MRMS
Pak-81	9	0	0	0	0
Pirsabak-85	9	0	0	0	0
Kohinoor-83	9	0	0	0	0
Sarhad-82	9	0	0	0	0
Punjab-85	9	0	0	0	0
Faisalabad-85	9	0	0	0	0
Sutlej-86	9	0	0	0	0
Khyber-87	9	0	0	0	0
Local white	0	70S	90S	70S	70S

*Disease Response =R = resistant, MR = moderately resistant,
MS = moderately susceptible,
S = Susceptible.

Yr6 gave high infection similar to that on the differential host Heines Kolben *(Yr6)*, CVS Lyallpur 73 and Zamindar 80 also possessing *Yr6* however, gave intermediate reaction at all the locations (except Baluchistan) thus indicating that these cultivars might be carrying some additional hitherto unidentified gene(s) responsible for resistance in the adult stage. CV. Barani 83 with *Yr7+2* gave resistant to moderately resistant reaction at Pirsabak, Kaghan and Faisalabad. Similar ITs though in low intensities were observed on Faisalabad 83 *(Yr7+2)*, indicating possibility of the presence of additional adult plant resistance (APR) genes in these cultivars.

No virulence was observed on either the differential host Clement *(Yr9)* or on the cultivars Pak-81, Sarhad-82, Kohinoor-83, Pirsabak-85 Punjab-85, Sutlej-86 and Khyber-87, all of which also carry *Yr9* in the IB/IR translocation (Table 1), thus indicating that virulence against *Yr9* was not available in the prevailing populations of *P. striiformis* in the country. Similarly virulence against resistance genes *Yr3, Yr4, Yr5, Yr8, Yr10, YrSu92* and *Yr15* was also not found.

The absence of virulence in Baluchistan against any of the resistance genes being currently used indicates that this province forms a distinct epidemiological zone as far as yellow rust is concerned. Local White, which is not known to carry any gene for stripe rust resistance is the predominant cultivar in the Baluchistan. It, therefore, appears that *P. striiformis* population prevailing in this area has not been subjected to any selection pressure sufficient enough to force it to mutate to a new virulence as is the case in other provinces of Pakistan.

The pathotype analyses of the samples collected from the Punjab and NWFP during 1986-88 have revealed the presence of race *7E150* of stripe rust (Kirmani, 1986; Stubbs, 1988). This race has virulence for host genes *Yr1, Yr2, Yr6, Yr7* and *YrA* (Table 1) in adult stage. It is widely distributed in the stripe rust zones of the Punjab and NWFP as well as in India and Nepal (Dubin & Stubbs, *Pers. Comm.*) Changes in virulence and their geographic distribution should therefore be monitored through common trap nurseries in Pakistan and also in neighbouring countries.

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